Light-Mediated Hormonal Regulation of Plant Growth and Development

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Abstract

Light is crucial for plant life, and perception of the light environment dictates plant growth, morphology, and developmental changes. Such adjustments in growth and development in response to light conditions are often established through changes in hormone levels and signaling. This review discusses examples of light-regulated processes throughout a plant's life cycle for which it is known how light signals lead to hormonal regulation. Light acts as an important developmental switch in germination, photomorphogenesis, and transition to flowering, and light cues are essential to ensure light capture through architectural changes during phototropism and the shade avoidance response. In describing well-established links between light perception and hormonal changes, we aim to give insight into the mechanisms that enable plants to thrive in variable light environments.

Contents

1. INTRODUCTION

1.1. Light Is Life

Given that light is essential for plant life, it is not surprising that plants have evolved an array of receptors to monitor the light environment. By perceiving light quantity and quality, plants obtain information not only about whether light is present (and, if so, how much is present), but also about the season (day length), the direction of the light, the presence of competitors, and cues for circadian clock entrainment. This information allows plants to time their development such that they go through different stages of their life cycles under the best environmental circumstances. Furthermore, through their remarkable phenotypic plasticity, plants can adapt their growth and architecture to cope with adverse light conditions.

Plant hormones are essential in the regulation of developmental transitions and growth regulation. Knowledge of plant hormone signaling pathways has increased tremendously in the last

decade, and receptors are now known for all major hormones. Many light responses in plants are mediated through changes in hormonal metabolism and distribution, and the links between light perception and hormonal regulation are being elucidated for many processes.

1.2. Light Perception

Plants have distinct sets of photoreceptors for several parts of the light spectrum, ranging from near-UVB (280–315 nm) to far-red (FR) (~750 nm) wavelengths. There are several types of photoreceptors; with the exception of the UVB receptor, all of them contain a bound cofactor, known as the chromophore, in which photon absorption of particular wavelengths actually takes place (89). Instead of a chromophore, the UVB receptor UVR8 uses specific tryptophan residues from the protein itself for UVB absorption (62).

The phytochromes respond primarily to the red (R) and FR parts of the spectrum (600–750 nm) and are involved in many light-regulated processes (126). Their signaling mechanism is based on conformational changes between the active and inactive photoconvertible isoforms. There are three types of flavin-based blue light (350–500 nm) receptors. The cryptochromes are involved in entrainment of the circadian clock, flowering, and photomorphogenesis (17). The phototropins and the ZEITLUPE family are both light-oxygen-voltage (LOV) domain proteins (19). In addition, the phototropins contain a serine/threonine kinase domain, and light activation leads to enhanced kinase activity, which results in photomovement responses such as phototropism, chloroplast movement, and stomatal opening. Members of the ZEITLUPE family contain an F-box domain and several Kelch repeats, and their regulation of light-mediated protein degradation plays a role in the entrainment of the circadian clock and the onset of flowering. UVB light is perceived by UVR8 (108); upon light activation, the UVR8 homodimer breaks down into monomers, which can subsequently interact with downstream targets.

Some photomorphogenic responses are regulated by specific photoreceptors, whereas others can be induced by several light signals and different types of receptors; coaction or antagonism of photoreceptors have both been described (48, 101). Common downstream signaling components that are targeted by several photoreceptor pathways include CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) and PHYTOCHROME-INTERACTING FACTOR (PIF) proteins (55, 78), which appear to be important points of convergence for different light signaling pathways.

CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1): a ubiquitin E3 ligase that interacts with and regulates the abundance of numerous transcription factors

PHYTOCHROME-INTERACTING FACTORs (PIFs): a family of basic helix-loop-helix transcription factors that interact with active phytochrome and regulate photomorphogenic responses

1.3. The Aims of This Review

In this review, we provide an overview of important events during plant development in which light acts as a source of information or developmental switch, thereby dramatically altering plant growth and architecture. Rather than providing an exhaustive list of all hormones and hormonal interactions that have been related to these processes, we highlight examples for which a link between the perception of a light signal and subsequent hormonal regulation has been best established. With this focus, we hope to give insight into the mechanism by which light regulates hormone signaling. This specific scope does, however, greatly limit the extent to which we can elaborate on each case, and we therefore apologize in advance to colleagues whose research we could not include.

Each section contains (*a*) an introduction describing the biological context and the light-regulated physiological response; (*b*) an overview of the light signal or signals that trigger the process, the photoreceptors that mediate the response, and (if known) where the perception takes

place inside the plant; and (*c*) a detailed description of the light-dependent hormonal regulation involved in the process. Where appropriate, we also briefly introduce and describe the hormonal pathways in sidebars. Because in the last few decades most research in this field has been done on *Arabidopsis thaliana*, we focus mainly on this species.

Phytochrome B (phyB): a photoreceptor activated by red light and inactivated by far-red light

Phytochrome A (phyA): a photoreceptor activated by red and far-red light

2. SEED GERMINATION

2.1. Toward Emergence from the Seed

Nondormant seeds monitor their environment to germinate at the right season and in conditions suitable for seedling establishment and subsequent growth and reproduction. Temperature, moisture, oxygen, and nutrients all affect germination. Light is another important environmental factor that can trigger germination, especially in sun-loving species with small seeds. Germination starts with water uptake, or imbibition, of the dry seed, which restarts many cellular processes, including transcription, protein synthesis, energy metabolism, cellular repair, and cell elongation. Preceded by testa (seed coat) and then endosperm rupture, this eventually leads to the emergence of the embryo, usually the root radicle, through its surrounding structures (10).

2.2. Germination Induced by Light Perception

The discovery that germination can be induced by R light and then reversed by subsequent irradiation with FR light in dark-imbibed lettuce seeds (*Lactuca sativa* L.) was instrumental in the discovery of the photoconvertible phytochromes (11). Later research in *Arabidopsis* established that this process is regulated by phytochrome B (phyB), the main photoreceptor in R light–induced germination. PhyA can induce germination in continuous FR light and at very low fluence rates (114). Interestingly, although shortly after seed imbibition R-induced germination can be inhibited by a pulse of FR through the inactivation of phyB, a second pulse of FR at a later stage of imbibition can induce germination through phyA (114). Using dissected embryos and seed coats, Lee et al. (77) established that phyA- and phyB-dependent induction of germination are spatially separated. The initial phyB-dependent inhibition of germination by FR is mediated in the endosperm and involves an inhibiting abscisic acid (ABA; see sidebar Abscisic Acid) signal to the embryo to prevent phyA-dependent germination. This result was consistent with the endosperm inhibiting germination by release of ABA in dormant seeds (77). The repressive ABA signal from the endosperm weakens over time, and a later pulse of FR induces phyA-dependent germination in the embryo (77). It thus appears that light-induced germination is controlled by the endosperm shortly after imbibition and by the embryo itself at a later stage (**Figure 1*a***).

ABSCISIC ACID

ABA is synthesized from carotenoids. The rate-limiting step in ABA biosynthesis is catalyzed by the 9-*cis*-epoxycarotenoid dioxygenases (NCEDs), and ABA is deactivated by CYP707A enzymes (136). ABA can be perceived in various cellular compartments, of which the cytosolic PYRABACTIN RESISTANCE (PYR/PYL/RCAR) receptors are the best characterized. Binding of ABA to a PYR/PYL/RCAR receptor leads to interaction with a PROTEIN PHOSPHATASE 2C (PP2C)–SNF1-RELATED PROTEIN KINASE 2 (SnRK2) complex, thereby activating SnRK2. This in turn leads to activation of members of the ABF/AREB/ABI5 clade of basic leucine zipper (bZIP) transcription factors in the nucleus through their phosphorylation by SnRK2 (138).

