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Author: Casal, Jorge J.

Source: The Arabidopsis Book, 2012(10)

Published By: The American Society of Plant Biologists

URL: <https://doi.org/10.1199/tab.0157>

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First published on January 19, 2012: e0157. doi: 10.1199/tab.0157

Shade Avoidance

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The presence of neighboring vegetation modifies the light environment experienced by plants, generating signals that are perceived by phytochromes and cryptochromes. These signals cause large changes in plant body form and function, including enhanced growth of the hypocotyl and petioles, a more erect position of the leaves and early flowering in *Arabidopsis thaliana*. Collectively, these so-called shade-avoidance responses tend to reduce the degree of current or future shade by neighbors. Shade light signals increase the abundance of PHYTOCHROME INTERACTING FACTOR 4 (PIF4) and PIF5 proteins, promote the synthesis and redirection of auxin, favor the degradation of DELLA proteins and increase the expression of auxin, gibberellins and brassinosteroid-promoted genes, among other events downstream the photoreceptors. Selectively disrupting these events by genetic or pharmacological approaches affects shade-avoidance responses with an intensity that depends on the developmental context and the environment. Shade-avoidance responses provide a model to investigate the signaling networks used by plants to take advantage of the cues provided by the environment to adjust to the challenges imposed by the environment itself.

INTRODUCTION

Shade-avoidance responses are the changes in plant body form and function that occur in response to the light signals provided by neighboring vegetation and that tend to reduce the degree of current or future shade. Plants rely on the availability of photosynthetically-active radiation (PAR, 400–700 nm) to produce the carbohydrates used in their metabolism. The reduction of PAR below saturation levels lowers photosynthesis and can seriously compromise plant fitness. This has provided the evolutionary force to generate shade-avoidance responses.

Figure 1 shows shade-avoidance responses at different stages of the life cycle in *Arabidopsis thaliana*. The germination of *Arabidopsis* seeds can be repressed by shade light and this is a shade-avoidance response because it prevents the generation of seedlings immediately exposed to the limiting PAR levels at the base of deep canopies. The seeds will then germinate when the canopy becomes disturbed and the seeds exposed to unfiltered sunlight. *Arabidopsis* seeds can germinate under dense canopies but this requires sensitization by incubation in full darkness, a condition experienced when the seeds are buried (Shinomura et al., 1996; Botto et al., 1996). For a more detailed treatment of seed-germination responses we refer to the chapter dedicated to this subject (Bentsink and Koornneef, 2008).

At the seedling stage, shade light signals promote hypocotyl extension growth (Figure 1), and a longer stem places the cotyledons and early true leaves of the rosette at a higher position

within the canopy, reducing the degree of future or current shade. In addition, the gradients of light between sunflecks and shaded areas deviate the axis of hypocotyl growth towards the illuminated side. For a more detailed treatment of phototropic responses we refer to the chapter dedicated to this subject (Pedmale et al., 2010).

At the rosette stage, shade light signals cause upward bending of the cotyledons and true leaves, enhance petiole extension growth and can reduce leaf-lamina expansion (Figure 1). The upward bending of the leaves, caused by faster growth on their lower than their upper side, is called hyponasty. Hyponasty is a rapid response that places leaf lamina at a higher, better light position within the canopy. Longer petioles enhance this effect by increasing the amplitude of the change in leaf position. In addition, longer petioles can horizontally displace the position of the leaf lamina away from the shade of neighbors. If a plant is partially shaded by neighbors, reduced expansion of shaded leaves would reduce the proportion of shaded leaf area.

Shade light signals also accelerate flowering (Figure 1) and reduce branching. Since *Arabidopsis* typically grows as a rosette during the vegetative phase, the extension of the internodes that accompanies reproductive development places cauline leaves at higher strata within the canopy. Early flowering also reduces the chance of future shade by completing the cycle before the canopy becomes too closed. The advantage of extending the vegetative phase while the environment is favorable is that the more developed foliage can supply photoassimilates to a more developed

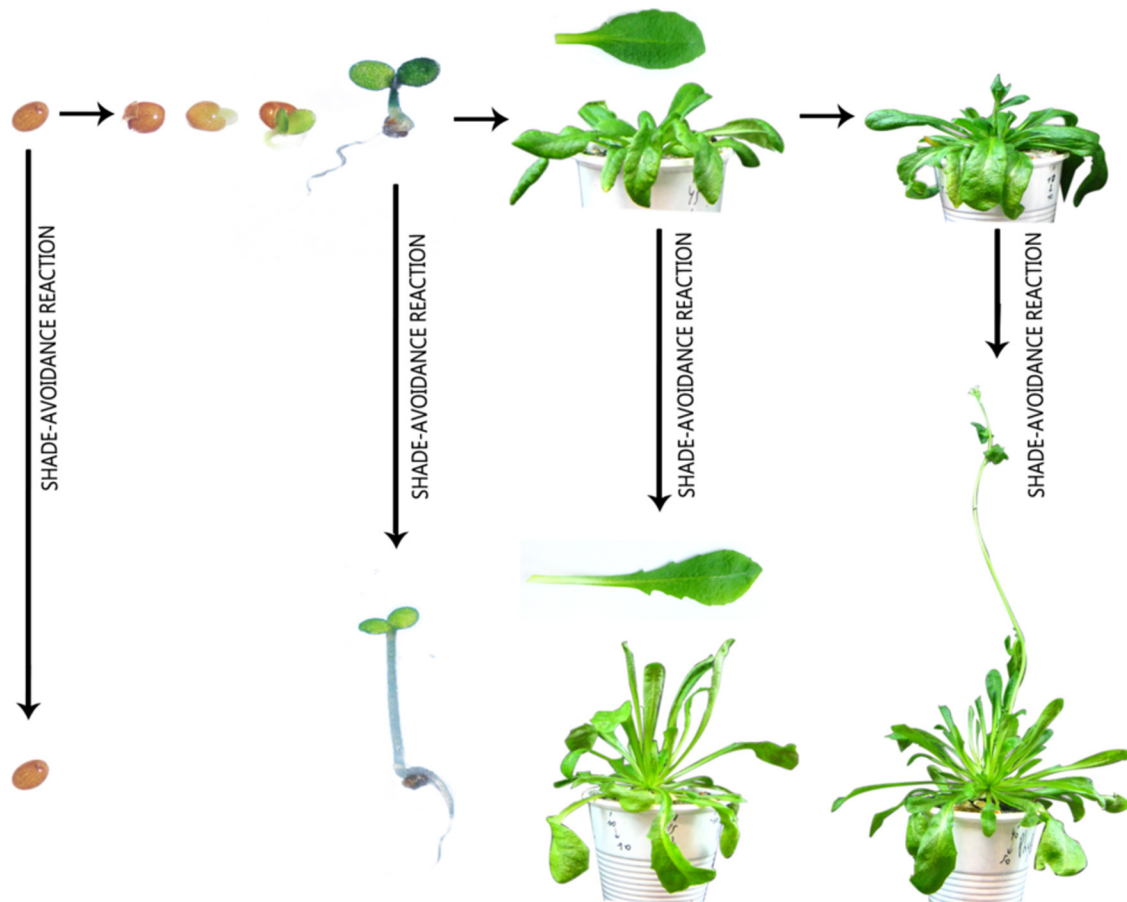


Figure 1. Shade light signals cause shade-avoidance responses throughout the life cycle of plants of *Arabidopsis thaliana*.

The upper set of pictures shows the progression of *Arabidopsis* plants under open conditions. Shade light causes alternative growth and developmental patterns including the repression of seed germination, the promotion of hypocotyl growth, the promotion of petiole growth and more erect leaves and early flowering.

reproductive structure, thus increasing fitness. However, delayed flowering would not be justified if the leaves become severely shaded. Reduced branching is a shade-avoidance reaction because new branches originate from the rosette, placed at the base of the plant and profuse branching would therefore increase the proportion of shaded tissues.

The adaptive benefits of the plasticity involved in shade-avoidance responses has been demonstrated by using mutants unable either to produce these responses in crowded vegetation stands or to restrain shade-avoidance reactions in open places (Ballaré et al., 1994; Casal et al., 1994; Schmitt et al., 1995; Keuskamp et al., 2010). The changes in plant body form that occur in response to shade light signals are beneficial in crowded environments and detrimental in open places where they, for instance, increase wind impact. Shade-avoidance responses are likely to have evolved with shade, as early as the Devonian (Mathews, 2006).

The first sections of this chapter describe the light signals able to initiate shade-avoidance responses and the photoreceptors that perceive these signals in *Arabidopsis thaliana*. Subsequent

sections present the mechanisms involved in the control of stem growth, leaf growth and flowering by shade light signals in *Arabidopsis thaliana*. These responses are presented separately to highlight differences that are often not recognized. The final sections deal with the differences and connections between shade-avoidance and shade-acclimation responses; a brief description of shade-avoidance responses in other species (in particular species of agricultural importance) to aid the use of *Arabidopsis* in translational approaches to improve crops, and final remarks.

LIGHT SIGNALS PROVIDED BY NEIGHBORING VEGETATION

The differences between sunlight and shade light

A light signal is a change in the light environment that can be perceived by plant photoreceptors. We will therefore define the changes of the light environment caused by the presence of neighbors and discern which of them are actually perceived by

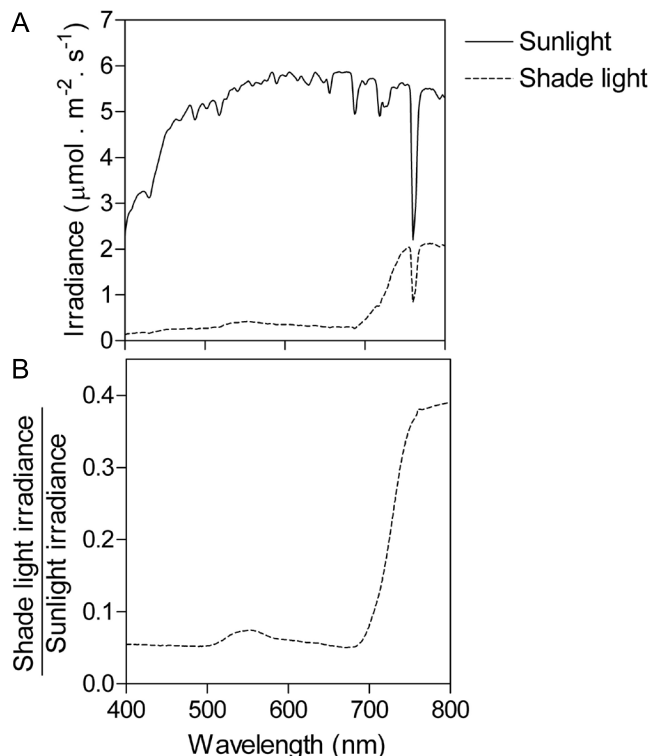


Figure 2. Differences between sunlight and shade light.

