
REVIEWS

Phytochromes and Other (Photo)Receptors of Information in Plants

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Abstract—In land plants, at least five groups of photoreceptors perceive information about light conditions and diurnal rhythm, as well as about ambient temperature, presence of pathogens or competing neighbors, direction of the gravity vector and other factors. The photoreceptor toolkit allows plants to integrate environmental information and “make decisions” necessary for survival and successful reproduction, i.e., whether to enter or exit dormancy, accelerate or stop growth, promote or delay flowering; choose the direction of growth, induce or suppress the formation of side shoots, as well as regulate the synthesis of volatile substances affecting the growth of neighbors or pathogens. These photoreceptors include phytochromes, cryptochromes, phototropins, the ZTL (ZEITLUPE) family proteins, and the ultraviolet-B receptor UVR8. In spite of the diversity of plants photoreceptors, their functionality follows several “common rules.” Transformation of the information on light quality and quantity into metabolic and morphogenetic responses occurs via controlled degradation of transcription factors mediated by interactions of the active form of a photoreceptor and the COP1-SPA1 E3-ubiquitin ligase complex in the nucleus. Apart from interacting with COP1, the active forms of photoreceptors in the nucleus can directly bind to transcription factors and trigger their degradation. Phytochromes belong to the largest (the molecular mass of a monomer is ca. 125 kD) and most sophisticated plant photoreceptors. In addition to the abovementioned mechanisms, they also regulate alternative splicing and the selection of alternative promoters for thousands of plant genes. The interaction of phytochrome with jasmonate signaling is of special interest, as phytochromes regulate the jasmonate-mediated cessation of growth in response to stress. This review focuses on data revealing the potential for the application of novel information on plant photoreceptors for the generation of crop varieties capable of high performance under stress conditions.

Keywords: higher plants, photoreceptors, phytochromes, cryptochromes, phototropins, shade avoidance syndrome, jasmonates, growth, stress

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INTRODUCTION

A thorough understanding of the mechanisms regulating photosynthesis and sink-source relations in plants is of primary importance for research aimed to increase crop productivity [1]. Adolf T. Mokronosov, famous for his research of crop photosynthesis and the development of sink-source relations during plant ontogenesis, pointed at the importance of photoperiodic regu-

lation both for photosynthetic performance and the establishment of the architecture of plant body ultimately influencing the distribution of photoassimilates between plant organs [1]. In plants, at least five groups of photoreceptors perceive information on light conditions and photoperiod, as well as on ambient temperature, on the presence of pathogens or competing neighbors, gravitational forces and other

Abbreviations: APA—ACTIVE PhyA BINDING; APB—ACTIVE PhyB BINDING; B—blue light; BZR1—BRASSINAZOLE-RESISTANT 1; CCE—CRYPTOCHROME C-TERMINAL EXTENSION; CDF—CYCLING DOF FACTORS; ChlH—a subunit of the ChlH Mg-chelatase; CIB—CRY-INTERACTING bHLH; CO—CONSTANS; COI1—CORONATINE-INSENSITIVE 1; COP1—CONSTITUTIVE PHOTOMORPHOGENESIS 1; CRY—cryptochrome; CRY-DASH—DASH (*Drosophila*, *Arabidopsis*, *Synechocystis*, *Homo*)-class cryptochrome; FHL—FHY1-like protein; FHY—FAR-RED ELONGATED HYPOCOTYL 1; FKF1—FLAVIN-BINDING, KELCH REPEAT, F-BOX 1; FR—far red light; FT—FLOWERING LOCUS T; G—green light; GAF—cGMP-specific phosphodiesterases, adenylyl cyclases, FhlA domain; GI—GIGANTEA; GUN4—GENOMES UNCOUPLED 4; R—red light; HFR1—LONG HYPOCOTYL IN FAR-RED 1; HIR—high irradiance response; HY5—elongated hypocotyl 5; HYH—homolog of long hypocotyl in far-red 1; JAZ—jasmonate-ZIM domain; KELCH—a 50-amino acids-motif in proteins which repetitions form a ‘propeller’ structure; LAF1—long after far-red; LFR—low fluence response; LKP2—LOV Kelch Protein 2; LOV—light-oxygen-voltage-sensing domain; PAS—Per-Arnt-Sim domain; pAtSUC2—promoter of the AtSUC2 gene; Pfr—active phytochrome; PHOT—phototropine; PHR—photolyase homology region; Phy—phytochrome; PIF—phytochrome-interacting factor; Pr—inactive phytochrome; RUP—REPRESSOR OF UV-B PHOTOMORPHOGENESIS; SAS—shade avoidance syndrome; SPA1—SUPPRESSOR OF PHYTOCHROME A; TOC1—TIMING OF CAB1; UV-B—ultraviolet B radiation; UVR8—UV RESISTANCE LOCUS 8; VLFR—very low fluence response; ZTL—ZEITLUPE.

factors [2–4]. These are: phytochromes perceiving red- (R) and far-red- (FR) light; cryptochromes, phototropins and the proteins of the ZEITLUPE family perceiving UV-A radiation, blue (B) and green (G) light; and the receptor for ultraviolet B-radiation (UV-B), the UVR8 protein [5–11]. It is likely that plants contain further, yet undiscovered, photoreceptors, including a type specific for G [12]. Altogether, a complex combination of photoreceptors enables plants to integrate environmental information and ‘make decisions’ necessary for survival and successful reproduction: enter or exit a dormant state, accelerate or stop growth, promote or retard the transition from vegetative growth to flowering, determine the optimal direction for growth, start the formation of tillers, and regulate the synthesis of volatiles able to influence the propagation of pathogens or the growth of competing neighboring plants, and the transition to programmed cell death [13–18]. Such a wide spectrum of physiological responses is enabled by interactions of photoreceptor-based signaling pathways with phytohormone systems and with signaling pathways based on the production of reactive oxygen species (ROS) in the photosynthetic apparatus and in mitochondria [19–21]. Apart from this, recent studies have shown that photoreceptors can lead to switches in genetic programs via the regulation of alternative splicing as well as through selection of alternative promoters for thousands of plant genes [22, 23]. Here, a short summary of current knowledge on plant photoreceptors will be presented, with a focus on the interactions of phytochrome-based signaling with jasmonate signaling with the potential to be used for the breeding of crop varieties able to ensure high yield under stress conditions.