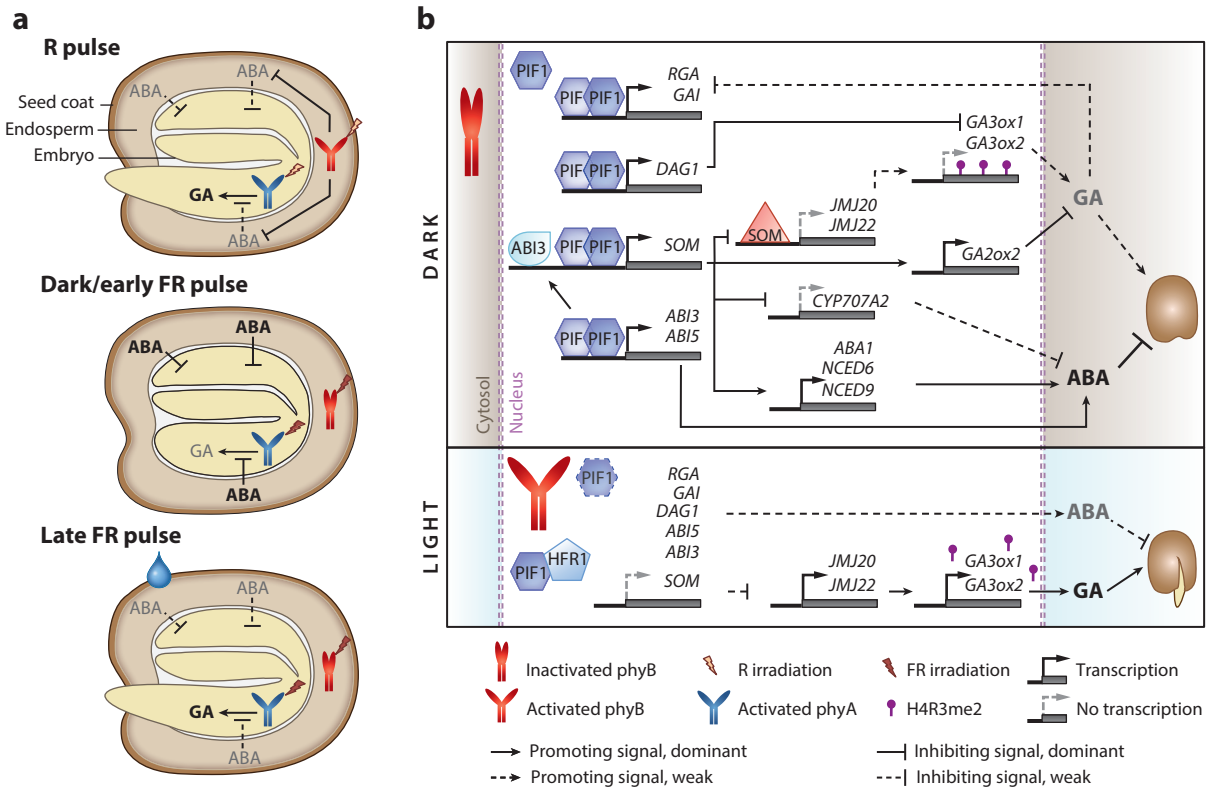

Figure 1

Light-regulated germination signaling. (*a*) Schematic illustrations of an *Arabidopsis* seed. The thickness of the endosperm has been exaggerated to allow graphical representation of signaling elements. (*Top*) Phytochrome A (phyA) and phyB are activated by a pulse of red (R) light, leading to gibberellic acid (GA) biosynthesis and subsequent light-induced germination. (*Middle*) PhyB in the endosperm is inactivated in the dark or by a pulse of far-red (FR) light early during imbibition. PhyB inactivation enables PIF1 accumulation, leading to the biosynthesis of abscisic acid (ABA), which is released toward the embryo. In the embryo, ABA inhibits GA biosynthesis stimulated by phyA, which is activated by both R and FR light. ABA overrides the weaker phyA effect on GA, thereby inhibiting germination. (*Bottom*) The ABA signal from the endosperm weakens over time, and at a later stage of imbibition (≥48 h, represented by the *drop of water*), a pulse of FR leads to a phyA-mediated increase in GA concentration in the embryo, stimulating germination. (*b*) Signaling pathways in the dark and in the light. (*Top*) In the dark, phyB is inactive and resides in the cytosol. This allows PHYTOCHROME-INTERACTING FACTOR 1 (PIF1) to accumulate in the nucleus and regulate transcription of its downstream targets *REPRESSOR OF GA1-3* (*RGA*), *GIBBERELLIC ACID–INSENSITIVE* (*GAI*), *DOF AFFECTING GERMINATION 1* (*DAG1*), *SOMNUS* (*SOM*), *ABSCISIC ACID–INSENSITIVE* 3 (*ABI3*), and *ABI5*. The ABA pathway is stimulated through the signaling genes *ABI3* and *ABI5* and the biosynthesis genes *ABA1*, *9-CIS-EPOXYCAROTENOID DIOXYGENASE 6* (*NCED6*), and *NCED9*, whereas the catabolic gene *CYP707A2* is inhibited. GA accumulation is inhibited by repression of the GA biosynthesis genes *GIBBERELLIN 3-OXIDASE 1* (*GA3ox1*) and *GA3ox2*, whereas the catabolic gene *GA2ox2* is induced. This leads to high ABA concentrations and low GA concentrations, inhibiting germination. (*Bottom*) PhyB is activated by red light and subsequently translocates to the nucleus, where it mediates degradation of PIF1 (indicated by a *dashed line*). PIF1 activity is further inhibited by the formation of inactive heterodimers with LONG HYPOCOTYL IN FAR-RED 1 (HFR1). ABA synthesis is no longer stimulated, and inhibition of GA synthesis through DAG1 and SOM is relieved. De-repression of the histone arginine demethylase genes *JUMONJI 20* (*JMJ20*) and *JMJ22* leads to the removal of repressive histone arginine methylations (H4R3me2) on the GA biosynthesis genes *GA3ox1* and *GA3ox2*, stimulating GA biosynthesis and subsequent germination. Gray text represents low hormone levels; black text represents high hormone levels.

GIBBERELLIC ACID

Among the many GAs that have been identified in plants, only a few are bioactive; of these, GA_4 is the major one. The last steps in GA biosynthesis are catalyzed by GA20 oxidases and GA3 oxidases, while GA2 oxidases deactivate bioactive GAs (140). GA binding to the receptor GIBBERELLIN-INSENSITIVE DWARF 1 (GID1) leads to interaction with the GA signaling repressors called DELLA proteins, which are subsequently ubiquitinated and degraded by the 26S proteasome via the SCF^{SLY1} complex. Increased GA levels thus relieve the repression of the DELLAs on various classes of transcriptional regulators (24).

2.3. Germination Signaling: Balancing Abscisic Acid and Gibberellic Acid Metabolism

It has long been known that germination depends on regulation of the hormones ABA, which inhibits germination, and gibberellic acid (GA; see sidebar Gibberellic Acid), which promotes germination. PIF1, also known as PIF3-LIKE 5 (PIL5), strongly inhibits germination in the dark by mediating the transcription of ABA and GA metabolic genes (93). Upon light activation, the phytochromes interact with PIF1, leading to its phosphorylation and subsequent degradation via the $CUL4^{COP1\text{-}SPA}$ E3 ubiquitin ligase (147). PIF1 activity is further modulated through accumulation of LONG HYPOCOTYL IN FAR-RED 1 (HFR1), which forms nonactive heterodimers with PIF1 (112). Thus, inactivation and degradation of PIF1 in the light promotes light-induced germination (**Figure 1*b***).