(A) Spectral distribution of sunlight reaching the top or the base of a dense vegetation canopy (respectively labeled sunlight and shade light).

(B) Spectrum of the ratio between the irradiance reaching the base of the canopy (shade light) and the irradiance of sunlight before making contact with the vegetation (calculated after A).

the plant and can therefore be considered as signals. Figure 2A shows the spectral distribution of incoming solar radiation between 400 and 800 nm compared to that observed under a dense canopy of green vegetation. The interference caused by the dense canopy reduces irradiance. Figure 2B shows the ratio between shade light and sunlight for the different wavelengths. Clearly, shade light shows a severe reduction of blue and red light, a slightly weaker reduction of green light and a relatively poor reduction of far-red light. This is caused by the optical properties of the green foliage, which absorbs more strongly in red and blue light than in green or far-red light.

Shade light signals

Not all the changes caused by shade are shade-avoidance signals. Only those changes of the light environment perceived by photoreceptors and wired to the responses in body form that reduce the degree of current or future shade. Shade light signals include the reduction in red / far-red ratio (R:FR) perceived by phytochrome, the reduction in red plus far-red irradiance perceived by phytochrome, and the reduction in blue-UV-A irradiance perceived by cryptochromes. Plants are also able to respond to the reduced

blue / green ratio in a cryptochrome-dependent manner. As light penetrates into the canopy these signals become more intense.

Dynamic changes in R:FR and irradiance signals

Full sunlight and deep shade light represent two extreme conditions. Figure 3 presents the different intensity stages of shade light signals in growing canopies. Compared to a fully isolated plant (Fig. 3A), a plant grown in a sparse canopy may experience increased levels of far-red light of the horizontally propagating light due to selective light reflection on the foliage of relatively distant, non-shading neighbors (Fig. 3B). This early warning signal of the presence of neighboring vegetation anticipates actual shade because the horizontally placed leaves are receiving full sunlight from above (Ballaré et al., 1987; Smith et al., 1990). With canopy growth there is a reduction of the irradiance of horizontally propagating radiation (Fig. 3C). Again, these changes impact mainly on vertical organs (typically the stem) and occur before the horizontal leaves become affected (Ballaré et al., 1989). Mutual shading of the most important photosynthetic organs begins only when the development of the foliage causes interference of direct light potentially reaching a given leaf by a neighbor leaf placed above it (Fig. 3D). This temporal pattern shows that neighbor signals able to initiate shade-avoidance reactions anticipate actual shade. For this reason shade avoidance can reduce the degree not only of current but also of future shade.

Experimental simulation of shade light signals

Shade light signals are complex due to their spatial and temporal variation. One system often used to investigate shade-avoidance reactions is to reduce the R:FR while keeping PAR constant (Morgan and Smith, 1976). This is achieved by adding far-red light to a source of white light common to all the treatments. PAR can be provided by artificial sources or by sunlight. The advantage of this approach is that plants can be exposed to realistic R:FR without altering other features of the environment. Strictly speaking this does not reproduce any of the situations described in Figure 3 because although the second stage involves increasing far-red without large changes in other wavebands, the R:FR signal reaches the vertical stem and not the horizontal leaves (Fig. 3B), whereas the simulation affects the R:FR reaching the leaves. Far-red light can be provided from one side. Under sunlight this can be achieved by using artificial sources (Casal et al., 1987b), far-red light reflecting mirrors (Ballaré et al., 1987) or actual green neighbors that reflect far-red light (Ballaré et al., 1987). Fiber-optic probes can be used to direct far-red light to the stem (Morgan et al., 1980), and this approach reproduces the second stage in Figure 3.

Another approach is to use a pulse of far-red at the end of the photoperiod, i.e. end-of-day far-red (EODFR) (Downs et al., 1957). The principle of this treatment is that although the exposure to light is brief it causes changes in the status of the photoreceptors involved in the perception of the R:FR that persist during the subsequent night (see phytochromes below). To be effective this brief reduction in R:FR has to be severe (i.e. pure far-red light) as weak reductions of R:FR below sunlight values have no effects (contrary to similar reductions provided during daytime)

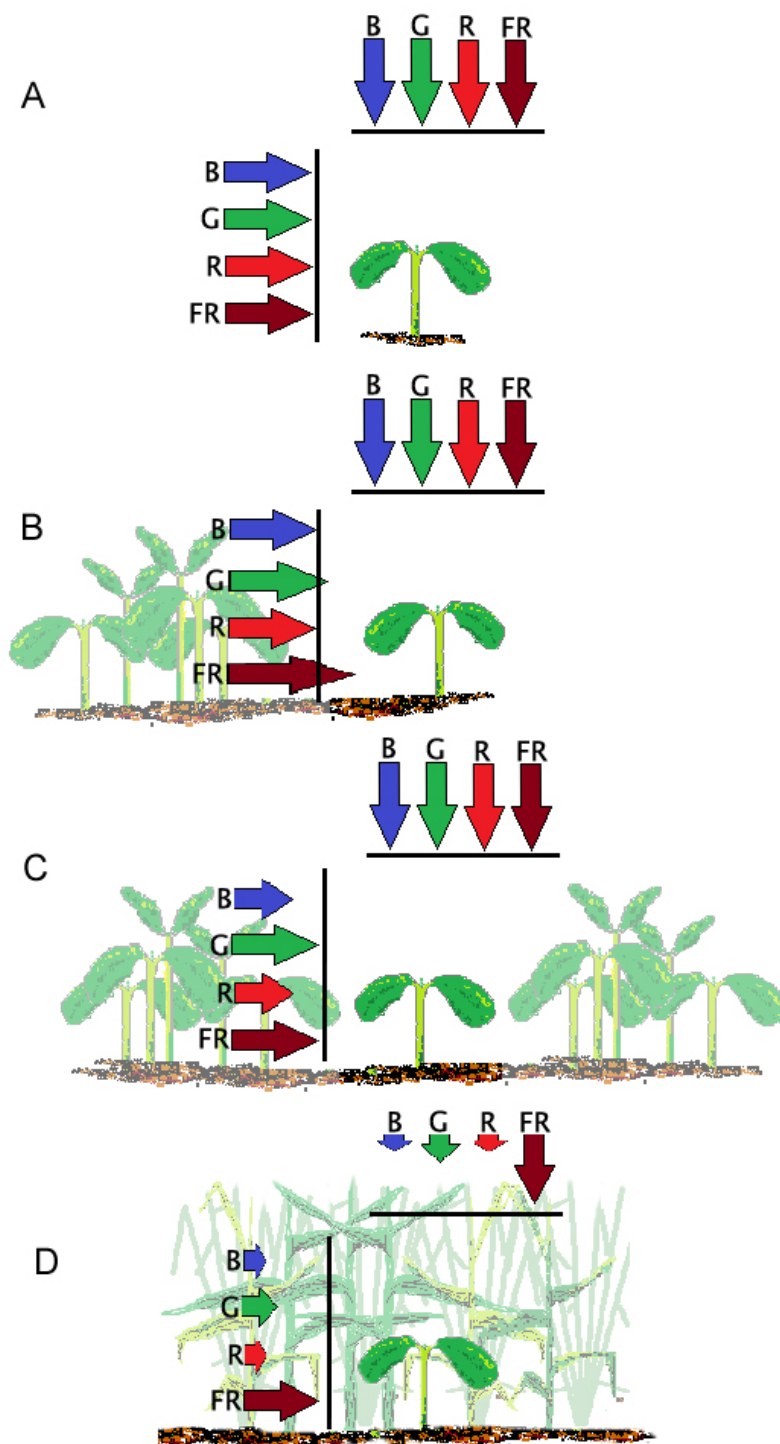


Figure 3. The signals of the light environment change in response to the density of the surrounding vegetation.

(A) Plant isolated from nearby vegetation. The arrows represent the irradiance of vertically- and horizontally-propagating blue (B), green (G), red (R) and far-red (FR) light. The plant receives full incoming radiation in each waveband (represented by the size of each arrow touching the 100% black line).

(B) Plant surrounded by neighbors that do not project their shade on it but reflect far-red light (note that the horizontal arrow corresponding to far-red light crosses the black line, indicating more than 100% irradiance).

(C) Plant surrounded by neighbors that shade its stem (note arrows shorter than 100% for horizontally-propagated light).

(D) Plant shaded with horizontal foliage shaded by neighbors (note reduced size of vertical arrows).

(Casal et al., 1990a; Sellaro et al., 2011). This weaker sensitivity to reductions in R:FR at the end of the photoperiod prevents a response to the reduced R:FR caused by atmospheric factors at the extremes of the photoperiod. Clearly, a very severe reduction in R:FR restricted to the end of the photoperiod (i.e. EODFR) is not found in the natural situation.

Neutral filters can be used to simulate the reductions in irradiance and colored filters to reduce selected wavebands (Yanovsky et al., 1995a). For instance, yellow and orange filters cut blue light, copper-sulphate filters reduce far-red. Green filters can simulate all aspects of shade light (Figure 3D) (Sánchez et al., 2011). The filters can be placed above the plants (then they will also affect PAR reaching the leaves) or surrounding the stem (Ballaré et al., 1991b), which is not a main photosynthetic organ.

While the aforementioned approaches simulate aspects of shade light and use a control exposed to unfiltered sunlight or an artificial white-light PAR source with high R:FR, it is also possible to use natural shade as a control and increase the R:FR by means of copper-sulphate filters placed either above the plants (Ballaré et al., 1991a) or surrounding the stem (Ballaré et al., 1990), or by using red-light emitting diodes selectively increasing the R:FR of photoreceptive tissues (Deregibus et al., 1985).

Finally, with the currently-available genetic tools it is also possible to compare real sunlight and shade light conditions and elucidate which of the environmental changes between these two conditions causes the observed physiological or molecular outputs by evaluating mutants disabled in the response to selected signals (Sellaro et al., 2010; Sellaro et al., 2011).

