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Update on Light Signaling

Shade Avoidance Responses. Driving Auxin along Lateral Routes[1]

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To grow and develop optimally, all organisms need to perceive and process information from their environment. As sessile organisms, plants need to sense and respond to external stimuli more than most organisms. Therefore, plants have to adapt their developmental pattern to the environmental changes to ensure survival and reproduction. As a consequence, numerous environmental factors, including temperature, touch, water, gravity, and light, can exert a profound influence on the form assumed by individual plants, affecting overall plant size and the number and size of individual organs.

Cues from the light environment are involved in the regulation of seed germination, the establishment of seedlings, the determination of growth habit, and the transition to flowering. To perceive information about their light environment, plants have evolved at least three families of photoreceptors that specifically recognize different wavelengths of light: the red (R)/far-red (FR)-sensing phytochrome family, the blue/UV-A photoreceptors called cryptochromes, and the UV-B photoreceptors (Kendrick and Kronenberg, 1994).

The phytochromes are chromoproteins that exist in two photo-interconvertible isoforms: Pr, a red-light-absorbing form, and Pfr, a far-red-light-absorbing form. They are large proteins of approximately 120 kD that exist as dimers in solution. Each monomer folds into two major structural domains separated by a short protease-sensitive region. The NH_2-terminal domain, with a covalently attached tetrapyrrole chromophore, is sufficient for photosensory activity, while the COOH-terminal domain contains regions necessary for dimerization and regulatory activity (Quail, 1997).

A fundamental function of phytochromes is the perception of changes in the light quality occurring within a plant canopy. As a plant canopy grows and fills up space, a reduction in the ratio of R:FR light occurs because FR light is filtered through or reflected by vegetation. Plants have sophisticated sensing mechanisms operating through the phytochromes that perceive the R:FR ratio as an accurate indicator of neighbor proximity, and trigger morphological changes to avoiding shade. In evolutionary terms, the ability to avoid shade appears to be a relatively recent invention, since it is predominantly found in the angiosperms (Smith, 1995).

Responses to shade are many and varied. The most dramatic shade avoidance response is the stimulation of elongation growth (Fig. 1). This response is remarkably rapid, with a lag phase of a few minutes, and it is reversible. Many plants react within 5 to 10 min of exposure to FR-rich light by accelerating extension up to 3- or 4-fold. Conversely, returning plants to R-rich light results in an equally rapid deceleration of extension. Elongation responses are most easily observed in internodes, but hypocotyl and petioles also show strong responses. In dicotyledonous plants, elongation growth induced by FR-rich light is often associated with a reduction of leaf development, a marked strengthening of apical dominance, and reduction in branching (Fig. 1). Moreover, very important responses to canopy shade are an acceleration of flowering and a reduction of resources for storage and reproduction. This is associated with reduced seed set, truncated fruit development, and often a reduction in the germinability of the seeds produced (Smith, 1995; Smith and Whitelam, 1997).

An inverse quantitative relationship between stem extension rate and estimated phytochrome photoequilibrium has been shown for both shade-avoiding and shade-tolerant herbaceous plants. This implies that the shade-tolerant species are able to perceive the low R:FR ratio, but their responses are very much reduced compared with those of the shade avoiders. This also leads to the conclusion that only plants evolutionarily adapted to live in open habitats acquired an effective genetically determined mechanism to react to shade (Smith, 1995).

All of the shade avoidance responses have been seen both in natural shade and in low R:FR simulations. Furthermore, similar responses are induced by exposing plants to horizontal FR radiation with white light from above. This is consistent with the notion that "shade-avoiding" plants are able to perceive light reflected by neighboring plants as partially depleted of the red wavelengths, and to avoid shade they respond morphologically even before canopy closure and actual shading occurs (Smith and Whitelam, 1997).