CLASSES OF PLANT PHOTORECEPTORS

Thus far, at least 14 photoreceptors have been identified in the model plant *Arabidopsis thaliana*. These include five phytochromes (PhyA–E), three cryptochromes (CRY1–3), two phototropins (PHOT1, PHOT2), three proteins of the ZEITLUPE family (ZTL, FKF1, LKP2) and a UV-B receptor, UVR8. These classes of photoreceptors are present in bacteria, fungi, algae and land plants but not in all subgroups of these organisms [24].

The Red-Light Receptors: Phytochromes

Phytochromes are the key regulators of plants growth and development. With monomers of 125 kD, they represent the largest and most sophisticated plant photoreceptors. The N-terminal part of phytochrome performs light sensing. It consists of three conserved domains, PAS, GAF and PHY; the GAF domain binds the chromophore, phytochromobilin, via a thioester bond with a cysteine residue [25]. The C-terminal part forms a serine-threonine kinase which can perform autophosphorylation required to form a dimer, the active form of a phytochrome. Phyto-

chromes are synthesized in the cytoplasm in the dark phase as inactive proteins. Absorption of a light quantum by phytochromobilin leads to minor conformational shifts (in the order of less than one angstrom) of parts of the chromophore molecule which, due to the particular structure of the photoreceptor protein, lead to major conformational changes in the phytochrome, its activation and mobilization to the nucleus and ultimately to a switch in the activity of genetic programs in the cell [25]. Upon absorption of a light quantum, the C15=C16 double bond within the phytochromobilin molecule undergoes isomerization leading to the rotation of the D-ring plane by 180° [25]; an alternative mechanism is the rotation of the A-ring plane by 90° around the double bond C4=C5 [26]. Phytochromes form unique three-dimensional structures: the ‘knot’ (formed by a ‘looping’ of polypeptide chain around itself) necessary for stabilization of hydrophobic interactions within the protein, and the ‘hairpin’ formed by antiparalleled beta-sheets. Upon illumination with R, the liberation and rotation of the ‘hairpin’ lead to the formation of a single alpha-helix in the place of two beta-sheets within the PHY domain; upon illumination with FR, the phytochrome structure returns to the initial inactive state [25]. The changes of the conformation of the phytochrome also lead to the activation of its kinase activity.

The absorption maximum of the chromophore in inactive phytochromes lies in the red part of the light spectrum (660 nm). After illumination with R, the activated phytochrome acquires the second absorption maximum of the chromophore in FR region (730 nm); these two forms of phytochromes are designated as Pr and Pfr, respectively. Illumination with FR promotes the transformation of the active Pfr to the inactive Pr. Due to a gradual thermal relaxation of phytochromobilin, the reversion of Pfr to Pr will also occur in the dark. The absorption of both Pfr and Pr is negligible in the region of 450–550 nm, and low in the UV-A region [9]. However, in the cells containing very high amounts of phytochrome, the absorption by the inactive Pr form of even very low amounts of light from regions of the solar spectrum other than R, including FR, becomes physiologically significant [27]. This situation takes place in etiolated *Arabidopsis* seedlings accumulating very high amounts of PhyA, leading to a ratio of PhyA, PhyB, PhyC, PhyD and PhyE of 85 : 10 : 2 : 1.5 : 1.5 [28]. In this case, even very low intensities of FR or any other light will, with high probability, enable the transition of at least several PhyA molecules into their active form, and these molecules will suffice for the induction of the photomorphogenetic program. This explains the special role PhyA plays in de-etiolation, and also means that in etiolated seedlings, photomorphogenesis, once activated, cannot be ‘switched off’ by irradiation with FR [27]. The photomorphogenetic program in seedlings activated by PhyA includes the cessation of hypocotyl growth in dicots (and of mesocotyl growth in monocots, respec-

tively), the growth of coleoptile in monocots, unfolding of cotyledons, greening of seedlings and the completion of the development of the photosynthetic apparatus in plastids. The opposite program, scotomorphogenesis, includes the activation of hypocotyl growth in dicots and of mesocotyl growth in monocots, the inhibition of the expression of many genes encoding components of the photosynthetic apparatus, the enzymes of chlorophyll biosynthesis etc. [27].

PhyA is a photolabile phytochrome which in high light undergoes photodestruction. PhyA can be activated by less than $1 \mu\text{mol}/\text{m}^2$ light quanta of any wavelength within the 300–780 nm region. Upon activation, it mediates the complex of reactions designated as ‘very low fluence response’ (VLFR). However, PhyA can be activated also by high light intensities ($1000 \mu\text{mol}/\text{m}^2$ and more quanta) in response to an increase in FR, or in response to a decrease of light quantity [29]. This type of responses is summarized as ‘high irradiance response’ (HIR). Owing to the HIR, seedlings can complete deetiolation even under a dense canopy [27, 30].

In the dark, PhyA is located in the cytosol, as the apoprotein lacks a nuclear localization signal. Here, PhyA can regulate gravitropism [31]; the cytoplasmic phytochromes might directly interact with plasma membrane proteins [32]. Many publications report phytochrome-mediated plant responses occurring within 10 min after illumination with R or FR, i.e. not enough time for changes in gene expression patterns [33]. An example of such a response is the migration of the chloroplasts within the cells of the alga *Mougeotia* sp. initiated by the illumination of cells with polarized R [34]. In vascular plants, an example would be the rapid (within a few seconds) depolarization of plasma membrane in barley root tips in response to R, abolished by FR (the so-called Tanada effect [35]), or the folding of leaves of some Fabaceae at the onset of the dark phase, etc. (reviewed in [33]). These reactions might be mediated by interactions of cytosolic phytochromes with other photoreceptors, namely with plasma membrane-anchored phototropines (see below). Such interactions were demonstrated in cells of *Physcomitrella patens* [36] but not yet in vascular plants; they might be mediated by the cytosolic PKS (phytochrome kinase substrate) proteins [37]. At last, phytochromes in the cytosol can regulate the translation of mRNAs as was demonstrated for transcripts encoding protochlorophyllide oxidoreductase A [38].