PIF1 regulates GA signaling both directly and indirectly. It directly induces expression of two *DELLA* genes, *GIBBERELLIC ACID–INSENSITIVE* (*GAI*) and *REPRESSOR OF GA1-3* (*RGA*), which are negative regulators of GA signaling (94). It also indirectly regulates GA levels through activation of DOF AFFECTING GERMINATION 1 (DAG1), which subsequently suppresses transcription of the GA biosynthesis gene *GIBBERELLIN 3-OXIDASE 1* (*GA3ox1*) (40). Similarly, PIF1 activates transcription of its direct target *SOMNUS* (*SOM*); SOM, in turn, represses expression of the GA anabolic genes *GA3ox1* and *GA3ox2* and activates the *GA2ox2* gene, which is involved in GA inactivation (69). Cho et al. (18) recently elucidated a mechanism by which SOM suppresses *GA3ox1* and *GA3ox2* transcription: When transcriptionally activated by PIF1 in the dark, SOM directly inhibits expression of the histone arginine demethylases encoded by *JUMONJI 20* (*JMJ20*) and *JMJ22*. With low JMJ20 and JMJ22 levels, *GA3ox1* and *GA3ox2* chromatin contains high levels of H4R3 dimethylation (H4R3me2), which represses their transcription. However, when phytochrome activation in the light decreases PIF1 abundance and consequently SOM activation, *JMJ20* and *JMJ22* are de-repressed and transcribed. JMJ20 and JMJ22 subsequently target *GA3ox1* and *GA3ox2* chromatin and remove the H4R3me2 marks, resulting in more open chromatin accessible for transcription (18). This transcriptional regulation by SOM through JMJ20 and JMJ22 is specific for *GA3ox1* and *GA3ox2*, and the mechanisms by which SOM regulates other downstream targets have yet to be clarified.

DELLA: a family of GRAS proteins that contain an N-terminal DELLA domain and act as repressors of GA signaling

ABA levels are regulated by PIF1 through the same downstream regulators it uses to affect GA signaling. The DELLA proteins GAI, RGA, and RGA-LIKE 2 (RGL2) promote ABA synthesis under low GA levels, for example, in FR conditions soon after imbibition (103). SOM activates the ABA anabolic genes *ABA1*, *9-CIS-EPOXYCAROTENOID DIOXYGENASE 6* (*NCED6*), and *NCED9* but suppresses the catabolic gene *CYP707A2*, all of which mediates increased ABA levels (69). Furthermore, PIF1 directly induces the expression of *ABSCISIC ACID–INSENSITIVE 3* (*ABI3*) and *ABI5*, which encode two ABA-responsive transcriptional regulators (93), and ABI3 activates the *SOM* promoter (98), which could further enhance ABA levels and decrease GA

levels. Taken together, these results indicate that PIF1 inhibits germination by suppressing GA synthesis and signaling while simultaneously increasing ABA biosynthesis and signaling. Early during germination, this inhibition is controlled in the endosperm by phyB; phyB activation by R leads to PIF1 degradation, whereas its inactivation by FR results in PIF1 stabilization. At a later stage, FR can activate phyA in the embryo, leading to GA biosynthesis and induction of germination (**Figure 1**). Light, through phytochrome-mediated PIF1 degradation, thus acts as the switch that tips the balance in ABA and GA metabolism.

Cryptochrome 1 (cry1): a blue light photoreceptor active in high and low fluence rates

Cryptochrome 2 (cry2): a blue light photoreceptor active in low fluence rates

Phototropin 1 (phot1): a blue light photoreceptor that functions at a broad range of light intensities

3. PHOTOMORPHOGENESIS

3.1. De-etiolation and Emergence from Soil

The previous section described germination in light; however, many species also germinate in the dark when their seeds are buried in soil. Germination in the dark triggers etiolated growth of the seedling, which consists of strong elongation of the embryonic stem (hypocotyl) and formation of an apical hook while the development of the embryonic leaves (cotyledons), apical meristem, and root system is inhibited (**Figure 2*a***). Using the energy reserves present in the endosperm, this growth strategy, called skotomorphogenesis, allows seedlings to reach light aboveground. The depth at which the seed is buried and the soil texture are important factors in skotomorphogenesis because resources are limited during etiolated growth. While the seedlings are pushing through the soil, the apical hook and closed cotyledons protect the inactive shoot apical meristem (SAM). Once they reach the soil surface and perceive light, the photomorphogenic growth program is induced. This de-etiolation involves growth inhibition of the hypocotyl, unfolding of the apical hook, cotyledon expansion, chloroplast differentiation, and development of the SAM and root apical meristem (**Figure 2*c***). Completing these developmental changes establishes the seedling as a self-sufficient photoautotroph (3).

3.2. Photomorphogenesis Induced by Light Perception

Initial screens for positive regulators of photomorphogenesis that display an etiolated phenotype in the light have revealed that phyA and phyB are required for de-etiolation induced by FR and R light, respectively, and that cryptochrome 1 (cry1) is required for de-etiolation induced by blue light and UVA (65). Blue light perception by cry2 also induces de-etiolation, as do low fluence rates of UVB perceived by UVR8 (62, 65). Inhibition of hypocotyl elongation is initiated within 30 s of blue light perception by phototropin 1 (phot1) and within several minutes of R light irradiation of etiolated seedlings (100), reflecting a rapid switch in the developmental program.

Site-specific phytochrome chromophore deficiency and complementation of the *phyB* mutant phenotype by enhancer trap–induced *PHYB* expression both showed that light perception for phytochrome-mediated photomorphogenesis predominantly takes place in the mesophyll (34, 128). The specific tissue in which cryptochromes perceive photomorphogenesis-inducing light signals is unknown. The vast array of photoreceptors involved and the subsequent rapid changes in development underline the significance of light perception for seedling establishment after germination in the soil.

3.3. Photomorphogenesis Signaling

The activated photoreceptors transduce the photomorphogenesis signal through the repressors CONSTITUTIVE PHOTOMORPHOGENIC (COP), DE-ETIOLATED (DET), and FUSCA (FUS) (55). Of these, COP1 forms an E3 ubiquitin ligase complex together with the SUPPRESSOR OF PHYA (SPA) proteins. This COP1-SPA complex interacts with