This brief overview demonstrates that there is a clear correlation between the accuracy of the simulation of the natural environment and the technical difficulties involved in this simulation. This should not discourage studies in this area because any of the above experimental conditions can be extremely useful to investigate shade-avoidance reactions provided that one is aware of the benefits and limitations (Fankhauser and Casal, 2004).

PHOTORECEPTORS INVOLVED IN THE PERCEPTION OF SHADE LIGHT SIGNALS

Phytochromes

Phytochromes are red and far-red light photoreceptors that bear a linear tetrapyrrole chromophore (Li et al., 2011). There are five phytochrome genes in *Arabidopsis* (*PHYA* through *PHYE*). The contribution of each phytochrome can vary with the different physiological outputs but phyA and phyB are clearly the most important. Phytochromes are synthesized in the Pr form that has the peak of absorption in red light. Upon light absorption excited Pr relaxes into the Pfr form, which has the peak of absorption in far-red light. Upon light absorption, excited Pfr relaxes to the Pr form. Pfr is biologically active and migrates from the cytosol to the nucleus. Under saturating light, a photoequilibrium is established between Pr and Pfr and the proportion of Pfr at photoequilibrium depends on the R:FR (Holmes and Smith, 1977; Smith et al., 1990). Phytochromes have a secondary peak of absorption in the blue-light region and can mediate responses to blue light compared to darkness (i.e. de-etiolation responses). However, in

the context of shade-avoidance reactions where red and far-red light are present the blue-light component has no major influence on phytochrome status (Mancinelli, 1986).

The total amount (Pr+Pfr) of phyB is relatively stable. However, *Arabidopsis* phyB can undergo dark reversion from Pfr to Pr (Elich and Chory, 1997; Sweere et al., 2001). Under weak light, this dark reaction competes with photoconversion and therefore the level of Pfr of phyB will decrease not only with low R:FR but also with low irradiance. At high irradiance, photoconversion is much faster than dark reversion and the level of Pfr depends only on R:FR. The amount of phyB Pfr will depend on the R:FR but under low irradiances these levels will be below those corresponding to photoequilibrium. Therefore, phyB is a sensor of R:FR and of red irradiance. phyD, phyE and phyC are predicted to operate in a comparable manner (Franklin et al., 2003). The dark reversion of Pfr to Pr is variable in magnitude in different cellular contexts and biologically meaningful amounts of Pfr can persist for several hours even in full darkness. This is the principle of the EODFR treatment, which reduces Pfr levels to a minimum immediately prior to the beginning of the night. In the absence of EODFR, Pfr persists in darkness and a demonstration of this persistence is that plants can respond to a pulse of far-red (which acts by severely reducing Pfr levels) even if delayed several hours into the night (Downs et al., 1957; Casal, 1996).

The photoconversion properties of phyA are similar to those of phyB and the occurrence of phyA dark reversion shows natural variation in *Arabidopsis* (Eichenberg et al., 2000). However, while total phyB levels are relatively stable, phyA Pfr is rapidly ubiquitinated and degraded in the 26S proteasome (Clough and Vierstra, 1997; Hennig et al., 1999). Therefore, the steady state level of phyA Pfr is not well related to R:FR because higher R:FR shifts photoconversion towards Pfr but exposes more Pfr to destruction. Furthermore, phyA Pfr migration from the cytosol to the nucleus requires binding FAR-RED ELONGATED HYPOCOTYL 1 (FHY1) and FHY1-LIKE (FHL) but once in the nucleus, FHY and FHL binding reduces Pfr activity. Therefore, Pfr must be photoconverted to Pr to release FHY/FHL and subsequently it must be photoconverted back to Pfr for biological activity (Rausenberger et al., 2011). As a result of phyA turnover (synthesis and degradation) and the photoconversions between Pr and Pfr and vice versa in the cytosol and the nucleus, the peak of phyA activity occurs at wavelengths closer to the peak of absorption of Pfr than of Pr absorption (Rausenberger et al., 2011), which corresponds to very low R:FR. phyA activity is strongly fluence-rate dependent, likely because higher fluence rates increase the rate of FHL/FHY-phyA complex assembly and disassembly and hence phyA transport capacity (Rausenberger et al., 2011). While the peak of phyA activity is in far-red light, phyA also operates under red light showing a weak contribution at very-low fluence rates (Mazzella et al., 1997) but a significant contribution at high irradiances (Franklin et al., 2007). Therefore phyA is a sensor of the reductions in red plus far-red irradiance caused by increasing shade. The activity of phyA is not significantly affected by R:FR in the range between unfiltered sunlight values (1.1) and 0.3 (Sellaro et al., 2010) but it increases with the very low R:FR observed in severely shaded environments (Smith et al., 1997). In summary, the activity of phyA decreases with increasing degrees of shade due to the reduced irradiance levels and under very dense canopies it increases again thanks to a very low (more favorable) R:FR.

Cryptochromes

Cryptochromes are photolyase-like blue light receptors that bind flavin adenine dinucleotide and could also bind a putative second chromophore (Yu et al., 2010). Arabidopsis has three cryptochrome genes but only *CRYPTOCHROME 1* (*CRY1*) and secondarily *CRY2* genes have been shown to participate in shade-avoidance reactions. The activity of cryptochromes increases with the levels of blue light and therefore cryptochromes are sensors of irradiance levels modified by the degree of shade. There are shade-avoidance responses to the blue / green ratio that require the action of cryptochromes (Sellaro et al., 2010) but the molecular basis of this dependency is a matter of debate (Banerjee et al., 2007; Bouly et al., 2007; Liu et al., 2010).

Other photoreceptors

In addition to the changes of the light environment described above, shade also reduces UV-B irradiance and this change perceived by UVR8 (Rizzini et al., 2011) could initiate shade-avoidance reactions (Ballaré et al., 1991c) but this possibility remains to be tested. Canopies are heterogeneous and create horizontal gradients that can be perceived by phototropins (Briggs and Christie, 2002). Some responses to green light are present in mutants of phytochromes and cryptochromes (Zhang et al., 2011). Therefore, while phytochromes, especially phyB, are the most important photoreceptors for shade-avoidance reactions followed by cryptochromes, especially cry1, other photoreceptors could enlarge the list in the future.

STEM GROWTH

Hypocotyl growth is promoted by different signals of neighboring vegetation. At the rosette stage, internode elongation is arrested but this is not the case in the *cry1 phyB* double mutant if grown at high temperatures (Mazzella et al., 2000), in the quadruple *phyA phyB cry1 cry2* mutant (Mazzella et al., 2001), in the *phyA phyB* mutant exposed to EODFR (Devlin et al., 1996) and in the *phyA phyB phyE* mutant (Devlin et al., 1998). Although the photoreceptors are clearly important to maintain the rosette structure, internode elongation at this stage cannot be regarded as a physiological shade-avoidance response in Arabidopsis until the wild type is shown to extend these internodes under shade conditions. At a later stage, compared to the wild type grown under high R:FR, the height of the inflorescence stem is promoted by low R:FR and by the *phyB* mutation but these effects are relatively small (Finlayson et al., 2010). For this reason, most of the research on stem responses to shade light signals in Arabidopsis involves hypocotyl growth.

The hypocotyl is a transition organ that shows two temporally separable responses to light. When first exposed to light the hypocotyl shows de-etiolation (induced by light compared to darkness) and only then it becomes fully competent to respond to shade-light signals. The inhibition of hypocotyl-growth in response to light during de-etiolation is not simply the mirror image of the promotion of hypocotyl growth during shade avoidance. One important difference is that low R:FR are suitable for de-

etiolation and inhibit early hypocotyl growth almost as well as high R:FR, whereas at later stages low R:FR promote hypocotyl growth compared to high R:FR. A low R:FR can be achieved experimentally by adding far-red light to a PAR source with high R:FR. This treatment inhibits early hypocotyl growth when compared to the high R:FR condition because the higher level of red plus far-red irradiance perceived by phyA (Salter et al., 2003; Strasser et al., 2010) dominates over the low R:FR. Therefore, for hypocotyl-growth analysis under such experimental conditions it is advisable to allow a couple of days for de-etiolation under a high R:FR ratio source before applying the different R:FR treatments (Johnson et al., 1994).

For the promotion of hypocotyl growth, the reduced red irradiance and R:FR caused by increasing shade are perceived by phyB (Figure 4), the reduced red plus far-red irradiance is perceived by phyA, the reduced blue irradiance is perceived by cry1 in the Landsberg *erecta* accession and by cry1 and cry2 in Columbia and the reduced blue / green ratio is perceived by or requires cry1 in Landsberg *erecta* (Sellaro et al., 2010). In addition to its direct role inhibiting hypocotyl growth in response to increasing irradiance, phyA has a more indirect role reducing the extent of phyB-mediated inhibition of hypocotyl growth (Cerdán et al., 1999). This regulation of phyB signaling by phyA is observed under natural radiation (Mazzella and Casal, 2001; Sellaro et al., 2010) and makes the seedlings more sensitive to shade-light signals as the *phyA* mutant does not show the promotion of hypocotyl growth caused by neighbors that reflect far-red light without shading (stage shown in Fig. 3B) (Casal, 1996).

When transferred from high to low R:FR, Arabidopsis seedlings show no hypocotyl-growth response during a lag of approximately 45 min, followed by a first phase of growth promotion between 45 and 150 min, a phase of reduced growth between 150 and 230 min and a second promotion of hypocotyl growth beyond 230 min (Cole et al., 2011). This kinetics of induction is similar (with quantitative differences) to that reported in classical experiments with *Sinapis alba* (Morgan et al., 1980; Child and Smith, 1987). The reversal of the promotion once the seedlings return to high R:FR depends on the duration of the exposure to low R:FR (Cole et al., 2011), and this could reflect an effect derived from leaf perception of low R:FR (Casal and Smith, 1988). Under free-running conditions the promotion of hypocotyl growth recorded 24 h after 2-h exposure to low R:FR is stronger if low R:FR are given during subjective afternoon (Salter et al., 2003). This promotion is attenuated and phase-shifted in *timing of cab expression 1* (*toc1*) mutant affected in a central component of the clock (Strayer et al., 2000) indicating a circadian gating of hypocotyl shade-avoidance responses (Salter et al., 2003). However, a clock regulation is not apparent during the first 10 h of exposure to low R:FR when growth is monitored at high resolution (Cole et al., 2011). Under day-night cycles of natural radiation, shade events (2 h) are more effective to promote hypocotyl growth if they occur in the afternoon (Sellaro et al., 2012).