Upon perception of light of any wavelength, PhyA rapidly translocates into the nucleus. This translocation is mediated by FHY and FHL proteins which contain a nuclear localization signal [27]. They bind the phosphorylated form of PhyA, which then undergoes dephosphorylation in the nucleus and uncouples from FHY and FHL. In the nucleus, PhyA directly interacts with the COP1-SPA1 (Constitutive Photomorphogenic 1/Suppressor of PhyA 1) E3-ubiquitin

ligase complex, and with the transcriptional regulators PIF1 and PIF3 (see below).

In contrast to PhyA, PhyB and all other phytochromes in *Arabidopsis* are stable in the light. In seedlings grown at constant illumination, PhyB becomes the dominating phytochrome, as indicated by the ratio of PhyA, PhyB, PhyC, PhyD and PhyE (5 : 40 : 15 : 15 : 25, as shown by immunoblotting [28]). However, under these conditions, the levels of PhyB are still much lower than PhyA levels in etiolated seedlings; therefore, neither very low R nor high FR nor any other wavelength can activate a physiologically significant number of PhyB molecules. Altogether, only R of an intensity in the range of 1 to $1000 \mu\text{mol}/\text{m}^2$ can lead to a specific activation of the PhyB and thereby of the complex of PhyB-regulated reactions called ‘low fluence response’ (LFR). This explains the inhibition of the PhyB-activated photomorphogenetic programs by FR [27]. As PhyB contains a nuclear localization signal, its default localization is in the nucleus; there, it interacts with COP1-SPA1 and with transcription factors such as PIF4, PIF5, PIF7, and also performs other functions (see below). In the light, PhyB triggers photomorphogenesis which includes the completion of the development of the photosynthetic apparatus, intensification of the width growth of leaf laminae, compactification of rosettes and sprouts, and the onset of tillering [13]. In mature plants, PhyB also inhibits the opposite program, scotomorphogenesis, also called a shade avoidance syndrome (SAS). In mature plants, inactivation of PhyB by a decrease in the R : FR ratio in the incident light leads to the elongation of petioles and internodes as well as to leaf hyponasty and pale green coloration of leaves due to incomplete development of the photosynthetic apparatus [13]. Under high light conditions, it is the dysfunction of PhyB, not of PhyA, that results in a pale-green coloration of the leaves [30, 39]. In a rice mutant lacking PhyB, expression of the genes encoding the Mg-chelatase subunit ChlH and an activator of this enzyme, GUN4, was inhibited, leading to the establishment of a pale-green phenotype and the onset of SAS under direct light [40].

PhyB triggers formation of stomata [41]. The demonstration that the PhyB-regulated signals mediating the differentiation of stomata in leaf epidermis move cell-to-cell, i.e., act non-cell-autonomously, was the first evidence for the systemic action of PhyB [42]. In leaves, the *PhyB* gene is expressed in the guard cell precursors, in guard cells and in the epidermis. The number of stomata per leaf area unit was lowered in the *Arabidopsis* mutant lacking PhyB but restored upon complementation with the *phyB* cDNA, notably, not only under control of a guard cell-specific promoter, but also of any promoter active in cells other than stomata, including the phloem-specific promoter of the sucrose transporter *AtSUC2* [42]. Additionally, the non-cell autonomous action of PhyB was confirmed by complementation of negative gravitropism in hypo-

cotyls of the *phyb* knockout mutant by a *PhyB* construct expressed in the epidermis of hypocotyls [43].

In summary, PhyA represents a key 'switch' between scoto- and photomorphogenetic programs in etiolated seedlings as well as in mature plants grown in the shade, while PhyB is responsible for this function in green seedlings and in mature plants grown under high light [27]. This applies to both dicots and monocots. There are, however, some differences in the organization of the phytochrome system between dicots and monocots. In contrast with *Arabidopsis*, rice, wheat and barley contain only three phytochromes PhyA, PhyB and PhyC. While PhyB represses flowering in dicots, in all cereals studied it promotes flowering in combination with PhyC [44, 45]. Thus far, no other functional differences between dicot and monocot phytochromes have been revealed.

Many *phyA* and *phyb* mutant alleles have been characterized where specific phytochrome functions are 'switched off' owing to mutations leading to amino acid exchange in different domains of the apoprotein; for instance, PhyA-302 plants lack the HIR response but retain the VLFR. Very recently, plant phytochromes were shown to function as thermoreceptors [2]. A mutant form of PhyB where Tyr 276 is exchanged with His is constitutively active and does not revert to the inactive form in the dark; experiments with the corresponding plants showed that the function of phytochromes as thermosensors is related to the rate of Pfr to Pr reversion in the dark [2]. The higher the temperature during the night, the faster is the reversion of wild-type PhyB into the inactive form. Apart from this, the higher the temperature, the weaker is the binding of PhyB to the promoters of PhyB-regulated genes. This mechanism differs from the mechanism of temperature sensing in cyanobacteria: there, membrane integral two-component sensor histidine kinases Hik33, similar to phytochromes, function as sensors of both light and temperature conditions, but, in contrast with phytochromes, perceive the temperature-induced changes in membrane fluidity [46] while temperature sensing of phytochromes has been attributed to a temperature-responsive half-life of Pfr [2]. In *Arabidopsis*, five phytochromes regulate different plant functions in response to temperature: in warm conditions, seed germination is controlled mainly by PhyB, but in cold conditions by PhyE; at the same time, after a prolonged cold period seeds need PhyA for germination [47]. Inhibition of flowering is regulated by PhyE in cold conditions and by PhyB in warm conditions [47].