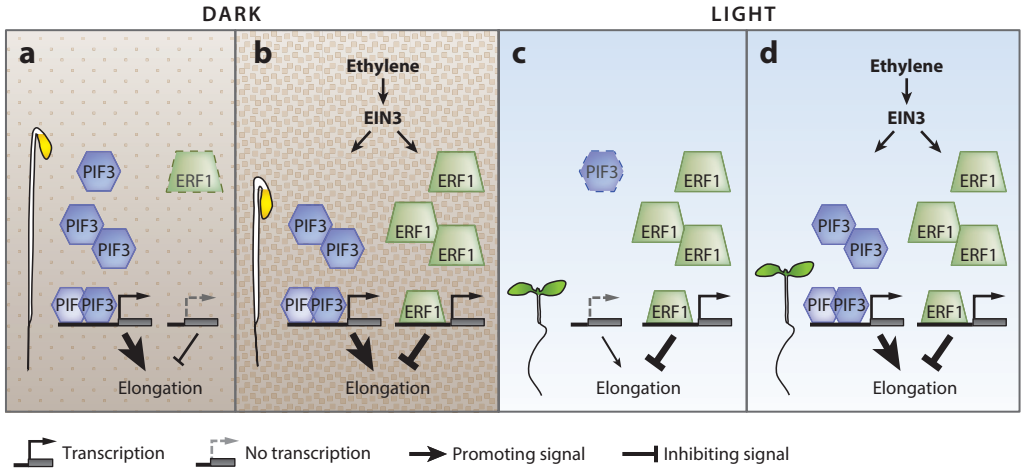

Figure 2

How light determines the effect of ethylene on hypocotyl elongation. (*a*) In soil, where no light penetrates, PHYTOCHROME-INTERACTING FACTOR (PIF) proteins are stable and bind the promoters of downstream targets to promote growth. ETHYLENE RESPONSE FACTOR 1 (ERF1), by contrast, is degraded in the dark (indicated by the *dashed outline*) and cannot regulate transcription of its elongation-inhibiting targets. This leads to typical etiolated growth, or skotomorphogenesis, after germination in the dark. (*b*) Physical obstruction of etiolated growth in soil (indicated by the *coarse pattern*) leads to ethylene accumulation in the seedling. Ethylene signaling through ETHYLENE-INSENSITIVE 3 (EIN3) promotes both the PIF3 and the ERF1 pathway. Because PIFs are already abundant in the dark, this does not have a major impact on their growth stimulation. Increased ERF1 abundance, however, leads to growth inhibition, resulting in attenuated hypocotyl elongation. Ethylene also stimulates thicker hypocotyls and a more closed apical hook. (*c*) In the light, PIFs are degraded (indicated by the *dashed outline*), whereas ERF1 is stable. The resulting inhibition of hypocotyl elongation, together with unfolding and greening of the cotyledons, creates the typical photomorphogenic phenotype of light-grown seedlings. (*d*) In light-grown seedlings, ethylene accumulation (e.g., after flooding or mechanical stress) induces both the PIF3 and ERF1 pathways. Increased PIF3 abundance leads to growth stimulation, whereas an increase of the already abundant ERF1 proteins does not have a large downstream effect on growth inhibition. Together, these effects lead to enhanced hypocotyl elongation in response to ethylene in the light. The line thickness of signals promoting or inhibiting elongation represents the strength of the signal.

photomorphogenesis-promoting transcription factors that are subsequently degraded by the 26S proteasome. Light signaling through the phytochromes and the cryptochromes inactivates COP1-SPA, resulting in the accumulation of positive regulators of photomorphogenesis such as the basic leucine zipper (bZIP) transcription factor ELONGATED HYPOCOTYL 5 (HY5) or the basic helix-loop-helix (bHLH) protein LONG HYPOCOTYL IN FAR-RED (HFR1) (55, 85, 111). In UVB-induced photomorphogenesis, photoactivated UVR8 monomerizes and associates with COP1 and thereby promotes HY5 stability (62). Among the numerous targets of HY5 are many regulators of hormone signaling, including ABA, GA, ethylene (ET), auxin, brassinosteroid, cytokinin, and jasmonic acid (75, 95, 127). Besides the COP/DET/FUS proteins, PIFs are essential repressors of photomorphogenesis in the dark, as demonstrated by the *cop*-like phenotype of the *pif1 pif3 pif4 pif5* quadruple mutant grown in darkness (79, 113). Promoting skotomorphogenic growth while stabilized in darkness, PIFs are dually regulated by light through induction of their rapid degradation and inactivation by DELLAs owing to lower GA levels in the light (4, 25, 38, 78, 96).

3.4. Ethylene-Mediated Emergence from the Soil

Growing out of the soil is a crucial process that requires tight coordination of growth and development to enable successful establishment in the light. The gaseous hormone ET (see sidebar

ETHYLENE

ET binding inactivates its receptors [ETHYLENE RECEPTOR 1 (ETR1), ETR2, ETHYLENE RESPONSE SENSOR 1 (ERS1), ERS2, and ETHYLENE-INSENSITIVE 4 (EIN4) in *Arabidopsis*], which in turn relieves the repression of CONSTITUTIVE TRIPLE RESPONSE 1 (CTR1) on the downstream signaling component EIN2 (41). EIN2 is subsequently cleaved and translocates to the nucleus, where it stabilizes the transcription factors EIN3 and EIN3-LIKE 1 (EIL1) and thus regulates the activation of ET-responsive genes (88).

Ethylene) accumulates upon physical obstruction of etiolated growth (45), and in recent years it has become clear that this plays an important role in coordinating growth and development to guide emergence from the soil and the shift to photoautotrophic growth.

The classical triple response to ET, first described for pea (*Pisum sativum*) hypocotyls in 1901, consists of shorter and thicker hypocotyls and roots and an exaggerated apical hook in etiolated *Arabidopsis* seedlings (33). This phenotype has long been hypothesized to protect the apical meristem during skotomorphogenic growth (33). The amount of ET production positively correlates with both the depth at which the seeds are buried and the firmness of the soil (45, 145). Furthermore, the phenotype in deeper and firmer soil corresponds with that of ET-treated seedlings grown in air (45, 145), confirming that the ET-mediated triple response is a response to soil cover.

In *Arabidopsis* seedlings, ET induces two pathways through ETHYLENE-INSENSITIVE 3 (EIN3) and EIN3-LIKE 1 (EIL1). One of these pathways depends on the induction of *ETHYLENE RESPONSE FACTOR 1* (*ERF1*), and the other depends on the induction of *PIF3*; these two genes are both direct targets of EIN3 but have opposite effects on growth (144). Because of their different protein stability, the light environment determines which of these two pathways ET uses to control growth. In the dark, the growth-promoting PIFs are stable (78), and therefore adding more ET-induced PIF3 to an already abundant pool of PIFs might not further enhance elongation. ERF1, by contrast, is normally degraded in the dark, and boosted levels of this protein through ET induction of *ERF1* expression leads to inhibition of etiolated hypocotyl elongation (144) (**Figure 2*a*,*b***). Soil-dependent ET accumulation thus leads to an ERF1-mediated reduction in growth rate, which could protect the meristem according to soil pressure. Delayed emergence from soil is synchronized with PIF-inhibited synthesis of the chlorophyll precursor protochlorophyllide in the cotyledons, which can cause photooxidative damage after light absorption when present at high levels (56, 113, 119, 145, 146).

When ET accumulates in seedlings in the light, the balance between *ERF1* and *PIF3* induction tips in the opposite direction and leads to enhanced hypocotyl elongation (115, 144). In contrast to what occurs in darkness, PIFs are degraded and ERF1 is stabilized in the light. PIFs now become limiting, and ET-increased PIF3 abundance stimulates growth in the light. On the other hand, ERF1 is saturated in the light, and further stimulation of *ERF1* by ET has no effect on growth (144) (**Figure 2*c*,*d***). Through degradation of PIFs by phytochrome and stabilization of ERF1, light thus acts as a switch that shifts the effect of ET on growth from the ERF1-inhibiting pathway to the PIF3-stimulating pathway.

4. PHOTOTROPISM

4.1. Bending Toward the Light

When seedlings emerge from the soil in suboptimal light conditions, they can reorient their cotyledons toward directional light to optimize photosynthesis. This bending toward the light, or

positive phototropism (as opposed to negative phototropism in roots, which grow away from the light), involves enhanced growth at the shaded side and reduced growth at the lit side of the bending organ (37). Phototropism occurs not only in seedlings first exposed to light, but also in light-grown seedlings, petioles, and inflorescence stems (21, 64), and it could be a mechanism to direct leaves toward canopy gaps during plant competition (5). The mechanisms behind unidirectional light perception and subsequent phototropic bending are, however, best understood in etiolated seedlings.