The strength of the hypocotyl-growth response to low R:FR shows substantial natural variation unrelated to the length of the hypocotyls under high R:FR, to the latitude of the location of original collection, or to the variability in the response of flowering to low R:FR (Botto and Smith, 2002). A few accessions, such as CT-1 and No-0, show long hypocotyls under white light and reduced response to EODFR (Coluccio et al., 2011).

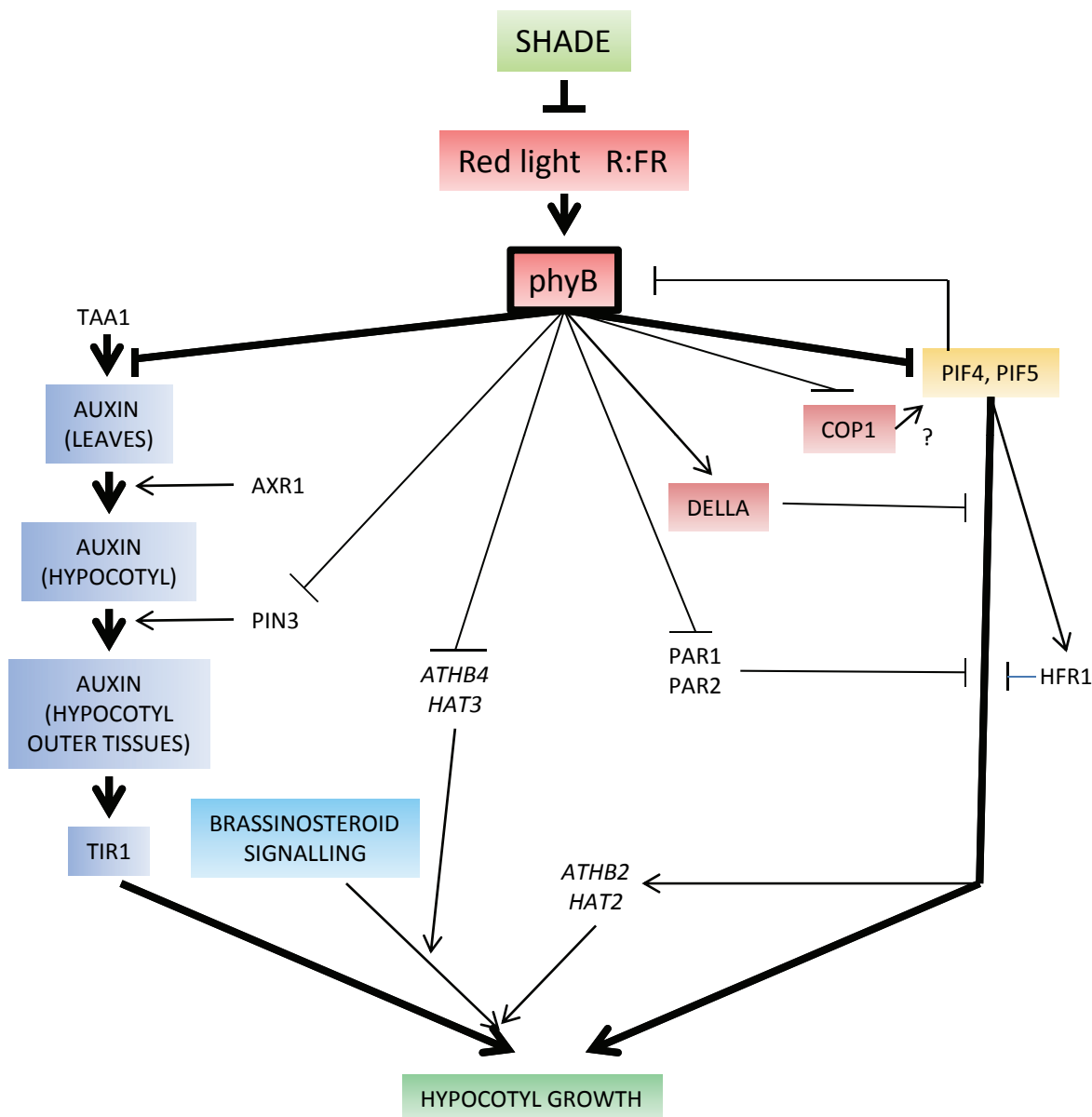


Figure 4. Simplified representation of the network involved in the promotion of hypocotyl growth by low R:FR and low red irradiance of shade light perceived by phyB.

Hypocotyl shade-avoidance responses require PHYTOCHROME-INTERACTING FACTOR 4 (PIF4), PIF5 and CONSTITUTIVE PHOTOMORPHOGENESIS 1 (COP1).

PHYTOCHROME-INTERACTING FACTOR (PIF) proteins are basic helix–loop–helix transcription factors that bind the active form of phyB (some also bind phyA) in the nucleus and as a result of this interaction become rapidly phosphorylated, apparently ubiquitinated and finally degraded in the proteasome (Leivar and Quail, 2011). The function of PIF proteins in shade-avoidance reactions is supported by genetic and molecular experiments. Under constant white light with high R:FR, compared to the wild type the hypocotyl is slightly shorter in the *pif4*, *pif5* and *pif4 pif5*

mutants and longer in the *PIF5* overexpressor. *pif4*, *pif5* and *pif4 pif5* mutants and the *PIF5* overexpressor have reduced hypocotyl growth responses to low R:FR and are partially epistatic to the *phyB* mutation (Lorrain et al., 2008). These observations indicate that shade-avoidance responses require normal levels of PIF4 and PIF5. The *pif4 pif5* mutant lacks key components of the signaling mechanisms leading to enhanced hypocotyl growth and the *PIF5* overexpressor already has a long hypocotyl in the absence of shade signals. When *Arabidopsis* seedlings are transferred from white light with a high R:FR to a low R:FR PIF5 and PIF4 re-accumulate. Increased PIF5 levels are already observed 15 min after transfer to low R:FR and persists at least during the subsequent 2 h (Lorrain et al., 2008). While PIF4 and PIF5 levels

increase rapidly, the *pi4 pi5* double mutant shows only a weak reduction of the promotion of hypocotyl growth by low R:FR during the first 10 h of treatment (Cole et al., 2011), suggesting that the impact of PIF protein levels on growth is relatively slow. These results indicate that the long-term promotion of hypocotyl growth caused by the low levels of active phyB established by low R:FR is partially accounted for by the accumulation of growth promoting PIF4 and PIF5 transcription factors (Figure 4). In addition to the regulation of PIF protein levels by phyB there is a reciprocal regulation as PIF3, PIF4 and PIF7 also help to maintain low levels of phyB (Leivar et al., 2008a). Shade-avoidance reactions would be favored by both the re-accumulation of PIF proteins allowed by the reduced phyB Pfr levels and the further reduction of phyB Pfr by PIF proteins.

The hypocotyl-growth response to low R:FR or EODFR requires the E3 ligase CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) (McNellis et al., 1994). However, the mechanism of COP1 action during shade-avoidance responses is not well established. In this regard, COP1 is required for the accumulation of PIF3 (and likely other PIF proteins) in the dark (Bauer et al., 2004; Leivar et al., 2008b) and it would be interesting to evaluate whether COP1 is also necessary for the low R:FR-induced re-accumulation of PIF (Figure 4). EARLY FLOWERING 3 (ELF3) may act at the biochemical level as an adaptor/scaffold protein facilitating COP1 activity (Yu et al., 2008) or by forming an evening complex required for the correct diurnal expression of *PIF4* and *PIF5* (Nusinow et al., 2011). ELF3 is a likely candidate gene to account for a quantitative trait locus for hypocotyl growth responses to low R:FR between the accessions Bayreuth-0 and Shahdara (Coluccio et al., 2011). This is also true for other shade-avoidance responses (see below) but the mechanisms of action of ELF3 during shade-avoidance responses remain to be elucidated.

Hypocotyl shade-avoidance responses require auxin, gibberellins and brassinosteroid signals

Auxin. Several mutations or pharmacological treatments that affect auxin synthesis, auxin transport or auxin perception impair the promotion of hypocotyl growth by low R:FR. The *SHADE AVOIDANCE 3* (*SAV3*) / *TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1* (*TAA1*) gene is expressed in the cotyledons (very poor expression in the hypocotyl) and *SAV3/TAA1* catalyzes the formation of indole-3-pyruvic acid from L-tryptophan (L-Trp) in the auxin biosynthetic pathway (Tao et al., 2008). *PINFORMED 3* (*PIN3*) (Friml et al., 2002) and *PIN7* are auxin efflux transmembrane transporters, *AUXIN RESISTANT 1* (*AXR1*) is an ubiquitin-activating enzyme that controls stability of auxin efflux carriers (Sieberer et al., 2000), and *TRANSPORT INHIBITOR RESPONSE 1* is an auxin receptor (Dharmasiri et al., 2005; Kepinski and Leyser, 2005). The promotion of hypocotyl growth by low R:FR is reduced in the *sav3* (Tao et al., 2008), *pin3*, *pin7*, *axr1* and *tir1* mutants (Keuskamp et al., 2010) and in wild-type seedlings exposed either to naphthylphthalamic acid (NPA), which blocks polar auxin transport, or α -(phenylethyl-2-one)-IAA, which is an antagonist for the auxin receptor TIR1 and its homologs (Steindler, 1999; Keuskamp et al., 2010). The hypocotyl of the *sav3-2* mutant does not respond to low R:FR during the first 4 h of treatment, and only then shows a mild promotion of extension growth (Tao et al., 2008; Cole et al., 2011).

Low R:FR not only require normal auxin synthesis, transport, perception and signaling; they also modify key aspects of these processes. At the whole-seedling level of resolution, low R:FR promote free auxin levels (Tao et al., 2008) and the expression of *PIN3*, *PIN7* and the auxin-induced transcription factor genes *IAA1*, *IAA3*, *IAA5*, *IAA11*, and *IAA19* among several other auxin-related genes (Devlin et al., 2003). Low R:FR increase auxin signaling in the cotyledons (Tao et al., 2008). Both free-auxin and cotyledon auxin signaling responses require *SAV3*, but the expression of *SAV3/TAA1* is not increased by low R:FR (Tao et al., 2008). In the hypocotyls, low R:FR promote the expression of *PIN3* and direct PIN3-GFP from the basal to the lateral side of the membrane of the endodermal cells, increase free IAA levels in a PIN3-dependent manner and increase the activity of the auxin-responsive *IAA19* promoter fused to GUS, particularly in the outer tissues of the hypocotyl (Keuskamp et al., 2010). Therefore, low R:FR favors *SAV3/TAA1*-mediated auxin synthesis in the leaves (Tao et al., 2008) and *PIN3-3* mediated lateral auxin redistribution towards epidermal and cortical cells of the hypocotyl (Keuskamp et al., 2010), which in turn would promote the elongation of these cells and of the whole organ (Morelli and Ruberti, 2000).