Phytochromes are major regulators of transcriptional activity of the plant genome. Both PhyA and PhyB can directly bind to the promoters of the regulated genes [2, 48]. In seedlings transferred from darkness to light, phytochromes regulate the expression of several thousands of genes and alternative splicing of transcripts of over one thousand of genes [22]. Another mechanism is the light-dependent selection

of promoters [23]. The occurrence of genes with alternative promoters opens the possibility to produce mRNAs differing in 5'-UTR, which can influence the stability of the mRNA, as well as the amino acid sequence at the N-terminus of the encoded protein, depending on the selection of transcriptional start site. Peptide signals targeting proteins to mitochondria or chloroplasts usually reside at the N-terminus of the protein. PhyB has been shown to mediate promoter selection for transcription of more than two thousands of genes, probably by direct binding to their promoters; this can lead to the synthesis of proteins locating either to the cytosol or to organelles, depending on light conditions [23]. For instance, in *A. thaliana* growing in the shade, both the chloroplastic as well as a novel cytosolic isoform of the photorespiration enzyme, glycerate kinase, were detected; both isoforms are products of one and the same gene but, depending on PhyB-mediated promoter selection, the N-terminal chloroplast signal peptide is either present or absent. The cytosolic isoform functions in a 'photorespiratory shunt' important for the protection from photoinhibition during the transfer of plants from the shade to high light [23].

While the mechanisms of synthesis and degradation of the phytochrome proteins, and the related signaling function of phytochromes, are objects of intensive study, much less is known about the regulation of the activity of the phytochrome-encoding genes. The regulation of *PhyA* transcription can involve the (de)methylation and (de)acetylation of histones, likely under participation of PhyB [10]. According to other data, in the light PhyB mediates the methylation of the promoter of *PhyA* and inhibits its expression [27]. In *Arabidopsis* leaves, the expression of *PhyA* was inhibited by light, probably via PhyA itself, while the expression levels of *PhyB* did not change in response to light; in roots, expression of genes encoding both *PhyA* and *PhyB*, but in particular *PhyB*, was enhanced when roots were exposed to light [49]. In the dark, the expression levels of both *PhyA* and *PhyB* increased in *Arabidopsis* leaves, however, these data were obtained using plants expressing promoter-*GUS*, constructs which precluded the analysis of possible regulation of the stability of phytochrome genes transcripts in vivo based on 3'-UTR sequences [49]. A similar method was used to study *PhyA* expression patterns in rice [50], where *PhyA* was found to be active in all tissues in etiolated seedlings but the expression became confined to leaf vascular bundles upon illumination with R but not FR; this restriction was mediated by *PhyB* [50].

Another mechanism regulating the expression levels of phytochrome genes might be related to the levels of 5-aminolevulinic acid, a common biosynthetic precursor of chlorophylls, heme and phytychromobilin. As phytychromobilin synthesis occurs in plastids, this type of regulation would provide a 'feedback' mechanism to coordinate the biosynthesis of phyto-

chrome apoproteins and chromophores, respectively. Recently, joint regulation of light-responsive genes by phytochromes and by retrograde signals was demonstrated [21]. Phytochromes play a major role in the regulation of chlorophyll biosynthesis in seedlings [51].

Phytochrome-encoding genes have been found in the genomes of some fungi and in brown algae (including genomes of brown algal viruses), but are absent from the genomes of haptophytes, red algae and green algae [24]. Phytochromes of charophytes and land plants have a common origin. In liverworts, hornworts and heterosporous lycophytes, phytochromes are encoded by a single gene, while in mosses, homosporous lycophytes, ferns and seed plants, phytochrome genes underwent diversifications. Of special interest are neochromes, the photoreceptors found in zygnemous algae, hornworts and ferns, where the 'phytochrome-like' photosensory N-terminus is fused with a 'phototropin-like' C-terminus, and the protein binds both chromophores [24, 52].

The Blue Light Receptors: Phototropins, Proteins of the ZEITLUPE Family, Cryptochromes

Phototropins are light-activated protein kinases. They usually localize to the plasma membrane but do not represent integral membrane proteins; they also can be associated with the chloroplast outer envelope [6]. *Arabidopsis* has two phototropins: photostable PHOT1 and photolabile PHOT2. In *Arabidopsis*, the light-induced activation and autophosphorylation of phototropins promotes their translocation either to the cytosol (PHOT1) or to Golgi membranes (PHOT2). Phototropins carry FMN-binding LOV1 and LOV2 domains at the N-terminus, and a Ser/Thr-kinase domain required for autophosphorylation upon illumination with B at the C-terminus [5]. Multiple phosphorylation at the C-terminus leads to dimerization and activation of phototropins. In the dark, phototropins lack kinase activity, until the absorption of a light quantum by the chromophore within the LOV2 domain enables kinase activation. Apart from phosphorylation at Ser and Thr, B induces structural changes, most notably the partial unfolding of the LOV J α -helix [53]. Within LOV domains, upon absorption of a light quantum of 450 nm wavelength, the oxidized FMN non-covalently bound to the protein forms a covalent bond between a C-atom of FMN and a S-atom of a nearby cysteine residue. This adduct is formed within several microseconds and is unable to B absorption; its formation mediates the transition of the phototropin into the active state. In the dark, the photoreceptor reverts to the inactive state within several tens or hundreds of seconds. An incomplete list of phototropin-regulated functions in plants includes the phototropism of shoots, roots and leaves, migration of chloroplasts in response to light and temperature signals, and the movement of stomata [6, 54]. The regulatory mechanism best studied is the opening of sto-

mata in response to B: phototropins activate the BLUS1 (blue-light signaling 1) kinase specific for guard cells, which in turn activates the plasma membrane H⁺-ATPase leading to hyperpolarization of the membrane and inward currents of potassium via inwardly-rectifying channels, swelling of guard cells and stomata opening [55].

As the rate of the so-called 'dark' reactions of photosynthesis depends on the temperature, one and the same light level can result in a different 'excitation pressures' on the photosystems depending on the ambient temperature. Therefore, under similar light conditions, at low temperatures chloroplasts move to the anticlinal cell walls of mesophyll cells, while at high temperatures they move to the periclinal cell walls [54]. This observation suggested that phototropins in plants can function as thermosensors. Using *Marchantia polymorpha*, an organism containing only a single copy of the *MpPHOT* gene, it was demonstrated that the migration of chloroplasts, of the nucleus and of peroxisomes in response to the lowering of the ambient temperature is controlled by B and *MpPHOT* [54]. Combination of B and low temperature leads to an increase in the autophosphorylation levels of *MpPHOT* and in the half-life of its activation state. Together, this promotes the migration of the chloroplasts to the anticlinal cell walls, in order to prevent photooxidation [54, 56]. Thus, phytochromes and phototropins are plant thermoreceptors functioning under different light conditions and on different time scales (the life time for the activated form of the photoreceptor is several tens of minutes for phytochromes but only tens of seconds for phototropins) [54].