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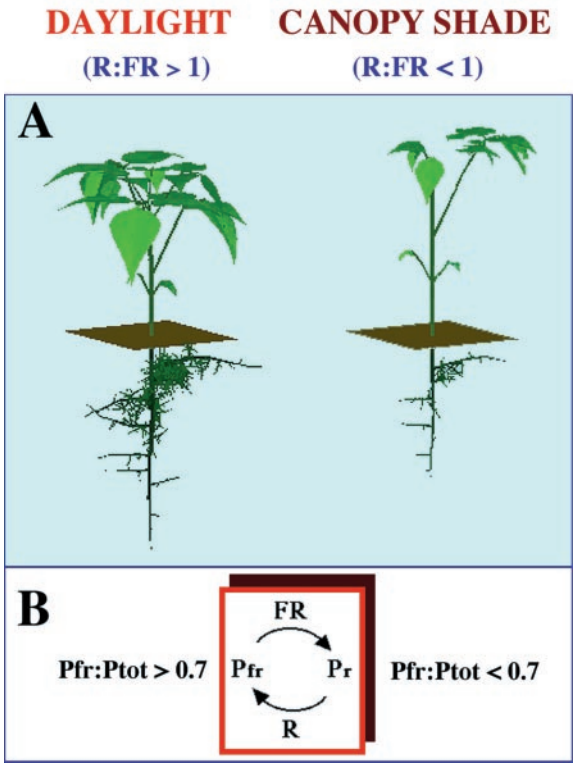

Figure 1. A simplified schematic representation of shade avoidance responses in plants. The phytochrome-active light in daylight and canopy shade is given as the R:FR ratio. A, Morphology of a typical "shade-avoiding" plant grown in sunlight (left) and in shade (right) environments. B, Interconversion of the two forms of phytochrome. Pr, A R-light-absorbing form is converted by R light to a FR-light-absorbing form called Pfr. The Pfr form, in turn, can be converted back to Pr by FR light. The relative ratio of Pfr to total phytochrome (Pfr:Ptot) is indicated in open (left) and shade (right) environments (Smith, 1982).

Several recent and excellent reviews have discussed physiological and ecological details of shade avoidance responses (Schmitt, 1997; Ballaré, 1999). This *Update* emphasizes some recent progress on the identification of molecular components regulating these responses, mainly derived from the study of conventional and transgenic photomorphogenic Arabidopsis mutants.

THE SENSORS OF ENVIRONMENTAL R AND FR LIGHT IN ARABIDOPSIS

In all higher plants studied, the phytochromes are encoded by a small family of divergent genes. Phylogenetic analyses suggests that there are three major types of phytochromes in angiosperms: PHYA, PHYB, and PHYC, which are encoded by the *PHYA*, *PHYB*, and *PHYC* genes, respectively. These genes apparently arose from gene duplications that occurred at about the time of the origin of seed plants and near the time of origin of flowering plants. In dicots, additional *PHY* genes are found, perhaps the products of more recent duplications within the *PHYB* lineage (Mathews and Sharrock, 1997).

In Arabidopsis, the phytochrome apoproteins are encoded by five genes called *PHYA* to *PHYE*. *PHYE* is thought to have originated from a duplication within the *PHYB* lineage early on in the evolution of dicots. The *PHYD* gene, which encodes a protein that shares approximately 80% amino acid sequence identity with PHYB, apparently arose from a relatively recent gene duplication within the Brassicaceae (Mathews and Sharrock, 1997).

Molecular and genetic analysis of light-insensitive mutants has allowed the identification of *phyA* and *phyB* (Fankhauser and Chory, 1997; Fig. 2). Moreover, the identification of a naturally occurring mutation in the Arabidopsis *PHYD* gene has been described (Aukerman et al., 1997). Finally, the isolation of a *phyE* mutant in Arabidopsis has been recently reported (Devlin et al., 1998). No mutants in phytochrome C have yet been identified.

Seedlings of mutants lacking PHYB display a marked insensitivity to R light for many responses during the de-etiolation process, including the inhibition of hypocotyl elongation and the opening and expansion of cotyledons (Fankhauser and Chory, 1997; Fig. 2). The analysis of the phenotypic effects of deficiency for phytochrome D has revealed a minor role of PHYD in R light sensing, which is additive with the much larger role of PHYB in this response (Aukerman et al., 1997). *phyA* mutants are essentially blind to FR light and exhibit a typical etiolated phenotype similar to that of dark-grown seedlings (Fankhauser and Chory, 1997). It has been proposed that PHYA could be important for seedling development under dense canopies. Consistent with this proposal is the observation that de-etiolation of Arabidopsis *phyA* mutants was severely impaired compared with wild-type plants if grown in deep canopy shade, which led to premature death. However, the de-etiolated seedling rapidly loses the contribution of PHYA, because its coding gene is down-regulated by light and the PHYA molecule is light labile. As a consequence, the role of