Low R:FR could also alter the responsivity to auxin. The expression of the homeodomain-leucine zipper transcription factor genes *ARABIDOPSIS THALIANA HOMEODOMAIN PROTEIN 2* (*ATHB2*) / *HAT4* and *HAT2* is rapidly and reversibly promoted by low R:FR (Carabelli et al., 1993; Roig-Villanova et al., 2006). This promotion depends partially on PIF4 and PIF5 (Lorrain et al., 2008) (Figure 4). Transgenic seedlings with elevated levels of expression of *ATHB2* or *HAT2* have long hypocotyls under high R:FR and altered responses to auxin whereas transgenic seedlings with reduced levels of *ATHB2* expression show short hypocotyls (Steindler, 1999; Morelli and Ruberti, 2002; Sawa et al., 2002).

The promotion of hypocotyl growth caused by lowering blue light is reduced in the *sav3*, *pin3*, *pin7*, *tir1* and *tir1afb1afb2afb3* mutants (where *afb1*, *afb2* and *afb3* are mutations at AUXIN SIGNALING F-BOX protein genes, homologs to *TIR1* (Dharmasiri et al., 2005; Kepinski and Leyser, 2005)) compared to the wild type (Keuskamp et al., 2010). The response is also reduced in the wild type by the application of NPA or the inhibitor of auxin perception α -(phenylethyl-2-one)-indole-3-acetic acid (Keuskamp et al., 2010). These observations indicate that the promotion of hypocotyl growth by lowering blue light also requires auxin synthesis, auxin transport and auxin perception.

Gibberellins. The promotion of hypocotyl growth by the *phyB* mutation (Reed et al., 1996), low R:FR or low blue light (Djakovic-Petrovic et al., 2007) is impaired by the addition of the inhibitor of gibberellin biosynthesis paclobutrazol and by the use of mutant backgrounds deficient in gibberellin synthesis. Whether phyB affects total seedling levels of this hormone is not clear because some active gibberellins do not show differences between the wild type and the *phyB* mutant and others could not be detected (Reed et al., 1996). The expression of the *GIBBERELLIN 20-OXIDASE 3* (*GA20OX3*) gene, which encodes an enzyme involved in the synthesis of gibberellins, is strongly promoted by low R:FR (Devlin et al., 2003) but this response is difficult to interpret because *GA20OX3* expression is down-regulated by elevated gibberellin signaling (Sun, 2008).

While phyB-mediated changes in active gibberellins have not been detected, phyB clearly reduces the responsivity to gibberel-

lins (Reed et al., 1996), suggesting alterations downstream of hormone levels. The DELLA proteins REPRESSOR OF GA (RGA), GIBBERELLIC ACID INSENSITIVE (GAI), RGA-Like1 (RGL1), RGL2 and RGL3 become degraded after the activation of the receptor of gibberellins (Sun, 2008). DELLA proteins are negative regulators of stem extension growth that reduce hypocotyl growth in part by impeding PIF4 and PIF3 binding to DNA (De Lucas et al., 2008; Feng et al., 2008). Although low R:FR strongly promote the expression of the *GAI* gene (Devlin et al., 2003), both low R:FR and low blue light reduce the abundance of DELLA proteins in the hypocotyls and these responses are blocked by paclobutrazol (Djakovic-Petrovic et al., 2007). The gibberellin-insensitive *gai* gain-of-function mutant, which has a stable GAI protein, shows reduced responses to low R:FR or low blue light, indicating that the induction of DELLA degradation by these shade light signals is a requisite for the growth response. In addition, a quadruple *gai rga rgl1 rgl2* loss-of-function mutant shows partially elongated hypocotyls, suggesting that degradation of DELLA could by itself account for part of the hypocotyl response to low R:FR (Djakovic-Petrovic et al., 2007). The quadruple *gai rga rgl1 rgl2* mutant has a response to low blue light even larger than that observed in the WT (Djakovic-Petrovic et al., 2007) and this response is almost completely abolished by the addition of NPA (Pierik et al., 2009), suggesting that DELLA proteins could restrain auxin-mediated responses to low blue light.

Brassinosteroids. A full hypocotyl-growth response to shade light requires brassinosteroids. When grown under a plant canopy, the brassinosteroid biosynthesis mutant *diminutol dwarf1* (Klahre et al., 1998) is unable to show the typical promotion of hypocotyl growth caused by shade light (Luccioni et al., 2002) (Figure 4). The promotion of hypocotyl growth observed in response to lowering blue light is reduced in the brassinosteroid synthesis mutant *rotundifolia 3 (rot3)* (Kim et al., 1998), in wild-type seedlings treated with the brassinosteroid synthesis inhibitor brassinazole and in the *bri1* mutant (Keuskamp et al., 2011) affected in the *BRASSINOSTEROID INSENSITIVE 1 (BRI1)* brassinosteroid-receptor gene (Wang et al., 2001). However, none of these effects is complete and only the simultaneous blocking of both auxin and brassinosteroid pathways eliminates the response to low blue light (Keuskamp et al., 2011). XYLOGLUCAN ENDOTRANS-GLUCOSYLASE /HYDROLASE (XTH) enzymes represent a potential control point for cell elongation (Cosgrove, 2005) and the expression of several *XTH* genes is promoted by lowering blue light and the response of some of these genes requires either auxin perception or brassinosteroid synthesis (Keuskamp et al., 2011). Interestingly, the combined application of auxin and brassinosteroids has additive promotion effects on hypocotyl growth (Keuskamp et al., 2011). One mode of integration of auxin and brassinosteroid pathways involves the phosphorylation of AUXIN RESPONSE FACTOR 2 (ARF2) by brassinosteroid-regulated activity of the GSK3 kinase BRASSINOSTEROID-INSENSITIVE 2 (BIN2) (Vert et al., 2008). This reduces ARF2 DNA binding and repression activities, leading to synergistic increases in transcription of auxin-response genes (Vert et al., 2008).

The expression of *BRI1* (Devlin et al., 2003) and of the homeodomain-leucine zipper transcription factors genes *ARABIDOPSIS THALIANA HOMEODOMAIN PROTEIN 4 (ATHB4)* and *HAT3* (Roig-Villanova et al., 2006; Sorin et al., 2009) is promoted by low R:FR. The *athb4 hat3* mutant has normal hypocotyl length under

high R:FR but it fails to respond to low R:FR and responds poorly to the addition of brassinosteroids (Sorin et al., 2009), suggesting that low R:FR could increase the sensitivity to brassinosteroids (Figure 4).

Negative regulation of hypocotyl shade-avoidance responses

The previous paragraphs describe a largely positive action of shade signals on pathways that promote hypocotyl growth as those involving PIF proteins, auxin, gibberellins and brassinosteroid. This section describes the positive action of shade signals on negative regulators of hypocotyl shade-avoidance responses. While several genes show a transient promotion of expression in response to low R:FR, the bHLH family member *LONG HYPOCOTYL IN FAR-RED LIGHT (HFR1)* gene, is rapidly promoted by low R:FR and retains elevated levels of expression several days later (Sessa et al., 2005). This promotion depends on PIF4 and PIF5 (Lorrain et al., 2008). When the seedlings are grown for 3 d under high R:FR photoperiods and then transferred for 4 d to low R:FR, the *hfr1* mutant shows an exaggerated promotion of hypocotyl growth and a stronger and/or more persistent enhancement of the expression of the *ATHB2*, *PHYTOCHROME INTERACTING FACTOR 3-LIKE 1 (PIL1)*, *PIF6/PI2*, *BIM1*, *PHYA* and *XTR1* genes among others typically promoted by low R:FR (Sessa et al., 2005; Roig-Villanova et al., 2007; Hornitschek et al., 2009). Similarly, *PHYTOCHROME RAPIDLY REGULATED 1 (PAR1)* and *PAR2* are atypical bHLH genes whose expression is rapidly and reversibly promoted by low R:FR even in the presence of the protein synthesis inhibitor cycloheximide (Roig-Villanova et al., 2006). Overexpression of *PAR1* or *PAR2* reduces the hypocotyl-growth response to low R:FR, and the expression response to low R:FR of selected auxin-signaling genes, whereas reduced expression of *PAR1* and/or *PAR2* increases hypocotyl growth (Roig-Villanova et al., 2007). As noted by Roig-Villanova et al. (2007) the *hfr1* mutant has an exaggerated response to low R:FR under low irradiances but the *hfr1*, *par1* or *par2* phenotypes are weak when low R:FR are provided at high irradiances. Therefore, *HFR*, *PAR1* and *PAR2* are negative regulators of shade-avoidance responses whose expression is promoted by shade light generating a negative feed-back (Figure 4).

PAR1, *PAR2* and *HFR1* proteins lack a typical basic domain necessary for binding to E-box and G-box motifs in the promoter of target genes. *HFR1* forms non-DNA-binding heterodimers with PIF4 and PIF5, preventing their binding to DNA and biological activity (Hornitschek et al., 2009). The mechanism of action of *PAR1* is apparently similar to that of *HFR1* (Galstyan et al., 2011).

PIL1 expression shows a strong and rapid promotion by low R:FR (Salter et al., 2003), which depends on PIF4 and PIF5 (Lorrain et al., 2008). The *pil1* mutants show phase-shifted and attenuated promotion of hypocotyl growth caused by 2 h exposure to low R:FR measured 24 h later (Salter et al., 2003) and an enhanced promotion when the seedlings are grown for 5 d under low R:FR (Roig-Villanova et al., 2006). Therefore, *PIL1* can be a positive or negative regulator of shade-avoidance responses apparently depending on the kinetics of the shade light signal.