The ZEITLUPE family proteins ZTL, FKF1 and LKP1 are E3-ubiquitin ligases which are activated by illumination with B and facilitate the ubiquitination and proteasome degradation of their specific substrates [6, 57]. The N-termini of these proteins contain a single LOV domain with the FMN chromophore, followed by an F-box domain responsible for specific binding of the E3-ubiquitin ligase to its substrate, and by a KELCH domain mediating protein-protein interactions. ZTL exerts control over circadian rhythms, and FKF1 controls the transition to flowering, while LKP1 is required for both processes. Photoactivation of the LOV domain changes the affinity of these proteins to the regulatory protein GIGANTEA (GI), and also leads to an increase in the levels of E3-ubiquitin ligase activity. The GI-FKF1 complex triggers the degradation of CDFs (Cycling DOF Factors), the repressors of *CO* (*CONSTANS*) gene expression. *CONSTANS*, in turn, activates transcription of the *FT* gene encoding 'florigen', the main activator of flowering in *Arabidopsis*. The GI-ZTL complex, on the contrary, restricts the capacity of ZTL to ubiquitinate its substrates, components of circadian oscillator. In the ZEITLUPE proteins, similar to phototropins, the photoactivation of the FMN chromophore promotes formation of a cysteinyl adduct, but the degradation of

this adduct is slow in FKF1 (the life time of the adduct can reach several days), while in ZTL it occurs with the same rate as in phototropins [6, 57]. In the dark, ZTL mediates the degradation of Timing of CAB expression 1 (TOC1), the repressor of core circadian genes, while B inhibits it: GI binds to ZTL leading to the accumulation of TOC1. Similarly, GI binds to FKF1 (after its activation by B), but this leads to an increase in the ubiquitin ligase activity of the latter, and, as a result, the long day photoperiod leads to the degradation of CDFs, the repressors of *CO* and *FT* genes, via the FKF1-GI complex [6, 57].

Cryptochromes, another class of B photoreceptors, evolved as FAD-dependent DNA photolyases, i.e. as the enzymes repairing breaches in DNA caused by B. In land plants, some cryptochromes, e.g. cryptochromes of the CRY-DASH group (*Drosophila*, *Arabidopsis*, *Synechocystis*, *Homo*), are capable of binding DNA and still retain the DNA photolyase activity [58]; notably, these cryptochromes thus far have not been shown to function as photoreceptors. The PHR (photolyase homology region) domain binds two chromophores: FAD which absorbs light of 320–500 nm, and pterine (5,10-methenyltetrahydrofolate) which functions as an antenna transferring the energy of near UV light (370–390 nm) to FAD [11]. With oxidized FAD, a cryptochrome is in its inactive form; after photoreduction, protonation and formation of a neutral semiquinone FADH, the photoreceptor has achieved its active conformation [59]. Further reduction of the chromophore to FADH₂ (e.g. upon absorption of green light quanta) again promotes the transition of the cryptochrome to its inactive form [59]. However, fully reduced FADH₂ is required for DNA photolyase activity. Cryptochromes bind AMP and ATP which might stabilize the active semiquinone-containing form of the photoreceptor [6]. Within cryptochromes, there is an intramolecular electron transport chain: electrons are transferred from tryptophan residues to FAD after photoactivation of the latter; an aspartyl residue within the polypeptide chain provides a proton for FAD protonation. The C-termini of cryptochromes (except for CRY-DASH cryptochromes) contain a highly variable signaling CCE (cryptochrome C-terminal extension) domain [11]. Although cryptochromes do not contain kinase domains, their activation requires multiple phosphorylation events at the C-terminus, followed by dimerization. The activation leads to the exposure of the so-called NC-motif at the C-terminus which consists of 80 amino acids. Expression of a cDNA encoding only these 80 amino acid residues can complement the late flowering phenotype in *Arabidopsis cry* knock-out mutants [11].

In *Chlamydomonas reinhardtii*, single cryptochrome perceives both B and R. The *Physcomitrella patens* genome contains two cryptochrome genes, while the genome of the fern *Adiantum capillus-*

veneris contains five. In rice, there are two CRY1 and one CRY2 cryptochrome while wheat contains two CRY1, one CRY2 and one CRY-DASH [11]. There are three cryptochromes in *Arabidopsis*: a photostable CRY1 activated by high light, a photolabile CRY2 which perceives only very weak light and undergoes degradation in high light, perhaps via the COP1-SPA1 E3-ubiquitin ligase complex [60], and CRY3 which in *Arabidopsis* belongs to the CRY-DASH group. CRY1 and CRY2 localize to the nucleus while CRY3 occurs in chloroplasts and mitochondria. Cryptochrome-based light responses are best studied for *Arabidopsis*. In natural shade, cryptochromes sense the B : G ratio [61, 62]. It was shown that the SAS can be elicited not only via a decrease in the R : FR ratio but also via an increase of the G proportion in the incident light; the latter mechanism involves cryptochromes [12, 61, 62]. Notably, Zhang et al. [12] have found that some *Arabidopsis* responses to G do not involve phytochromes or cryptochromes but rather a novel, not yet identified photoreceptor.