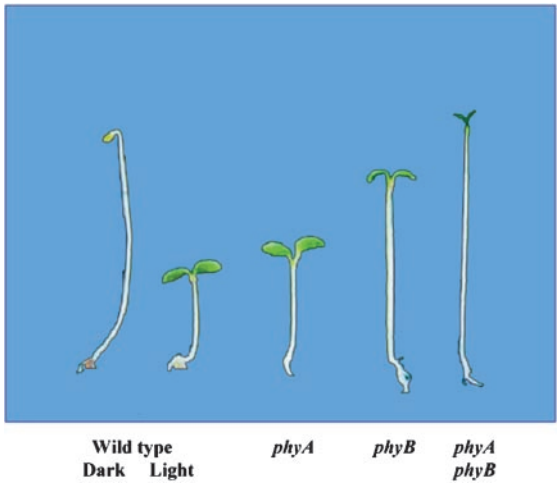

Figure 2. Photomorphogenesis in *phyA*, *phyB*, and *phyA phyB* Arabidopsis mutants. Side view of dark- and light-grown wild-type seedlings and of light-grown *phy* mutants. Note the elongated hypocotyl and the reduction of cotyledon expansion in *phyB* and *phyA phyB* mutants.

light-stable phytochromes soon becomes predominant in controlling the elongation growth within a plant canopy.

ROLE OF ARABIDOPSIS PHYTOCHROMES IN SHADE AVOIDANCE RESPONSES

Arabidopsis is a typical "shade avoiding" plant. For example, when grown in low R:FR ratios, Arabidopsis displays a reduction of cotyledon and leaf expansion and an increased elongation of hypocotyl and petioles (Fig. 3). Despite initial suggestions that the shade avoidance response is triggered by a single member of the phytochrome family, the study of Arabidopsis mutants deficient in one or more phytochromes has revealed that multiple phytochromes are involved.

Arabidopsis *phyB* mutants show phenotypes reminiscent of the shade avoidance responses that are induced in wild-type seedlings by FR-rich light, i.e. they display a constitutively elongated phenotype (Fig. 2) and are early flowering. However, *phyB* null mutants also show typical shade avoidance responses to supplementary FR light given during the photoperiod. In fact, although already elongated, they show increased elongation growth responses to FR-rich light. Moreover, the *phyB* mutant plants grown in simulated vegetational shade flower earlier than *phyB* plants grown in normal light conditions. These observations indicate that PHYB is not the sole photoreceptor involved in the regulation of the shade avoidance response (Smith and Whitelam, 1997).

The analysis of *phyA* mutants and *phyA phyB* double mutants has suggested that during seedling establishment the action of PHYA in plants exposed to FR-rich light antagonizes that of PHYB (and other light-stable phytochromes) in the regulation of hypocotyl elongation. Nevertheless, once the seedling is established, PHYA seems to have little if any role in the shade avoidance response.

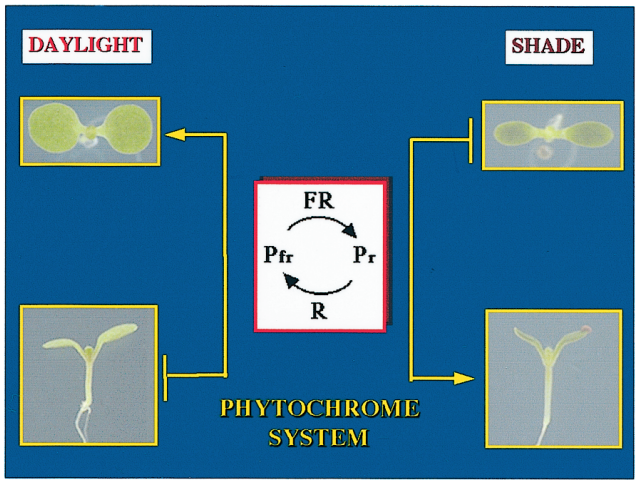

Figure 3. Morphology of Arabidopsis seedlings grown under daylight and shade. The phytochrome system inhibits hypocotyl elongation (side view) and stimulates cotyledon expansion (top view) in daylight (left panels). Conversely, the phytochrome system stimulates hypocotyl elongation and inhibits cotyledon expansion in shade (right panels).

Consistently, it has been observed that *phyA phyB* double mutants still respond to FR-rich light, indicating that one or more of PHYC, PHYD, or PHYE are involved in the regulation of the shade avoidance response (Smith and Whitelam, 1997).

phyD and *phyE* monogenic mutants are essentially indistinguishable from wild-type seedlings. However, *phyB phyE* and, to a lesser extent, *phyB phyD* double mutants flower earlier and had longer petioles than do *phyB* mutants. This led to the proposal that in conjunction with PHYB, PHYD and PHYE function in the regulation of shade avoidance responses (Aukerman et al., 1997; Devlin et al., 1998, 1999).