Several B-box-containing zinc finger transcription factors (B-BOX DOMAIN PROTEIN, BBX) are involved in hypocotyl-growth responses to shade light. The *bbx19*, *bbx21* and *bbx22* mutant

seedlings show long hypocotyls whereas *bbx18* and *bbx24* mutant seedlings show short hypocotyls under natural or simulated shade (Crocco et al., 2010). The *bbx21 bbx22* double mutation restores the hypocotyl-growth response to shade light in the *cop1* mutant background, suggesting that BBX proteins act downstream COP1 (Crocco et al., 2010). Compared to sunlight, natural shade promotes the expression of *BBX21*, *BBX19* and *BBX22* and reduces the expression of *BBX18* and *BBX24*, indicating that these genes are also part of the feed-back inhibition of shade-avoidance responses (Crocco et al., 2010).

Sunfleck repression of hypocotyl shade-avoidance response

Plant canopies are heterogeneous and have gaps that allow the penetration of sunflecks. These sunflecks depend on the position of the gap and on solar elevation and therefore, their occurrence is repeated every clear day at approximately the same time. The activation primarily of phyB and secondarily of phyA by sunflecks of 2 h duration causes a strong inhibition of hypocotyl growth compared to continuous shade light, particularly when the sunflecks take place late in the photoperiod (Sellaro et al., 2011). Sunflecks cause large changes in the transcriptome, including the enhanced expression of the *ELONGATED HYPOCOTYL 5 (HY5)* bZip transcription-factor gene (Oyama et al., 1997). The levels of *HY5* are low both in seedlings grown under sunlight as well as in seedlings grown under uninterrupted shade light. However, after several hours of shade light the expression of *HY5* is promoted by the transition to sunlight, i.e. the expression of *HY5* is selectively high under sunfleck conditions (Sellaro et al., 2011). The *hy5* mutant shows impaired hypocotyl-growth inhibition and transcriptome responses when exposed to sunflecks. In particular, sunflecks reduce the expression of auxin-related genes and *PHYTOCHROME KINASE 4 (PKS4)* (Schepens et al., 2008) in an *HY5*-dependent manner (Sellaro et al., 2011). Mutants with severely impaired rhythms fail to respond to sunflecks even in the afternoon while the *late elongated hypocotyl (lhy) circadian clock associated 1 (cca1)* double mutant (Mizoguchi et al., 2002) shows a slightly higher response to morning sunflecks but it retains the higher effectiveness of afternoon sunflecks. In summary, the activation of phyB and phyA after several hours of shade light in the day promotes the expression of *HY5* which represses the expression of auxin-related and *PKS4* genes causing reduced hypocotyl growth. A permissive action of the clock helps to reinforce the action afternoon compared to morning sunflecks (Sellaro et al., 2011).

LEAF GROWTH

Petiole elongation

Compared to Arabidopsis plants grown isolated from nearby vegetation, plants grown in dense canopies show longer petioles (Ballaré and Scopel, 1997; Djakovic-Petrovic et al., 2007). Lowering the R:FR causes a promotion of petiole growth already detectable 2 h after the beginning of the treatment (Djakovic-Petrovic et al., 2007; Sasidharan et al., 2010). EODFR also promotes petiole

growth compared to the controls directly transferred from white light to darkness (Kozuka et al., 2010). The *phyB* mutant has longer petioles than the wild-type (Nagatani et al., 1991; Reed et al., 1993) and the *phyA phyB phyD* and *phyA phyB phyE* mutants have longer petioles than the *phyA phyB* mutant, (Devlin et al., 1998; Devlin et al., 1999). The promotion of petiole growth in response to either low daytime R:FR or EODFR is absent or even inverted (inhibition) in the *phyB* mutant (Nagatani et al., 1991; Devlin et al., 1996; Pierik et al., 2009; Kozuka et al., 2010), which retains some response to increasing canopy shade (Ballaré and Scopel, 1997). Lowering blue light caused little promotion of petiole growth in some experiments (Djakovic-Petrovic et al., 2007; Pierik et al., 2009) and a robust promotion of petiole elongation in others, where the *cry1* mutant showed constitutively long petioles and no response to low blue light (Keller et al., 2011). Therefore, the promotion of petiole growth by shade light signals is mediated primarily by phyB and secondarily by phyD, phyE and cry1.

Under constant white light *pif5* but not the *pif4* mutant shows reduced petiole response to low R:FR. The *pif4 pif5* double mutant has a shorter petiole under high R:FR but apparently normal responses to low R:FR. The *PIF5* overexpressor shows long petioles under high R:FR that do not respond to low R:FR but do not reach the length observed in wild-type plants exposed to low R:FR (Lorrain et al., 2008). The *pif4*, *pif5* and *pif4 pif5* mutants show reduced petiole-growth responses to low blue light, (Keller et al., 2011). These observations indicate that while altered levels of *PIF4* and/or *PIF5* can distort petiole growth responses, the function of these proteins is probably less central for petiole than for hypocotyl growth.

The promotion of petiole growth by shade light signals is impaired in several auxin- or brassinosteroid-related mutants. The *doc1/big* mutant, affecting a callosin-like protein gene involved in auxin transport (Gil et al., 2001) and the *rot3* mutant, deficient in brassinosteroid synthesis have short petioles with reduced responses to EODFR and are epistatic to the *phyB* mutation (Kozuka et al., 2010). The *sav3* auxin synthesis mutant (Tao et al., 2008) and the *axr1* mutant and the *axr2-1/iaa7* gain-of-function mutant (Nagpal et al., 2000) also impair petiole-growth responses to daytime low R:FR (Sasidharan et al., 2010). The *sav3* mutant and a partial loss of function *bri1* mutant show severely reduced petiole-growth responses to low blue light, while these responses are normal in *pin3* and the quintuple *yuc3 yuc5 yuc7 yuc8 yuc9* mutant (Keller et al., 2011). YUCCA (YUC) enzymes catalyse a rate-limiting step in tryptophan-dependent auxin biosynthesis (Zhao et al., 2001). Clearly, petiole-growth responses to shade light signals require auxin and brassinosteroid signaling. However, there is only partial information concerning the effects of light signals on these hormone pathways controlling petiole growth. It is known that auxin-responsive and brassinosteroid-responsive genes are overrepresented among the EODFR-induced genes in the petiole 2 h after the transition to darkness and that EODFR does not cause a detectable increase in auxin levels in the petiole or leaf blade (Kozuka et al., 2010).

EODFR given only to the blade is more effective to promote petiole growth and petiole changes in gene expression than EODFR given to the petiole itself (Kozuka et al., 2010). The auxin-transport inhibitor NPA reduces the petiole growth response to EODFR when added to the agar in experiments with excised leaves and when sprayed to intact plants (Kozuka et al., 2010).

and the response to low daytime R:FR when brushed onto the leaves (Pierik et al., 2009). The *pin3* mutant shows a slightly delayed promotion of petiole extension in growing canopies (Keuskamp et al., 2010). Therefore, auxin transport might play a role in blade-petiole communication (Kozuka et al., 2010).

EODFR up-regulates the expression of the *GA20OX2* gene involved in gibberellin biosynthesis, and transgenic Arabidopsis plants with reduced *GA20OX2* expression show reduced petiole elongation response to EODFR (Hisamatsu et al., 2005). This observation suggests that the EODFR promotion of petiole growth could involve increased synthesis of gibberellins in response to EODFR, but the latter interpretation remains to be tested. Low R:FR reduce the abundance of DELLA in the petiole with a kinetics that matches that of petiole growth (Djakovic-Petrovic et al., 2007). Wild-type plants treated with paclobutrazol, the gibberellins-deficient *ga1* mutant and the *gai* mutant that bears a stable GAI protein respond deficiently to low R:FR, indicating that the promotion of petiole growth requires gibberellins and degradation of DELLA. However, quadruple mutants of DELLA proteins do not show elongated petioles and retain apparently normal responses to low R:FR. Therefore, the low R:FR-induced degradation of DELLA is a requisite for normal petiole responses to R:FR but in contrast to the case of hypocotyl growth, lowering DELLA levels genetically is not enough to phenocopy petiole-growth promotion by low R:FR (Djakovic-Petrovic et al., 2007). Although auxin facilitates the degradation of DELLA proteins in the petiole, they do not exert their effect via this pathway (Pierik et al., 2009). The quintuple *della* mutant conserves normal responses to reduced blue light. Lowering blue light does not trigger DELLA degradation in Arabidopsis petioles, but the gain of function *gai1* mutant fails to respond, implying that elevated levels of DELLA impair the response (Keller et al., 2011).

Low R:FR stimulates ethylene production by the shoot (Pierik et al., 2009). The *ethylene-insensitive 2* (*ein2*) mutant, the *ein3 ein-3 like 1* (*eil1*) double mutant (Schaller and Kieber, 2002) and wild-type plants treated with the ethylene action inhibitor 1-methylcyclopropane do not show the promotion of petiole growth by low R:FR indicating that ethylene is required for this shade-avoidance response. However, supplementary ethylene has only minor effects on petiole growth (Pierik et al., 2009).

In the petiole, enhanced xyloglucan degrading activity and enhanced expression of *XTH5* and *XTH17* (among other *XTH* genes) accompany the growth promotion induced by low R:FR or simulated shade light and this growth promotion is absent or reduced in *xth15* and *xth17* mutants (Sasidharan et al., 2010). Conversely, expansin activity (Cosgrove, 2005) does not obviously correlate with the petiole growth responses (Sasidharan et al., 2010). The *hfr1* mutant has exaggerated petiole elongation, indicating that the negative regulation of shade-avoidance responses operates beyond the hypocotyl stage (Sessa et al., 2005).

Leaf expansion

Compared to the wild type under high R:FR, leaf area is reduced by low R:FR and by the *phyB* mutation (Nagatani et al., 1991; Reed et al., 1993; Devlin et al., 1999). Leaf area is also reduced in *phyB phyD* compared to *phyB*, indicating a role of *phyD* (Devlin et al., 1999). However, the *phyB* mutation can increase leaf area

(Robson et al., 1993) suggesting that the final output depends on growth conditions (Devlin et al., 1999). The leaf area response to low R:FR is also strongly context dependent and plants grown at 16 rather than 22 °C show a promotion and not a reduction in leaf area compared to high R:FR controls (Franklin et al., 2003). The *hfr1* mutant has reduced leaf area expansion, indicating that HFR1 negatively regulates diverse shade-avoidance responses (Sessa et al., 2005).