Under B illumination, the active cryptochromes in the nucleus interact with two groups of transcription factors, PIFs (phytochrome-interacting factors) and CIBs (cry-interacting bHLH), mediating their degradation via the COP1-SPA1 ubiquitin ligase complex. Apart from this, CRY bind to COP1-SPA1 and promote its removal from the nucleus, triggering photomorphogenesis (see below) and the transition to flowering (in combination with other photoreceptors), as the ubiquitinylation and degradation of CONSTANS by COP1 in the nucleus is required for the onset of flowering. The active form of CRY2 binds the CIB1 transcription factor, leading to the activation of the expression of the 'florigen' gene, the *FT*. This process also involves CIB2 and CIB5. Also, in *Arabidopsis*, CRY2 negatively regulates the inhibitor of flowering, PhyB, upon exposure to B or to white light. Thus, CRY2 is the main positive regulator of flowering in *Arabidopsis*. The activation of photomorphogenesis by cryptochromes involves the same transcription activators HY5, HFR1, and HYH, as the activation by phytochromes (see below). Cryptochromes can upregulate the expression of all nuclear genes encoding photosynthetic proteins, such as *CAB* and *RbcS*. CRY1 mediates the greening of roots, i.e. the photomorphogenesis of plastids in root cells, upon illumination with B [63]. When the intensities of B decrease, cryptochromes initiate SAS which, in contrast to SAS induced via phytochromes, is uncoupled from the inhibition of the plant immune responses (see below) [62, 64]; this fact is potentially promising for practical use. Altogether, cryptochromes regulate circadian rhythms, the onset of flowering, B-dependent photomorphogenesis and other functions [6].

In response to illumination with B, *Arabidopsis* CRY1 migrates into the nucleus and activates anion (Cl⁻) channels [11]. This presumably leads to the temporary depolarization of the plasma membrane within

30 s after illumination, although the exact mechanism is unknown. The reduced states of the cryptochrome chromophore, semiquinone $FADH\cdot$ and $FADH_2^-$, can mediate the reduction of oxygen and production of reactive oxygen species (ROS) such as superoxide radical and hydrogen peroxide [11].

The Photoreceptor of Ultraviolet B Light: UVR8

Ultraviolet light of B-range (UV-B) light makes up only a small part of the solar spectrum (less than one percent). Nevertheless, plants possess photoreceptors capable of specific perception of UV-B photons. Currently, only one of such photoreceptors is known in *Arabidopsis*, the UV RESISTANCE LOCUS 8 (UVR8) protein [65]. However, homologous *UVR8* genes occur in all genomes of land plants, mosses and algae sequenced thus far [8]. The UVR8 protein in *Arabidopsis* is a 'beta-propeller', a dimer in its inactive state, located in the cytosol. After absorption of UV-B photons, it dissociates into two active monomers which can migrate into the nucleus. A major portion of the active monomeric form is retained in the cytosol, although its role there is not known. The UV-B-absorbing chromophores are tryptophan residues within the polypeptide chain of UVR8. In dimeric UVR8, the tryptophan residues interact with nearby arginine residues forming 'cation-pi' bonds; the absorption of UV-B quanta by tryptophans breaks these bonds and triggers the dissociation of the UVR8 dimer into monomers [8]. In the dark, the photoreceptor will undergo re-dimerization and inactivation after several hours; this process is facilitated by the regulatory WD40 proteins RUP1 and 2 (Repressor of UV-B photomorphogenesis) [8].

In the nucleus, UVR8 directly binds the promoters of genes encoding transcriptional activators of photomorphogenesis, such as HY5. Furthermore, in the nucleus, UVR8 interacts with the main repressor of photomorphogenesis, the COP1 E3-ubiquitin ligase. However, in contrast with other photoreceptors which bind COP1 to perform 'the inhibition of the inhibitor', COP1 acts as an activator in UV-B mediated signaling [8]. UVR8 can directly interact with COP1 independently of SPA1 and escape the degradation; moreover, although COP1 remains in the nucleus, the elicitation of phototropic responses and UV-B dependent photomorphogenesis occurs including de-etiolation, the arrest of hypocotyl growth, activation of flavonoid biosynthesis, regulation of circadian rhythms and an increase in resistance to pathogens and chewing insects [8]. UV-B light was shown to increase plant resistance to pathogens both dependent and independent of jasmonate signaling, probably by means of biosynthesis of flavonoids; moreover, natural levels of UV-B in the incident light are required for plant immunity responses to be established [14]. Thus, further study of photoreception and signal transduction pathways of UV-B is of special interest for potential applications in agriculture, because the UV-B-medi-

ated increase in plant resistance is not related to growth arrest, as it is the case for jasmonate signaling (see below). In spinach and potato, activation of PhyB led to increased resistance of plants to UV-B [66, 67], suggesting that the signal transduction pathways for PhyB and UVR8 overlap.

COMMON MECHANISMS OF PHOTORECEPTOR FUNCTIONING

In spite of the diversity of plants photoreceptors, their functionality follows several 'common rules'. For phytochromes, cryptochromes as well as phototropins, the active form represents a dimer the formation of which relies on (auto)phosphorylation; the activation is triggered by absorption of shorter-wavelength photon (B or R depending on the photoreceptor type), while inactivation occurs after absorption of longer-wavelength photon (G or FR). For phytochromes, cryptochromes and phototropins, two types of photoreceptors exist: one specifically perceives very low light levels and undergoes photodestruction in high light (for instance, PHOT2, CRY2 and PhyA in *Arabidopsis*), while the other one specifically perceives high light and is photostable (for instance, PHOT1, CRY1 and PhyB in *Arabidopsis*).

Transduction of the light signal perceived by photoreceptors can be mediated by second messengers such as Ca^{2+} , cAMP, cGMP, G-proteins and other components [32]. Transformation of the information on light quality and quantity into metabolic and morphogenetic responses occurs via controlled degradation of transcription factors: the active form of a photoreceptor (phytochrome, cryptochrome or UVR8) enters the nucleus and binds the COP1 E3-ubiquitin ligase, the enzyme which performs ubiquitinylation for the regulators of genetic programs and their degradation via 26S proteasome [60]. In the nucleus, COP1 inhibits photomorphogenesis, regulates circadian rhythms and flowering, plant immunity, interactions between various groups of phytohormones, movements of stomata and other functions. Photoreceptors induce the removal of COP1 from the nucleus, and thereby switch on or off genetic programs [60]. Apart from the COP1-dependent pathway, the active forms of photoreceptors can directly bind transcriptional repressors in the nucleus, interfering with their action, another mechanism leading to activation of a number of genetic programs in the cell [27]. The ZEITLUPE family proteins do not interact directly with COP1 but instead represent themselves E3-ubiquitin ligases which, similar to COP1, enter the nucleus and mediate the degradation of major regulatory proteins via the proteasome. Thus far, it is unknown whether phototropins can be transcriptional regulators of a number of genes. A recent study in tomato showed the phototropin-dependent regulation of more than hundred genes including those encoding proteins required for chromatin reorganization, regulation of transcription

and translation [68]. However, the underlying mechanism remains unknown; it possibly involves phytochrome signaling [69].