No mutants in phytochrome C have yet been identified, and the role, if any, of PHYC in shade avoidance responses remains to be investigated.

DOWNSTREAM COMPONENTS IN ARABIDOPSIS SHADE AVOIDANCE RESPONSES

A major goal is the dissection of the phytochrome signal transduction pathways by which FR-rich light perception is coupled to the changes in gene expression that underlie the growth and developmental responses. Genetic screens for Arabidopis mutants defective in PHYB or in both PHYA and PHYB transduction pathways have identified some candidate downstream components (Batschauer, 1999). Furthermore, Quail and colleagues recently identified a basic helix-loop-helix transcription factor, PIF3, which specifically interacts with PHYA and PHYB (Ni et al., 1998). Strong support for the functional importance of PIF3 in phytochrome signaling in vivo is provided by the discovery that a T-DNA insertion in the promoter region of the *PIF3* gene (*poc1* mutant) leading to overexpression of this gene in R light causes enhanced R-light-mediated responses such as short hypocotyl and expanded cotyledons (Halliday et al., 1999). Conversely, PIF3-antisense plants have reduced red-light-mediated responses with a phenotype reminiscent of plants grown in FR-rich light (i.e. long hypocotyl, reduced cotyledon expansion, and early flowering). The reciprocity of the effects in overexpressing and antisense plants implies that PIF3 is an important component of the PHYB signal transduction pathways (Ni et al., 1998; Fig. 4).

Finally, molecular studies have yielded the first genes (*ATHB-2*, also known as *HAT4*, and *ATHB-4*) specifically and reversibly regulated by changes in the R:FR ratio in green plants (Carabelli et al., 1993, 1996). ATHB-2 and ATHB-4 are members of a large class of proteins characterized by the presence of a homeodomain closely linked to a Leu zipper motif unique to higher plants (Ruberti et al., 1991). DNA-binding studies and transient expression assays demonstrated that HD-Zip proteins act as transcription factors (Sessa et al., 1993; Aoyama et al., 1995; Steindler et al., 1999).

ROLE OF ATHB-2 IN SHADE AVOIDANCE RESPONSES

Expression studies have shown that the light regulation of the *ATHB-2* gene is quite complex, involving at least

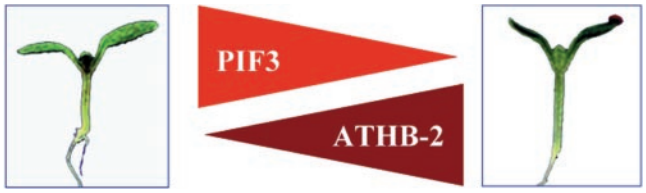

Figure 4. Effects of altered expression of ATHB-2 and PIF3 transcription factors on Arabidopsis development. Side view of transgenic Arabidopsis seedlings with reduced ATHB-2 levels or elevated PIF3 levels (left panel) and with elevated ATHB-2 levels or reduced PIF3 levels (right panel). In the wild-type plants, the antagonistic functions of PIF3 and ATHB-2 are regulated by the R:FR ratio as the ATHB-2 expression is strongly induced by FR-rich light (Carabelli et al., 1996), and PIF3 mediates the response of phytochrome B to red light (Ni et al., 1998).

three distinct phytochromes. In etiolated seedlings, the gene is expressed at relatively high levels and is down-regulated by R or FR light. PHYA is responsible for the rapid down-regulation of *ATHB-2* by a FR pulse, while a phytochrome other than A or B is responsible for the equally rapid down-regulation of *ATHB-2* by a R pulse. In young seedlings and mature plants, *ATHB-2* is expressed at low levels under R-rich light, but is rapidly and strongly induced by FR-rich light. Returning the plants to R-rich light results in an equally rapid decrease in the *ATHB-2* mRNA levels. Kinetics of FR-rich light induction and its reversibility by R-rich light performed in *phyB* and *phyA phyB* plants revealed that the *ATHB-2* gene is reversibly regulated by changes in the R:FR ratio largely through the action of a phytochrome other than A or B and secondarily by phytochrome B (Carabelli et al., 1996; Steindler et al., 1997).