Phototropin 2 (phot2): a blue light photoreceptor that functions only at a high light intensity

4.2. Phototropism Induced by Unidirectional Light Perception

Phototropism is induced mainly by blue light perception mediated by phototropins, which are named for the response in which they were discovered (13). Of the two phototropins present in flowering plants, phot1 functions at a broad range of blue light intensities, whereas phot2 functions only at a high intensity (37). Blue light–induced phototropic bending is enhanced by phyA-dependent perception of R light 1–2 h before directional blue light treatment (99). Because longer wavelengths penetrate deeper into the soil and phyA is highly abundant in etiolated seedlings, phyA may thus prepare seedlings to better respond to unidirectional light cues. In addition to the phytochromes, the cryptochromes and UVR8 modulate phototropism (48, 125), but the precise mechanisms behind this modulation are unknown.

Plant growth toward unilateral light was already described by Charles and Francis Darwin in the nineteenth century (20). By covering or removing the tips of monocotyledonous grass coleoptiles, they showed that the tip is necessary to induce directional growth, whereas the bending takes place in the middle section of the coleoptile. In dicotyledonous *Arabidopsis*, however, perception and bending spatially overlap. In etiolated seedlings, bending occurs in the hypocotyl elongation zone, and the light gradient is perceived in the elongation zone and lower part of the apical hook, i.e., the parts that first emerge from the soil (105, 141). Using tissue-specific promoters driving phot1 in the *phot1 phot2* mutant, Preuten et al. (105) showed that perception can take place at any cell layer within the upper part of the hypocotyl.

4.3. Phototropism Signaling: Establishing an Auxin Gradient

The work of the Darwins and later researchers led in 1937 to the Cholodny-Went hypothesis (130), which suggests that the phytohormone auxin (see sidebar Auxin) moves from the irradiated to the shaded side of an organ. Increased auxin levels in the cells on the shaded side would then enhance growth there and thus establish phototropic bending. Using various methods, Christie

AUXIN

Auxin is synthesized in the cytosol through several pathways, of which the TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1 (TAA1)–YUCCA (YUC) pathway is the main one (83). Auxin is transported out of the cell by the PIN-FORMED (PIN) and ATP-BINDING CASSETTE B (ABCB) auxin efflux proteins (143). It enters the cells via the AUXIN 1 (AUX1)/LIKE-AUX1 (LAX) influx carriers or, in protonated form, via diffusion (120). In the nucleus, auxin binds to the F-box TRANSPORT INHIBITOR RESPONSE (TIR)/AUXIN SIGNALING F-BOX (AFB) receptors. The auxin receptor complex binds the INDOLE-3-ACETIC ACID INDUCIBLE (IAA) coreceptors, which are subsequently degraded (8). This relieves their repression on the AUXIN RESPONSE FACTOR (ARF) transcription factors.

& Murphy (20) found that directional irradiation indeed results in lateral auxin redistribution, but the exact mechanisms that establish this auxin gradient are still unknown (37).

Phototropin autophosphorylation is required for phototropism and leads to a phosphorylation gradient across unilaterally irradiated oat coleoptiles (59, 110). This phosphorylation gradient is believed to be part of the mechanism driving lateral auxin distribution during phototropism. Several processes may contribute to auxin redistribution. The activity and subcellular localization of the well-studied PIN-FORMED (PIN) auxin efflux transporters depend on their phosphorylation, and several members appear to be required for phototropism (7, 131, 148). However, although studies have shown that PIN3 relocalizes laterally away from the irradiated site in endodermis cells (30), a direct link with phosphorylation by the phototropins has not been established. More direct evidence has been found for the regulation of auxin influx through pH-mediated diffusion. Unilateral blue light irradiation results in reduced phosphorylation of plasma membrane H^+-ATPases in a phototropin-dependent manner (52). Phosphorylation of the H^+-ATPases leads to their activation, which results in acidification of the apoplastic pH and thereby increases the fraction of protonated auxin that can diffuse from the apoplast into cells. Because H^+-ATPase activity is important for the formation of an auxin gradient during phototropism (52), regulation of pH-dependent auxin influx might be an important phosphorylation event mediated by the phototropins.

A link between auxin movement and light-activated phototropin was also established for the efflux transporter ATP-BINDING CASSETTE B19 (ABCB19), which is directly phosphorylated by phot1 in vitro (21). Light activation of phot1 resulted in reduced ABCB19-mediated auxin efflux activity in HeLa cells, which corresponded with the finding that auxin flux downward from the apex was reduced upon phototropism (21). Reduced downward auxin transport may augment the auxin pool locally in the bending zone and thus contribute to the potential to establish an auxin gradient. The *abcb19* mutant shows exaggerated phototropic bending in response to directional blue light, indicating that ABCB19 inactivation is indeed beneficial for phototropism.

Despite an almost 80-year-old hypothesis about the formation of an auxin gradient during phototropism, elucidating the regulation of such a gradient has proven difficult. The three processes described here are, however, not mutually exclusive, and perhaps the answers lie in a combination of many auxin-related events, including these and possibly others. In such a model, phototropin-mediated inactivation of ABCB19 and subsequently reduced auxin transport to the hypocotyl below the bending region would locally increase auxin levels, while pH-dependent auxin influx and PIN-dependent auxin efflux would stimulate lateral auxin redistribution in the bending zone.

5. SHADE AVOIDANCE

5.1. Reaching for the Sun

Once photoautotrophic seedlings are established, they may need to compete with other plants for light. Because an initial size difference or delayed growth is disadvantageous in this competition (102, 129), seedlings must adjust their growth to stay at least as tall as their neighbors. To keep up with the competition, they channel energy mainly into elongation growth at the expense of support tissue, leaf development, and, at a later stage, seed set (15). This so-called shade avoidance syndrome consists of elongated hypocotyls, stems, petioles, or internodes; hyponastic leaves; reduced leaf lamina size; enhanced apical dominance; and early flowering. It occurs in both seedlings and adult plants, and although links with hormone signaling have been established for several of these shade avoidance responses, they have been best described for auxin-mediated elongation of hypocotyls and petioles. (For a short description of shade-induced flowering, see Section 6.4.)

5.2. Elongation Growth Induced by Shade Perception

R:FR: the ratio between red and far-red light, which is high in sunlight but low in foliar shade

Plants specifically perceive shade from other plants through changes in the R:FR ratio detected by the phytochromes. R:FR decreases under foliar shade because green leaves absorb R and reflect FR. Through lateral reflection of FR light, neighboring plants can also be detected through a reduction in R:FR even before actual shading occurs (6).

R:FR is monitored by the phytochrome photoreceptors, which can exist in the inactive, R-absorbing form Pr or the active, FR-absorbing form Pfr. The phytochrome photoequilibrium thus reflects the R:FR of the perceived light (15, 53). PhyB is the main photoreceptor regulating shade avoidance, with phyC–E playing additional roles (39). In *Arabidopsis* seedlings, the main site of perception is in the cotyledons, whereas in older *Arabidopsis* leaves, petiole elongation is induced by irradiation of the lamina (72, 122). Plants also respond to decreases in blue light and photosynthetically active radiation (15, 101), but this is beyond the scope of this review.

5.3. Shade Avoidance Signaling: Boosting Auxin Biosynthesis

PhyB in the Pfr form translocates to the nucleus, where it interacts with several PIFs of the bHLH family of transcription factors (78). Of these, mainly PIF4, PIF5, and PIF7 play important roles in shade avoidance, with less prominent roles for PIF1 and PIF3 (80, 81, 84). The interaction between phyB and PIFs leads to PIF phosphorylation and subsequent inactivation or degradation (81, 84). Inactivation of phyB by a reduction in R:FR relieves this repression on the PIFs, leading to massive transcriptional changes upon perception of low R:FR (54, 80, 81).