The inhibition of COP1 in the light results in the accumulation of various transcription factors in the nucleus, which activate photomorphogenesis. Of these, the bZIP transcription factors HY5 and HYH, the bHLH transcription factor HFR1 and the MYB-protein LAF1 are central for the regulation of transcriptional activity by light and temperature signals [20]. In the dark, these proteins undergo COP1-mediated proteasomal degradation. Furthermore, in the dark, these master regulators interact with PIFs (Phytochrome-interacting factors), another system controlling plant responses to changing light conditions.

Phytochrome-interacting factors (PIFs) are bHLH transcription factors. The PIF1, PIF3, PIF4, PIF5, PIF6, PIF7 and PIF8 proteins activate scotomorphogenesis and inhibit photomorphogenesis throughout the life of the plant, mediating light-dependent growth regulation; even in high light, a basal level of PIFs activity is required to maintain growth [70]. PIFs enable growth by multilayered regulation of several classes of phytohormones. They mediate the expression levels of the *YUCCA* genes which encode the enzymes of auxin biosynthesis [71]; also, PIF3 and PIF4 bind DELLA proteins, the negative inhibitors of gibberellin-responsive genes, while PIF5 activates the accumulation of repressors of gibberellin biosynthesis. Furthermore, DELLA and PIFs directly bind BZR1, the regulator of brassinosteroid-responsive genes [13]. Notably, activation of the biosynthesis of auxin and gibberellins via PIFs occurs in response to a decrease of the R proportion in the incident light, while the PIFs-mediated activation of brassinosteroid signaling occurs in response to a decrease of the B proportion [62]. Another type of interaction between DELLA and PIFs mediates the growth arrest that occurs as part of the plant stress response induced via jasmonate signalling [70]. In the absence of jasmonates, the JAZ repressors bind the transcription factors MYC2/3/4, the activators of jasmonate-inducible genes, inhibiting their functions [72]. JAZ proteins also interact with DELLA, thereby partially decreasing the levels of JAZ available to bind MYC factors, and leading to a further decrease in the levels of DELLA-PIFs complexes; the unbound PIFs thus can function in growth promotion. The biosynthesis of jasmonates induces the degradation of JAZ and thereby increases the binding of DELLA to PIFs, leading to a decrease in the growth rate. This way, the balance between the MYC-dependent activation of jasmonate-responsive genes and PIF-dependent growth in low light is determined to a certain extent by gibberellin biosynthesis and DELLA proteins; gibberellin binding DELLA activates plant growth via PIFs and reduces the expression levels of jasmonate-responsive genes [72].

PIF transcription factors contain DNA-binding domains and domains mediating the interactions with phytochromes: the APB (Active PhyB binding) domain, and, in case of PIF1 and PIF3, also the APA (Active PhyA binding) domain. PhyA was shown to interact directly with PIF1 and PIF3, while PhyB interacts with most PIFs. When bound to PIFs, phytochromes promote phosphorylation, ubiquitinylation and proteasome degradation of the latter in the course of several minutes, while also inhibiting the binding of PIFs to the promoters of target genes [13]. Recent studies reveal that PIFs represent master switches of plant development, a regulatory hub receiving signals not only from phytochromes but also from other photoreceptors: cryptochromes and UVR8 [71, 73, 74]. Altogether, very different signals from most photoreceptors in plants are transduced to three major regulatory modules: the repressor of photomorphogenesis, COP1; the activator of photomorphogenesis, HY5; and the systemic integrators, PIFs (Fig. 1).

THE 'SHADE AVOIDANCE SYNDROME' AND ITS IMPORTANCE FOR CROP PRODUCTION

The shade avoidance syndrome (SAS) is a scotomorphogenetic program activated either by a decrease in the R : FR ratio in the incident light, or by a decrease in the total amounts of R, B, white or UV-B light, and, according to recent data, also by a decrease in the B : G ratio in the incident light [62]. All these changes are typical for light conditions under a dense canopy [4, 13]. This suggests that all photoreceptors participate in the establishment of SAS. Notably, a decrease in the R : FR ratio in the incident light can induce SAS even in high light: this makes plants aware of neighbors, i.e. the future competitors for the light resource, before light becomes limiting, and leads to the preemptive acceleration of growth and the induction of other adaptive reactions of the SAS program. A decrease in the R : FR ratio in the incident light occurs because of the absorption of R and reflection of FR by green plant parts; as a result, plants perceive the FR-enriched light horizontally reflected by stems of neighboring plants [4]. The decrease in the R : FR ratio promotes the inactivation of PhyB, the main inhibitor of the SAS in high light. In the shade, the main inhibitor of SAS is PhyA, specifically its dephosphorylated, relatively photostable form which appears in the cell in course of the activation of the HIR. The induction of SAS is mediated via COP1-dependent degradation of HY5 and HFR1, the activators of photomorphogenesis, and also via activation of PIF4- and PIF5-regulated genes.