The reduction in leaf lamina area can be the result of both increased demand of resources by the petioles and direct mechanisms of shade light action on the lamina. In experiments conducted in *Sinapis alba* where one leaf of the first pair was covered while the other leaf (and the rest of the shoot, including the stem) was exposed to EODFR, the leaf exposed to EODFR showed reduced extension growth and accumulation of dry matter and structural carbohydrates, and higher activities of sucrose-phosphate synthase (an enzyme positively linked to carbon export from the leaves), than the leaf not exposed to EODFR (Yanovsky et al., 1995b). Since both leaves were attached to the same internode these effects cannot be assigned to increased stem demand and demonstrate the occurrence of direct effects of shade light on the leaves. However, the leaf covered during the exposure to EODFR showed some growth reduction compared to the leaves of control plants not exposed to EODFR. This suggests that increased growth of the stem could reduce the growth of the leaf covered during the EODFR treatment (Yanovsky et al., 1995b).

Among the direct effects, in Arabidopsis the reduced leaf area under low R:FR is caused by decreased cell proliferation (Carabelli et al., 2007). When seedlings are transferred to low R:FR after 7 d under high R:FR, a significant reduction in the activity of a cell division marker is observed 8 h after transfer to low R:FR. In seedlings grown for only 4-5 d under high R:FR, transfer to low R:FR causes a rapid promotion of the synthetic *DR5* promoter activity (used to estimate auxin signaling intensity) and the *CYTOKININ OXIDASE 6* (*CKX6*) (Werner and Schmülling, 2009) promoter activity in leaf primordia (already detectable 4 h after transfer) and a severe arrest of leaf primordia growth that is not observed in the *tir1* or *ckx6* mutants (Carabelli et al., 2007). These observations support a rapid promotion in auxin signaling by low R:FR inducing cytokinin degradation via the promotion of *CKX6*, which would reduce cell proliferation (Carabelli et al., 2007). Despite the differential effect of EODFR on petiole and leaf-lamina growth, EODFR increases the expression of auxin-responsive and brassinosteroid-responsive genes in both organs (Kozuka et al., 2010). However, the *doc1/big* or *rot3* mutants that affect the petiole response, retain normal leaf-blade responses to EODFR (Kozuka et al., 2010).

Hyponastic leaf movement

When Arabidopsis plants are grown in dense canopies, the leaves adopt a more erect position (Ballaré and Scopel, 1997; Djakovic-Petrovic et al., 2007). This response is rapid and can be readily appreciated in a few hours (Faigón-Soverna et al., 2006). Both, lowering the R:FR (Vandenbussche et al., 2003; Tao et al., 2008; Keuskamp et al., 2010) or irradiance (Vandenbussche et al., 2003; Mullen et al., 2006) cause leaf hyponasty and lowering both has a stronger effect (Sasidharan et al., 2010). The re-

sponse to irradiance is gradual at least between 5 and 200 $\mu\text{mol m}^{-2} \text{s}^{-1}$, can easily be detected 2 h after the reduction in irradiance and reaches a maximum after 16 h (Millenaar et al., 2009). The *phyB* mutant has hyponastic leaves in the absence of shade and retains only a minor response to dense canopies (Ballaré and Scopel, 1997) and a delayed response to reduced irradiance (Millenaar et al., 2009). Lowering specifically blue light causes leaf hyponasty. The *cry1* mutant may present slightly hyponastic leaves that do not respond to low blue light (Keller et al., 2011) but this hyponastic phenotype is not always observed (Ballaré and Scopel, 1997; Mullen et al., 2006).

The hyponastic response to low R:FR is severely reduced in *sav3* (Tao et al., 2008; Moreno et al., 2009) and *pin3* mutants (Keuskamp et al., 2010). The hyponastic response to lowering irradiance is strongly reduced in wild-type plants treated with the polar auxin transport inhibitor, TIB, and in the *tir1*, *tir1 afb1 afb2 afb3*, *pin3*, *pin7*, *tir3*, *axr2* or *axr3* mutants (Vandenbussche et al., 2003; Millenaar et al., 2009), indicating that the response to low R:FR or low irradiance requires intact auxin signaling. Conversely, the response to lowering blue light is conserved in the *sav3*, *pin3*, quintuple *yucca*, quintuple *della*, *gai1* and *bri1* mutants and partially attenuated in the *pif4* and *pif5* and *pif4 pif5* mutants (Keller et al., 2011). There are conflicting results regarding the role of ethylene (Vandenbussche et al., 2003; Millenaar et al., 2009).

BRANCHING

Plants of *Arabidopsis* either of the *phyB* mutant or of the wild type grown under low R:FR produce less branches than plants of the wild type grown under high R:FR (Reed et al., 1993; Finlayson et al., 2010). This indicates that high R:FR perceived by *phyB* promote branching compared to low R:FR. Branching involves a series of steps that begin with the generation of the leaves and the generation of the buds in the leaf axils. Low R:FR-treated plants and *phyB* mutants grown under high R:FR cause accelerated flowering and therefore reduce the number of leaves. However, these plants reduce branching more than expected based on their reduction in leaf number as indicated by the number of branches standardized to a leaf number basis (Finlayson et al., 2010). Similarly, low R:FR and the *phyB* mutation cause some reduction in the number of buds per leaf but this effect does not account for the effects on branch numbers, because buds are always in excess of branches. Therefore, phytochrome affects branching mainly by modulating the bud outgrowth process (Finlayson et al., 2010). In turn, the effect of *phyB* on bud outgrowth has two opposite components. On the one hand *phyB* opposes the correlative inhibition (i.e. the inhibition imposed by one organ on the growth of another organ) resulting from the influence of the main shoot and other branches. On the other, *phyB* represses stem growth (see previous sections of this chapter) and therefore, low R:FR can promote the growth of some branches (Finlayson et al., 2010).

There is a correlative control of branching by signals received from more or less remote parts of the plant. Auxin produced in young expanding leaves at the shoot apex is transported basipetally down the stem and indirectly inhibits shoot branching, establishing apical dominance (Leyser, 2005). Strigolactones are synthesized in both the roots and the shoots and are transport-

ed acropetally, presumably in the xylem, to repress bud activity (Domagalska and Leyser, 2011). The control of bud outgrowth by *phyB* perception of low R:FR is impaired in *axr1*, *more axillary branches 2 (max2)* and *max4* (Domagalska and Leyser, 2011) mutants, indicating that this control requires intact auxin and strigolactone signaling (Finlayson et al., 2010). Domagalska and Leyser (2011) have proposed that *phyB* could theoretically exert part of its action on branching by altering strigolactone signaling and polar auxin transport.

In addition to the correlative or systemic regulation of branching there is a local control. The TCP-domain transcription factor genes *BRANCHED 1 (BRC1)* and *BRC2* are expressed in the buds and suppress bud outgrowth (Aguilar-Martínez et al., 2007; Finlayson, 2007). The control of branching by *phyB* and R:FR is impaired in the *brc1* and *brc2* mutants (Finlayson et al., 2010). When mRNA levels are analyzed in unelongated primary rosette buds at two different positions of plants of the wild type grown under high or low R:FR and plants of the *phyB* mutant grown under high R:FR, the expression of *BRC1* and *BRC2* correlate with different genes, indicating that they are part of different gene networks (Finlayson et al., 2010).

In *Arabidopsis* the effects of the white light irradiance are relatively small and increasing irradiance levels can either increase or decrease branching (Buchovsky et al., 2008; Su et al., 2011). High levels of PAR reduce the impact of the *phyB* mutation on branching (Su et al., 2011).

FLOWERING

Arabidopsis plants exposed to natural (Pigliucci and Schmitt, 1999) or simulated (Sánchez et al., 2011) shade light conditions (low irradiance, low R:FR) flower after producing less leaves than sunlight controls. Lowering the R:FR accelerates flowering and under high R:FR the *phyB* mutant flowers earlier than the wild type, while the *phyB phyD* and *phyB phyE* mutants flower earlier than the *phyB* mutant (Devlin et al., 1998; Devlin et al., 1999). The effects of the *phyB* and *phyB phyD* mutations involve increased expression of *FLOWERING LOCUS T (FT)* (Cerdán and Chory, 2003; Halliday et al., 2003). The FT protein is produced in the leaves in response to diverse flowering stimuli and migrates to the apex where it promotes the transition from the vegetative to the reproductive stage (Corbesier et al., 2007). In contrast to R:FR, irradiance levels have weak effects on the timing of flowering measured on a biological scale (i.e., leaf number) (Buchovsky et al., 2008; Zhao et al., 2011). Therefore, under shade, the low R:FR perceived primarily by *phyB* and secondarily by *phyD* and *phyE* induce *Arabidopsis* flowering at an earlier developmental stage.

The expression of *FT* depends on the balance between the repression mediated by *FLOWERING LOCUS C (FLC)* and that promotion mediated by *CONSTANS (CO)*, both of which act at the *FT* promoter itself (Li et al., 2008; Adrian et al., 2010). Extended periods of low temperatures (vernalization) reduce the expression of *FLC* (Michaels and Amasino, 1999) and allow the induction of flowering by long days. Low R:FR or *phyB phyD phyE* mutations accelerate flowering even in lines with constitutive high expression of *FLC* indicating that shade light signals can bypass the requirement of vernalization (Wollenberg et al., 2008).

In the photoperiodic pathway of flowering induction, the expression of *FT* in the leaves is induced by the coincidence between the presence of light and the expression of *CONSTANS*, which is controlled by the clock and peaks at night (Suarez-Lopez et al., 2001; Yanovsky and Kay, 2002). This coincidence occurs close to the end of the photoperiod under long days and is accounted for by the stabilization of the CO protein by light perceived by *phyA*, *cry2* and *cry1* (Valverde et al., 2004). Low R:FR increase the expression and activity of CO at dawn and dusk (Wollenberg et al., 2008). Active *phyB* reduces the stability of CO (Valverde et al., 2004) and this could account for the enhanced activity of CO under low R:FR. Therefore, shade light signals could partially act by enhancing the photoperiodic pathway and in accordance with this idea low R:FR has little effect under short days (Wollenberg et al., 2008). However, it is clear that not all the effects of lowering active phytochrome levels can be accounted for by enhancing the photoperiodic pathway. For instance, the *phyA phyB phyE co* mutant flowers earlier than the *phyA phyB co* (Halliday et al., 2003). Simulated shade (low irradiance, low R:FR) shows a stronger acceleration of flowering under short than under long days (Sánchez et al., 2011) and the *phyB* (Reed et al., 1993; Halliday et al., 1994) or *phyB phyD phyE* (Wollenberg et al., 2008) mutants flower early under short days. Early flowering of the *phyB* mutant or in response to EODFR requires PHYTOCHROME AND FLOWERING TIME 1 (PFT1), which is not required for the photoperiodic response (Cerdán and Chory, 2003). PTF1 is the MED25 subunit of the plant Mediator complex and promotes flowering through CO-dependent and independent mechanisms (Iñigo et al., 2011). The relative importance of each way of action could depend on the context. As a matter of fact, the acceleration of flowering by low R:FR or by the *phyB*, *phyD* and *phyE* mutations is reduced when the plants are grown at 16 °C instead of 22 °C (Halliday et al., 2003).