Many hormones play a role in the events leading from low-R:FR perception to shade avoidance–related architectural changes, including GA, ET, auxin, brassinosteroid, cytokinin, and jasmonic acid (12, 14, 46, 73, 92). In the last decade, auxin has emerged as a key player in mediating the elongation phenotype, and its signaling pathway is regulated at several levels upon neighbor detection (27). Remarkably, a large part of the shade avoidance transcriptomic profile consists of auxin-related genes (54, 81, 123). A role for auxin and its transport in the shade avoidance response was first established by the impaired hypocotyl elongation response in the *axr1-12* auxin response mutant and seedlings treated with the auxin transport inhibitor naphthylphthalamic acid (NPA) (118). This led to the hypothesis that during shade avoidance, auxin is redirected more laterally to allow elongation of expanding organs (90). Indeed, PIN3–green fluorescent protein (GFP) moves from a more basal to a more lateral location in endodermal hypocotyl cells under low R:FR, and the *pin3-3* mutant has an impaired shade avoidance response (68), indicating that auxin redistribution is important.

The importance of de novo auxin production during shade avoidance was demonstrated when a shade avoidance mutant screen for impaired hypocotyl elongation discovered TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS 1 (TAA1), which catalyzes the first step in a then newly discovered auxin biosynthesis pathway (123). Auxin levels rise quickly after the onset of low R:FR; in seedlings, this auxin appears to be generated in the cotyledons and subsequently transported down to the hypocotyl (106, 123). Interestingly, several *YUCCA* (*YUC*) genes, which encode enzymes catalyzing the rate-limiting step of TAA1-dependent auxin biosynthesis, are directly targeted by PIF4, PIF5, and PIF7 (54, 81). Correspondingly, the *yuc2 yuc5 yuc8 yuc9* quadruple mutant is affected in its response to low R:FR at the seedling, adult, and reproductive developmental stages (92), confirming the significance of auxin production through the TAA1-YUC pathway for the shade avoidance response.

As described above, there is a rather direct link from low-R:FR perception by the phytochromes, through the PIFs, to YUC-mediated auxin production. Apart from auxin biosynthesis, auxin

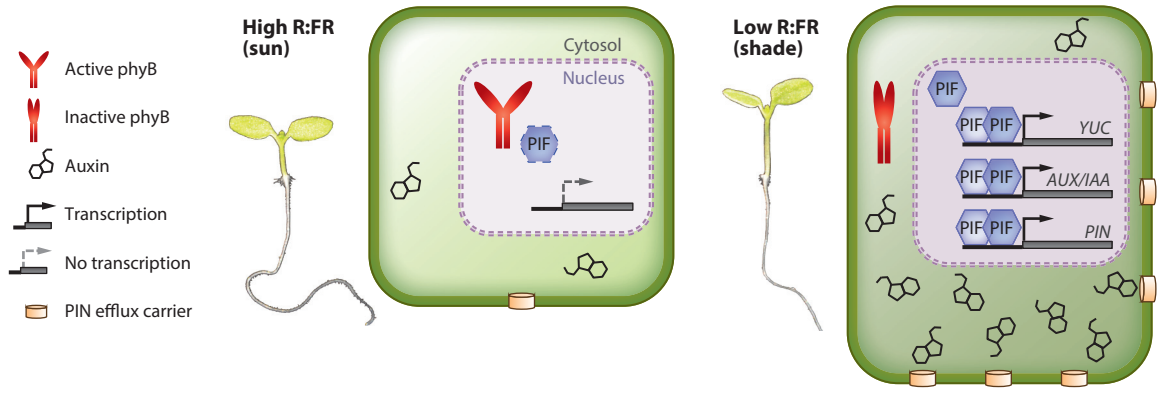

Figure 3

Shade avoidance signaling. Phytochrome B (phyB) is activated under a high red:far-red (R:FR) ratio and translocates to the nucleus, where it inactivates PHYTOCHROME-INTERACTING FACTOR 4 (PIF4), PIF5, and PIF7 and thus inhibits PIF-dependent transcription. Under low R:FR, phyB is inactivated, which allows the PIFs to accumulate and regulate transcription of their downstream targets. Among these are many auxin-related genes, resulting in increased auxin concentration in the cell through transcription of *YUCCA* (*YUC*) genes, changes in *AUXIN* (*AUX*)/*INDOLE-3-ACETIC ACID INDUCIBLE* (*IAA*) auxin signaling genes, and increased auxin transport through PIN-FORMED (PIN) proteins. This process ultimately leads to cell elongation, for instance, in the hypocotyls of *Arabidopsis* seedlings.

sensitivity may also be enhanced in low R:FR, as predicted by the many auxin-related genes regulated in low R:FR and a computational model of shade-induced hypocotyl elongation (49, 92, 123). Importantly, using chromatin immunoprecipitation followed by sequencing, Hornitschek et al. (54) found the auxin signaling genes *INDOLE-3-ACETIC ACID INDUCIBLE 19* (*IAA19*) and *IAA29* among the direct PIF targets, and also found PIF-binding peaks in the promoters of *AUXIN SIGNALING F-BOX 1* (*AFB1*) and *PIN3*. These results suggest that other components of auxin signaling besides biosynthesis are directly regulated in shade. *pif4 pif5* double mutants show reduced responsiveness to the synthetic auxin picloram (54, 91), which indicates that PIF4 and PIF5 are indeed involved in the regulation of auxin sensitivity. However, PIF-mediated auxin sensitivity may play a role mainly when auxin biosynthesis is attenuated, and the mechanisms behind such sensitization remain largely unknown (26, 49).

Taken together, the results described above show that inactivation of phyB leads to dramatic changes in the auxin pathway at the levels of biosynthesis, distribution, and possibly sensitivity (**Figure 3**). The phytochrome-dependent de-repression of PIFs directly links low-R:FR perception to auxin signaling at multiple levels, which underlines the importance of this hormone for shade avoidance.

6. FLOWERING

6.1. From Leaf Production to Reproduction

Flowering at the right time is essential for successful reproduction. It is especially important in climates with strong seasonal changes and for plants of non-self-fertile species, which need to synchronize their flowering time with surrounding individuals. To allow the energy-costly development of flowers and seeds when conditions are most favorable, the induction of flowering is controlled by environmental cues, such as day length and temperature, and endogenous cues such as hormonal status, sugars, and age-dependent signals. Both light quality (see Section 6.4)

and the duration of light during a day, or photoperiod, are important flowering cues. Photoperiod is a reliable seasonal indicator for many species, and plants can be grouped according to the day length at which they flower. Long-day plants, for example, flower when days exceed a critical length, typically in late spring to early summer.

FLAVIN-BINDING, KELCH REPEAT, F-BOX 1 (FKF1): a blue light photoreceptor of the ZEITLUPE family that regulates photoperiodic control of flowering

6.2. Perception and Signaling During Photoperiod-Induced Flowering

PhyA, phyB, and cry2 were the first photoreceptors found to be involved in photoperiod-induced flowering. The FLAVIN-BINDING, KELCH REPEAT, F-BOX 1 (FKF1) E3 ubiquitin ligase, which belongs to the ZEITLUPE family of photoreceptors, was later shown to play a crucial role in this flowering pathway (1).

Transcriptional and posttranslational regulation of CONSTANS (CO) is key for photoperiod-dependent induction of flowering. CO is a transcriptional activator of *FLOWERING LOCUS T* (*FT*), which subsequently activates the photoperiodic pathway (1). In short days, circadian-regulated *CO* transcription peaks in the dark, and CO protein is subsequently degraded by the COP1/SPA complex during the night (1, 61, 82), by the E3 ubiquitin ligase HIGH EXPRESSION OF OSMOTICALLY ACTIVE GENE 1 (HOS1) (76), and by phyB in the morning (35, 124).