Analysis of transgenic plants bearing constructs that alter *ATHB-2* expression revealed a series of interesting developmental phenotypes (Schena et al., 1993; Steindler et al., 1999). For example, seedlings overproducing ATHB-2 had longer hypocotyls and petioles and smaller and fewer leaves (Fig. 4). Moreover, these seedlings also had a thinner root mass, producing fewer lateral roots than wild-type controls. Conversely, seedlings with reduced levels of ATHB-2 had shorter hypocotyls, larger and more numerous leaves, and a thicker root mass than the wild type (Fig. 4). The phenotypes of adult transgenic plants were similar to seedlings but more exaggerated. Altogether, the phenotypes of plants overexpressing ATHB-2 were reminiscent of those displayed by wild-type plants germinated and grown in FR-rich light, and were even more severe than those observed in *phyB* mutants grown in R-rich light (Smith and Whitelam, 1997). Together with the tight regulation of the *ATHB-2* gene by the phytochrome system (R:FR ratio), these data imply a major role for this HD-Zip protein in the regulation of the shade avoidance response (Carabelli et al., 1996; Steindler et al., 1999).

Remarkably, anatomical studies in the hypocotyl of transgenic plants with reduced or elevated levels of ATHB-2 indicated that the alteration of elongation growth was the result of major changes in both the orientation of cell expansion and the production of the secondary vascular tissue. Plants with reduced levels of ATHB-2 showed shorter epidermal and cortical cells, while the proliferation of secondary vascular tissue was found to be strongly increased compared with wild-type plants. On the contrary, the elongated phenotype in the ATHB-2-overexpressing plants was found to be the consequence of the same two events but in the opposite direction: a change in the orientation of cell expansion toward elongation in cells that do not divide, as the epidermal and cortical cells in the hypocotyl, and the inhibition of secondary cell proliferation. Similar changes have been observed in wild-type seedlings grown in environmental light conditions simulating canopy shade, indicating that FR-rich light produces (through the action of ATHB-2) distinct but coordinated effects on different cell types (Steindler et al., 1999).

CROSS-TALK BETWEEN LIGHT AND AUXIN SIGNAL TRANSDUCTION PATHWAYS IN ARABIDOPSIS SHADE AVOIDANCE RESPONSES

To maximize the capability of an organ to expand or elongate, as in the shade avoidance response, plants must have evolved mechanism(s) tightly coupling distinct developmental processes: cell proliferation, cell differentiation, and direction of cell expansion. Many of these processes are dependent on the action of phytohormones, and considerable evidence is accumulating that light and plant hormones are intertwined. Several genes (*DET2*, *CPD*, and *DWF4*) involved in the control of subsets of seedling photomorphogenic responses encode enzymes that function in brassinosteroid biosynthesis, suggesting a role for brassinosteroids in light-dependent development (Li et al., 1996; Szekeres et al., 1996; Choe et al., 1998). Gibberellin biosynthesis seems to be regulated by the phytochrome system during seed germination, seedling growth, and photoperiodic induction of flowering (Kamiya and Garcia-Martinez, 1999). Furthermore, the characterization of HY5, a signaling pathway component that appears to lie at or downstream of the convergence of the cryptochrome and phytochrome transduction pathways, and SHY2, identified in a screen for suppressors of a phytochrome-deficient mutation (*hy2*), strongly suggest a link between light and auxin (Oyama et al., 1997; Tian and Reed, 1999).

Among plant phytohormones, auxin might act as a coordinator of growth across an organ, because it regulates many different aspects of plant development, including cell division, cell elongation, cell differentiation (e.g. vascular tissue), and patterning. Auxin is synthesized in young leaves of the shoot system and transported downward to the root tip through the vasculature. It has been shown that the transport of auxin in any given tissue is polar (i.e. it moves in one direction). A clear example of this polar transport is shown by cambium, the lateral meristem that gives rise to secondary vascular tissues.