Some of the pathways involved in the vegetative shade-avoidance responses also affect the early-flowering response to low R:FR but others do not. For instance, the *hfr1* mutant flowers earlier and has higher *FT* expression levels than the wild type under low R:FR (Sessa et al., 2005). Similarly, natural alleles of *ELF3* regulate the flowering time response to shade (Jiménez-Gómez et al., 2010). A role of natural variation at the *PIF4* locus in the control of flowering time has also been proposed (Brock et al., 2010) but its significance has been questioned (Shin et al., 2009; Leivar and Quail, 2011). However, the *sav3* mutant is affected in vegetative shade-avoidance responses but it flowers at the same time as the wild type (Tao et al., 2008). Conversely, the *doc1/big* mutant suppresses the accelerated flowering of the *phyB* mutant but retains normal hypocotyl EOD responses (Kanyuka et al., 2003).

ACCLIMATION TO SHADE AND SHADE-AVOIDANCE REACTIONS

The aforementioned processes are shade-avoidance reactions because they reduce the degree of current or future shade. Other responses to shade light do not reduce *per se* the degree of shade; rather, they help to optimize the use of resources under shade. Therefore, they could indirectly contribute to shade-avoidance when the plant or its lower leaves are already shaded.

The photosynthetic apparatus acclimates both in terms of morphology and stoichiometry of its components in response to the changes in irradiance and spectral composition associated to the degree of shade by neighbors. These changes in the light environment are perceived at least in part by the photosynthetic apparatus itself but phytochromes and cryptochromes are also involved (Walters, 2005). EODFR (Casal et al., 1990b), low R:FR (McLaren and Smith, 1978) and the *phyB* mutation (Reed et al., 1996) reduce leaf chlorophyll levels. Low irradiance (Casson et al., 2009) and low R:FR (Boccalandro et al., 2009) perceived by *phyB* reduce stomata density. This in turn reduces transpiration and maximum photosynthesis but increases water-use efficiency (Boccalandro et al., 2009). Cryptochromes are also involved in the control of stomata development (Kang et al., 2009) and indirectly regulate leaf conductance by reducing the levels of abscisic acid (Boccalandro et al., 2011).

Exposure to low R:FR and the *phyB* mutation increase the vulnerability of Arabidopsis plants to insect herbivores at least in part by reducing the sensitivity to jasmonic acid (Moreno et al., 2009; Ballaré, 2011). The expression of many plant disease resistance genes is modulated by the activity of phytochromes (Devlin et al., 2003). The growth of an incompatible strain of *Pseudomonas syringae* is enhanced in the *phyA phyB* double mutant (Genoud et al., 2002) and *P. syringae* pv. tomato (Pst.) DC3000 proliferates more abundantly in the *cry1* mutant of Arabidopsis than in the wild type (Wu and Yang, 2010). The reduced investment in defense would release resources for shade-avoidance reactions. The *constitutive shade-avoidance1 (csa1)* mutant shows shade-avoidance responses in the absence of shade signals (including elongated hypocotyls and petioles, hyponastic leaves, early flowering) and enhanced expression of shade-response marker genes like *HAT4* and *HFR1*. The *csa1* phenotype is caused by the expression of a truncated version of a *TOLL/INTERLEUKIN1 RECEPTOR–NUCLEOTIDE BINDING SITE–LEUCINE-RICH REPEAT (TIR-NBS-LRR)* gene (Faigón-Soverna et al., 2006). TIR-NBS-LRR proteins have been implicated in defense responses in plants and *csa1* shows enhanced growth of a bacterial pathogen (Faigón-Soverna et al., 2006). Therefore, CSA1 provides a molecular link between shade-avoidance and defense responses.

SHADE-AVOIDANCE RESPONSES IN CROPS AND WEEDS

The promotion of stem growth by low R:FR and/or EODFR has been demonstrated in many crop species including beans (Downs et al., 1957), mustard (*Sinapis alba*) (Morgan et al., 1980), tobacco (*Nicotiana tabacum*) (Kasperbauer, 1971), sunflower (*Helianthus annuus*) (Libenson et al., 2002), tomato (*Solanum lycopersicon*) (Selman and Ahmed, 1962) and cucumber (*Cucumis sativus*) (Ballaré et al., 1991c), and weeds such as *Chenopodium album* (Morgan and Smith, 1976) and *Datura ferox* (Ballaré et al., 1987). The response to reduced blue light or reduced red plus far-red light has also been demonstrated for instance in tobacco (Casal and Sánchez, 1994), tomato (Casal, 1994), *Sinapis alba*, and *Datura ferox* (Ballaré et al., 1991b). There are large quantitative differences among species in the extent of response to low R:FR, which are highly significant in species from open habitats such as *Senecio vulgaris* and *Chenopodium album* and hardly detectable

in species native of shaded woodland habitats such as *Mercurialis perennis* (Morgan and Smith, 1979). Despite the fact that the promotion of stem extension growth by shade light signals is arguably the most conspicuous shade-avoidance response, there are clear exceptions to this rule. In wheat, for instance, the lowermost internodes are very short and show some promotion of extension under low R:FR but the uppermost internode or peduncle, which bears the ear, shows reduced rather than enhanced growth in response to low R:FR (Casal, 1993; Ugarte et al., 2010). It is interesting to note that grain yield in sunflower can be reduced by promoting stem growth by selectively lowering the R:FR reaching the stem, suggesting an indirect effect of shade-avoidance responses on yield (Libenson et al., 2002), while wheat plants show a direct effect of low R:FR on grain yield in the absence of changes in plant stature (Casal, 1993; Ugarte et al., 2010).

Reduced branching in response to low R:FR and/or EODFR is another shade-avoidance response that has been observed in many crop species including tobacco (Kasperbauer, 1971) tomato, where EODFR has been suggested as a replacement for manual pruning in commercial crops (Tucker, 1975), wheat (*Triticum aestivum*) (Casal, 1988), barley (Skinner and Simmons, 1993), and forage grasses such as *Lolium multiflorum* (Deregibus et al., 1983). The enrichment of red light beneath the canopy by means of red-light emitting diodes directed towards the base of the plant has been shown to promote tillering of *Paspalum dilatatum* and *Sporobolus indicus* in natural grasslands (Deregibus et al., 1985). The *phyB* mutant of sorghum (*Sorghum bicolor*) and wild-type plants exposed to low R:FR show reduced bud outgrowth, and enhanced bud expression of the *SbTEOSINTE BRANCHED* and *DORMANCY-ASSOCIATED 1 SbDRM1* genes (Kebrom et al., 2006).

Leaf growth responses to low R:FR or EODFR show significant variation among dicotyledonous species ranging from inhibition to promotion (for references see (Casal and Smith, 1989). In grasses like *Lolium multiflorum*, *Paspalum dilatatum* and barley (*Hordeum vulgare* L.), low R:FR or EOD FR (Casal et al., 1987a; Skinner and Simmons, 1993) and low blue light (Casal and Alvarez, 1988) promote leaf sheath growth, placing leaf lamina at a higher stature. In some maize cultivars, the leaves grow away from the low R:FR signals of neighbors, reducing mutual plant shading in crops (Maddonni et al., 2002). The increased leaf senescence under reduced PAR and R:FR is a response to shade light observed in crop species like sunflower (Rousseaux et al., 1996) that has received little attention in *Arabidopsis thaliana*.

Accelerated flowering in response to shade light signals is not observed in many crop species. Low R:FR accelerate flowering in barley (Deitzer et al., 1979) and *Lolium multiflorum* (Casal et al., 1985). In wheat, EODFR can affect the timing of anthesis as a result of changes in the rate of development of the reproductive structures without apparent changes in the time of apex transition to the reproductive stage revealed by differences in leaf number (Casal, 1993; Ugarte et al., 2010).

Defense against biotic agents is also reduced by shade light signals in crop species. The *phyB* mutation in tomato increases susceptibility to insect herbivores (Izaguirre et al., 2006). The rice *phyA phyB phyC* triple mutant shows reduced expression of pathogenesis-related class 1 (PR1) proteins and enhanced susceptibility to blast fungus (*Magnaporthe grisea*) (Xie et al. 2011). The symbiosis between legumes and rhizobia provides a different pattern of light effects on biotic relationships. The *phyB* mutant of

Lotus japonicus and wild-type plants exposed to low R:FR show reduced nodule development after *Mesorhizobium loti* inoculation (Suzuki et al., 2011). This is caused by a shoot-derived signal which involves jasmonic acid.

CONCLUDING REMARKS

As sessile organisms, plants have evolved mechanisms to evade or alleviate the detrimental consequences of the prevailing environmental conditions, including the limitations imposed by shade and the consequent reduced availability of PAR. This involves wiring environmental signals perceived by photoreceptors to the endogenous signals and the structural components controlling growth and development (e.g. the enzymes that modulate cell-wall extensibility). Therefore, it is not unexpected to see changes in selected hormone signaling components in response to shade light signals. Auxin plays a dominant role but at least gibberellins, brassinosteroids, cytokinins and ethylene are also important. PIF proteins, discovered in the phytochrome field, are now considered hubs controlling different growth and developmental responses, and play a key role in shade-avoidance responses. This chapter presents a separate analysis of the various shade-avoidance responses to demonstrate that the significance of the different molecular and cellular events is dependent on the developmental context and on the environment (temperature, for instance, can change even the direction of a growth or developmental response). Fuller understanding of this complexity will lead us to learn how plants optimize their function dealing with the fluctuating environment they have to face.

ACKNOWLEDGMENTS

I thank Dr Santiago Trupkin and Mr Tomás E. Casal for their help with the figures. Work in my lab is supported by Agencia Nacional de Promoción Científica y Técnica (ANPCYT), Universidad de Buenos Aires, Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET) and International Centre for Genetic Engineering and Technology (ICGEB).

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