In long days, circadian-regulated FKF1 is light activated at the end of the day and can subsequently bind another circadian protein, GIGANTEA (GI). The FKF1-GI complex targets the CO transcriptional repressors CYCLIN DOF FACTORs (CDFs) for degradation, which leads to a peak in *CO* mRNA in the light (1). CO protein subsequently accumulates in the light, when the COP1/SPA complex is inhibited by light-activated phyA and cry2. In addition to its effect on *CO* transcription, FKF1 can also directly stabilize CO (116, 149). The coincidence of *CO*, *GI*, and *FKF1* circadian oscillation with an external light cue thus transduces day-length information through CO abundance. Thus, CO accumulation occurs only in long days and transmits the photoperiod signal to FT.

It has been known since the 1930s that perception of day length takes place in leaves (71). However, although photoperiod perception occurs in leaves, flowering requires reprogramming the SAM from leaf to flower production. It was therefore hypothesized that a mobile signal, the so-called florigen, is produced in the leaves and transported through the phloem to the SAM (117). Studies showed that *FT* is expressed in the vasculature of the apical part of the leaf, and that movement of the FT protein from phloem companion cells to the SAM is necessary to induce flowering (22, 60, 87, 121), indicating that FT acts as a florigen. In the meristem, FT induces transcriptional reprogramming of several downstream flowering-time genes (1). The FT homolog TWIN SISTER OF FT (TSF) has similar properties, and there might be additional signals that transduce day-length perception in the leaves toward the SAM to induce reproductive development (1).

6.3. Links Between Photoperiodic Flowering and Gibberellin

The hormones ABA, GA, ET, auxin, brassinosteroid, cytokinin, and salicylic acid have all been implicated in photoperiodic control of flowering (43). Of these, GA was studied in relation to flowering as early as the 1950s (74), and the molecular mechanisms behind its role in flowering are now beginning to emerge. Although GA was initially associated only with flowering in non-inductive short days (133), its role in photoperiodic flowering has recently received substantial attention. Mutants with altered GA or DELLA protein levels are affected in flowering induction in both short days and long days (42, 104, 132), clearly demonstrating the importance of GA in this process. Studies have also shown that GA plays a role in both the leaf vasculature and the SAM

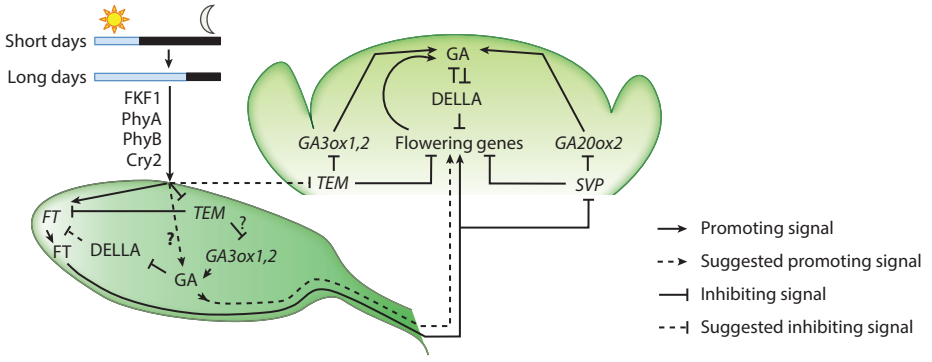

Figure 4

Gibberellic acid (GA) regulation during photoperiodic flowering. Day length is perceived in leaves through the combined action of the photoreceptors FLAVIN-BINDING, KELCH REPEAT, F-BOX 1 (FKF1); phytochrome A (phyA) and phyB; and cryptochrome 2 (cry2). The inductive photoperiod promotes expression of *FLOWERING LOCUS T* (*FT*) in the phloem companion cells. *FT* expression is further enhanced by downregulation of the *TEMPRANILLO 1* (*TEM1*) and *TEM2* repressors and possibly through de-repression by DELLA proteins owing to their GA-dependent degradation. GA biosynthesis may be upregulated by the photoperiod through an unknown mechanism (*question mark*) and through TEM de-repression of the GA biosynthesis genes *GIBBERELLIN 3-OXIDASE 1* (*GA3ox1*) and *GA3ox2*. Whether the latter affects *FT* expression in leaves is unknown. FT protein, possibly along with GA (which relocates in short days), subsequently travels through the vasculature to the shoot apical meristem, where it transduces the photoperiod signal to induce several flowering genes. Expression of these genes requires de-repression from DELLAs, which are degraded owing to increased GA levels, and is further de-repressed by downregulation of inhibitors such as the *TEM* and *SHORT VEGETATIVE PHASE* (*SVP*) genes. Downregulation of *TEM* expression, which is independent of FT, also de-represses its inhibition of *GA3ox1* and *GA3ox2* in the shoot apical meristem. Downregulation of *SVP* relieves its repression on the GA biosynthesis gene *GA20ox2*. Finally, flowering genes themselves appear to regulate GA biosynthesis genes, providing a feed-forward loop on GA signaling during photoperiod-induced flowering.

to control the expression of flowering-time genes during photoperiodic flowering (43, 63, 104, 142). In the leaf vasculature, GA controls *FT* expression independently of CO and GI through the abundance of DELLAs, which inhibit *FT* expression (42, 104). GA thus contributes to the FT signal in leaves by degrading DELLA proteins. Similarly, release of DELLA repression on floral regulators in the SAM appears to be a critical step for flowering induction downstream of the photoperiodic signal (42, 104, 142).

GA accumulates in the SAM before local induction of GA metabolic genes, and GA applied to the leaf can both travel to the shoot meristem and induce flowering (36). This indicates that GA is produced in both the leaf and the SAM and is transported from the leaf to the SAM to induce flowering. In agreement with this idea, GA biosynthesis is induced shortly after a transition from short days to long days (44, 135, 137). How GA biosynthesis is regulated by the photoperiod is not well understood, but studies have recently established some links between regulators of floral induction and GA metabolic enzymes (**Figure 4**).

The *TEMPRANILLO 1* (*TEM1*) and *TEM2* genes are expressed in both leaves and the meristem; they inhibit *FT* expression by directly binding to its promoter and inhibit flowering at the meristem (97). *TEM* expression is circadian regulated, and a transition from short days to long days immediately shifts their expression patterns and decreases overall transcription (97). Interestingly, TEM1 directly represses the GA biosynthesis genes *GA3ox1* and *GA3ox2* broadly in *Arabidopsis* (97). This suggests that, in addition to repressing *FT*, the TEMs inhibit floral

induction by downregulating GA levels. Consequently, photoperiod-mediated downregulation of *TEM* expression may facilitate the transition to flowering through induction of GA biosynthesis. Nevertheless, it is not entirely clear to what extent TEM-mediated GA regulation contributes to the control of flowering through FT expression in leaves.

Andres et al. (2) recently showed that another repressor of flowering-time genes, the MADS-box transcription factor SHORT VEGETATIVE PHASE (SVP), controls GA biosynthesis genes in response to photoperiod at the SAM. Their genetic analysis revealed that decreased *SVP* expression following a transition to long days correlated with an FT-dependent increase in expression of the GA biosynthesis gene *GA20ox2*. Interestingly, increased *GA20ox2* expression through reduced *SVP* expression led to the induction of transcription factors encoded by *SQUAMOSA PROMOTER BINDING–LIKE* (*SPL*), which regulate flower-inducing genes (2, 142). This demonstrates a positive feed-forward loop that removes the repressor and reinforces the flowering-inducing signal.

Other flowering-time genes also seem to interact with GA signaling, thus regulating GA levels through a feed-forward mechanism (32, 139). Moreover, recent genome-wide chromatin immunoprecipitation experiments have demonstrated the direct binding of flowering-promoting proteins to GA biosynthesis and signaling genes (57, 67, 86). However, determining whether this binding is functionally relevant will require further experiments.