Tropistic growth such as phototropism and gravitropism involves asymmetric elongation growth in response to a specific stimulus. It has been proposed that light and gravitropic stimuli cause a directional transport of auxin, creating an asymmetric distribution. This auxin gradient would differentially affect rapid elongation growth, result-

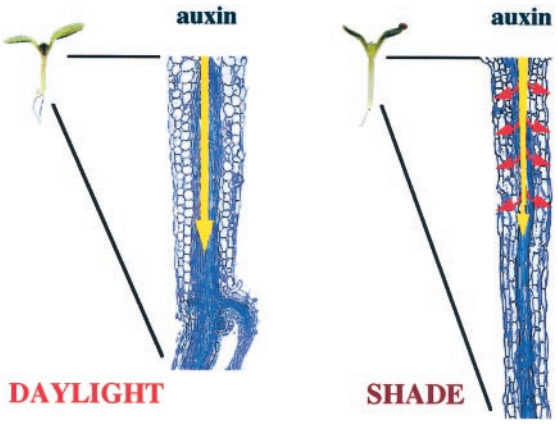

Figure 5. Working model depicting the role of auxin in seedling development under daylight and shade. The model postulates that auxin synthesized in the shoot is transported to the root through the vasculature (yellow arrows). In the shade, the auxin is redistributed laterally to epidermal and cortical cells of the hypocotyl (red arrows) producing the elongation of these two tissues. The lateral distribution of auxin also causes a net decrease of the auxin transported through the developing vascular system, resulting in a let up in vascular differentiation and a decrement in auxin concentration reaching the root. This, in turn, might result in a reduction of lateral root formation and, eventually, primary root growth.

ing in the observed asymmetric growth or curvature of an organ (Lehman et al., 1996; Chen et al., 1999). The formation and maintenance of auxin gradients is thought to occur through the action of a specific polar auxin-transport system that requires active efflux of auxin. Recently, a family of efflux carrier proteins have been identified in Arabidopsis. Two of them, PIN1 and AGR1/EIR1/PIN2, are specifically involved in processes such as vascular development and root gravitropism, respectively, suggesting that distinct auxin efflux carrier proteins are involved in distinct cellular processes (Gälweiler et al., 1998; Chen et al., 1999).

Analogous to the phenomena of phototropism and gravitropism, several recent findings indicated that auxin and auxin transport systems are also important components of the elongation process induced by shade. Consistent with the observation that phytochrome regulation of stem elongation is partly the result of changes in IAA levels (Behringer and Davies, 1992), it was found that the *axr1* mutant, which is severely impaired in the auxin response, does not elongate significantly in FR-rich light. Furthermore, it has been shown that napthylphthalamic acid, an auxin transport inhibitor, significantly reduces hypocotyl elongation of wild-type seedlings in response to FR-rich light (Steindler et al., 1999). These experiments extended recent data demonstrating that auxin transport is required for hypocotyl elongation in light-grown Arabidopsis seedlings (Jensen et al., 1998), and lead to a model for Arabidopsis shade-induced responses.

A FR-rich light regime might produce a reorientation of the auxin transport stream through a spatial redistribution of a specific auxin efflux carrier protein or the activation of regulatory protein(s) controlling specific auxin efflux carrier protein(s) or both. A higher lateral transport of auxin in the hypocotyl of shaded seedlings should result in a net reduction of auxin transported through the developing vascular system. This in turn should produce a let up in vascular differentiation and a decrement in the auxin concentration reaching the root, resulting in a reduction of lateral root formation and, eventually, in a slower growth of the main root (Fig. 5). In support of this hypothesis is the root phenotype of ATHB-2 seedlings. Primary root growth, lateral root formation, and secondary vascular growth are all inhibited by elevated levels of ATHB-2, and at least the lateral root phenotype of ATHB-2 seedlings is rescued by exogenous IAA (Steindler et al., 1999). A change in auxin distribution might be also implicated in the inhibition of cotyledon and leaf expansion by shade, although the phytochrome system (through the action of ATHB-2) might regulate distinct pathways in different organs and, eventually, cell types.

CONCLUSIONS

We have outlined recent advances in understanding the molecular processes that control shade avoidance responses in plants. The involvement of multiple phytochromes in these responses has been established, and various downstream components of the phytochrome transduction pathways have been identified and are being characterized. Some of these (PIF3 and ATHB-2) act as transcriptional regulators, and changes in their levels result in developmentally specific alterations that are reflected in whole plant morphogenesis. The direct (physical) interaction of PIF3 with the phytochrome molecules and the ability of ATHB-2 to affect auxin response pathways provided some insight into the mechanisms underlying the coordination of the overall response in different plant organs. A major challenge for the future will be to understand whether independent antagonistic pathways operate (through PIF3 and ATHB-2, respectively) in the control of shade avoidance responses, or if light signals originating from different photoreceptors are integrated together (through PIF3) in the control of the ATHB-2 gene expression.

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