Taken together, the results described above suggest that GA can control flowering in two ways. The first is by regulating *FT* expression in leaves, a process that includes regulation of GA biosynthesis by the photoperiod upstream of FT. Photoperiod-regulated GA levels in leaves may be controlled by the broadly expressed TEMs (97). Second, GA acts downstream of the photoperiod pathway at the SAM to regulate flowering-time genes. Through a feed-forward loop, GA biosynthesis genes are subsequently induced by flowering-time genes at the SAM, presumably reinforcing a swift transition to a new developmental program.

6.4. Flowering Induced by Light Quality

Early flowering is part of the shade avoidance syndrome, which is induced by changes in the light quality, namely the R:FR ratio (see Section 5). Accelerated flowering can enhance reproductive success under the adverse light conditions experienced by plants growing at high density, where competition for light increases with time (15). As described in Section 5.2, phyB is the main receptor mediating shade responses induced by low R:FR. Because *phyB* mutants also have an early-flowering phenotype (47, 107), phyB appears to be important for the regulation of shade-induced flowering. PhyD and phyE play additional roles, as shown by studies in which higher-order mutants flowered even earlier than *phyB* mutants (28, 29).

In accordance with the inhibitory role of phyB on CO during photoperiodic flowering (see Section 6.2), phyB inactivation in *phyB* mutants and low-R:FR-treated plants leads to upregulated *CO* expression (16, 134). Because *CO* was upregulated in *phyB* mutants only in long days, not in short days, and low-R:FR-induced flowering occurred only in long days, it was proposed that phyB-mediated acceleration of flowering enhances the photoperiod pathway (16, 134). However, *ft* mutants are not completely unresponsive to phyB-dependent induction of flowering (70), and *phy* mutants also flower early in short days without activation of the photoperiod pathway (28, 107). This suggests that light quality–dependent flowering is not completely regulated through the photoperiod pathway and that there must be additional mechanisms through which light quality induces flowering.

How phyB inactivation leads to the induction of photoperiod-related genes is not understood. PhyB-dependent flowering appears to require the Mediator subunit PHYTOCHROME AND FLOWERING 1 (PFT1), which regulates *CO* and *FT* expression in both CO-dependent and

CO-independent ways (16, 58). The COP1-SPA complex, which degrades CO during the night (see Section 6.2), does not seem to play a role in light quality–induced flowering, as the *cop1-4* and higher-order *spa* mutants show a wild-type flowering response to low R:FR (109).

In terms of hormonal regulation of shade-induced flowering, current knowledge is limited. As with the photoperiodic pathway, GA biosynthesis may be important for light quality–induced flowering. Low-R:FR treatment resulted in an increased GA concentration in bean (*Phaseolus vulgaris*) internodes and *Arabidopsis* seedlings (9, 12), and expression of the GA biosynthesis gene *GA20ox2* was upregulated in end-of-day FR-treated *Arabidopsis* petioles (51), indicating that GA levels are regulated by shade. Importantly, *GA20ox2* RNA-interference lines showed delayed flowering after a transition to long days in low light intensity enriched with FR (50), suggesting that regulation of this gene may indeed be important for phytochrome-dependent induction of flowering. Whether GA levels also rise in the SAM has not been shown, but *GA20ox2* expression is regulated in the apex during phyB-dependent flowering (50). DELLA abundance and activity decreased in a GA-dependent manner in low-R:FR-treated *Arabidopsis* seedlings and petioles (23, 31). Whether DELLA de-repression plays a role in light quality–induced flowering is unknown.

Auxin is a key regulator of the shade avoidance phenotype (see Section 5.3) and may play a role in regulating early flowering in shade as well. Mutations in the gene coding for the polar auxin transporter BIG attenuate flowering induced by inactivation of phyB (66). Flowering was not attenuated in the TAA1 mutant *shade avoidance 3* (*sav3*), but this may be related to the fact that TAA1 is not the rate-limiting step in auxin biosynthesis (123). By contrast, the *yuc2 yuc5 yuc8 yuc9* mutant, which lacks all the *YUC* genes previously shown to be upregulated in shade, displayed accelerated flowering (92). It is thus possible that PIF-regulated induction of auxin biosynthesis triggered by phyB inactivation is important for shade-related flowering.

7. CONCLUDING REMARKS

The dynamic research fields of photobiology and plant hormone signaling have generally been studied separately but are now coming together on many occasions. As discussed here, different light signals can trigger changes in plant growth and development, and these changes are typically regulated by plant hormones. For some of the processes described above, the importance of hormonal regulation has long been known, whereas the regulatory pathways have started to become clear only in recent years. As our understanding of individual hormone pathways and photomorphogenic processes grows, mechanisms through which light signaling pathways tap into hormone signaling will be further elucidated.

What becomes clear from the cases described in this review is that the signaling steps from light perception to hormone regulation can be rather direct (as is the case for shade-regulated auxin biosynthesis) or can go through many intermediate players (as is the case for chromatin remodeling leading to GA biosynthesis during germination). What is striking is that PIFs are involved in many of these light responses, but their function changes depending on the light conditions: In some cases, they have a promoting role through their transcriptional activity (in, for example, growth in darkness or low R:FR), whereas in others, their degradation in light relieves repression on downstream targets. Clearly, through this versatility, they act as molecular switches that control light-regulated growth and development.

By focusing on single events, researchers are beginning to elucidate the mechanisms of light-regulated hormone regulation, but we are only beginning to understand how different light signals are integrated and determine the plant's response to a complex light environment (95). Further open questions include how light signals are integrated with development such that the response depends on developmental age, and how this leads to organ- and tissue-specific responses. As

advancing technologies enable researchers to zoom in on spatiotemporal patterns of signaling and our knowledge of transcriptional and protein regulation expands, finding answers to these questions will be the realistic challenges to tackle in the future.

SUMMARY POINTS

1. During germination in the light, degradation of PHYTOCHROME-INTERACTING FACTOR 1 (PIF1) leads to reduced abscisic acid (ABA) biosynthesis and increased gibberellic acid (GA) biosynthesis. PIF1 degradation leads to relieved repression of the histone arginine demethylase genes *JUMONJI 20* (*JMJ20*) and *JMJ22* by the PIF1 target SOMNUS (SOM). JMJ20 and JMJ22 subsequently modify the chromatin of the GA biosynthesis genes *GIBBERELLIN 3-OXIDASE 1* (*GA3ox1*) and *GA3ox2*, thereby enhancing their transcription and increasing GA levels.
2. In developing seedlings, light acts as a switch that shifts the effect of ethylene (ET) on growth through differential light stability of the transcription factors PIF3 and ETHYLENE RESPONSE FACTOR 1 (ERF1). During etiolated growth in the dark, stimulation of the ERF1 pathway by ET leads to reduced hypocotyl elongation. During photomorphogenic growth in the light, stimulation of the PIF3 pathway leads to enhanced hypocotyl elongation.
3. Phototropic bending toward a directional light source requires an auxin concentration gradient between the shaded and lit sides of the bending organ. Light regulation of such gradient formation may depend on auxin transport through PIN-FORMED 3 (PIN3) and ATP-BINDING CASSETTE B (ABCB19) and on enhanced pH-regulated auxin influx.
4. Elongation growth following shade perception depends on auxin biosynthesis. Stabilization of PIFs in shade results in their direct induction of several genes of the *YUCCA* (*YUC*) family, which encode enzymes catalyzing the rate-limiting step in auxin biosynthesis.
5. Photoperiod-induced flowering requires increased GA levels, which leads to degradation of DELLA proteins and subsequent de-repression of flowering genes. In a feed-forward mechanism, flowering genes in turn regulate GA biosynthesis genes, further promoting the switch to reproduction.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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