SAS activation leads to the elongation of petioles, leaf hyponasty, arrest of width growth combined with acceleration of length growth of leaf laminae; elongation of internodes, retardation of chlorophyll biosynthesis and other scotomorphogenic reactions. During the SAS

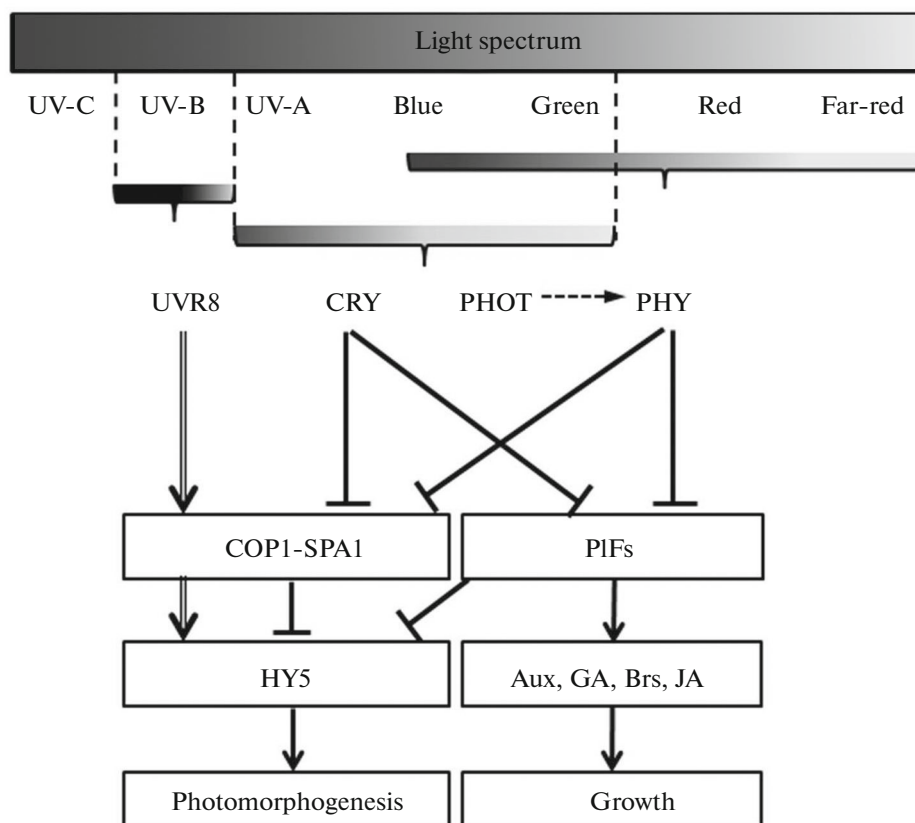


Fig. 1. A simplified scheme of the integration of signals elicited from photoreceptors activated by various regions of the light spectrum in *Arabidopsis thaliana*. Arrows show activation and inhibition, respectively. The double arrow shows the direct interaction of UVR8 with COP1 and the induced response. The hatched arrow shows interaction between phototropin-based and phytochrome-based signaling. Aux—auxins; GA—gibberellins; Brs—brassinosteroids; JA—jasmonates.

in monocot crops, the transcription factor GRASSY TILLERS increases the dormancy of lateral buds and inhibits tillering; GRASSY TILLERS is activated via another protein, TEOSINTE BRANCHED, which is inhibited by PhyB [75]. Activation of the SAS inhibits the emission of jasmonate-inducible volatiles, probably because of a decrease in active phytochrome levels in the cells [76]. The establishment of the SAS also involves mechanosensitive channels [4]. Mechanostimulation increases the strength of SAS symptoms in seedlings while decreasing it in mature plants. This can be explained by the fact that for seedlings, mechanostimulation signals the presence of neighbors, while for a mature plant, it indicates that the plant has grown over the grass canopy, where natural mechanostimulation is increased due to air migration and windblasts, and thus has escaped the potential competition from neighboring plants [4].

Jasmonates inducing plant defense against necrotrophic pathogens and chewing insects, and salicylates inducing defense against biotrophic pathogens, represent the backbone of plant immunity [14]. In plants growing in the shade compared to light grown plants, the extent of biotrophic and necrotrophic infections is higher, and feeding larvae are bigger [14]. In fact, SAS

induced by a decrease in the R : FR ratio often includes a decrease in plant pathogen resistance due to the weakening of responses to jasmonate signaling [14]. Furthermore, the inactivation of PhyB in the shade leads to a reduction of the biosynthesis of salicylic acid [14, 77]. Earlier, these phenomena have been interpreted as a means to redirect the energy and other plant resources from defense to growth, to outrun the competitors for light availability [14].

In crops, the induction of SAS results in a decrease in yield: for instance, in cereals, it leads to a redirection of the resources from leaves and ears to 'economically not significant' stems. At the same time, during thousands of years of domestication, along with other important traits, crops were selected for the ability to produce high yield at dense planting, i.e. for weakening of the SAS [78]. Thus, the question of how to inhibit the SAS and enable a further increase in planting density for crops, simultaneously with a change in biomass balance in favor of economically important plant organs, while not inhibiting plant immunity, is a primary issue for breeders and bioengineers. In this context, the possibility to manipulate plant responses to light, including 'switching off' the SAS, is of special interest [79]. For this purpose, Wille et al. [80] per-

formed chemical mutagenesis of six varieties of spring wheat followed by multiple screening steps, first with a green light filter and then under conditions of a decreased R : FR ratio, to identify the wheat mutant lines showing no growth acceleration under these conditions. After analysis of more than 1000 lines, five mutants were obtained displaying a high extent of reduction of the SAS: at higher planting density, they showed good growth, early flowering, wider leaves and higher biomass than the parent varieties under similar conditions. In another study aimed at revealing the components of signaling pathways regulating the SAS, the authors for the first time used phenotypic profiling [81], resulting in the identification of 18 new SAS-mediating genes (among them, the gene encoding a guard cell-specific potassium channel KAT1), and the description of three groups of genes controlling three types of shade responses, respectively: the genes controlling hypocotyl elongation, the genes controlling petiole elongation, and the genes controlling the transition to flowering. It should be pointed out that, because of the convenience of using a decreased R : FR ratio to induce the SAS, the most approaches targeted the SAS induced by inactivation of PhyB while studies of cryptochrome-induced or UVR8-induced SAS lag behind, although they also should have the potential for agricultural use [64, 77].

INTERACTIONS BETWEEN PHYTOCHROME-MEDIATED AND JASMONATE-MEDIATED SIGNALING

As discussed above, the induction of SAS can occur either by a decrease of the proportion of the active form of the main SAS inhibitor, PhyB, due to an increase of the FR ratio in the incident light, or of other photoreceptors like CRY1 or UVR8, in response to a decrease of light of the respective wavelength. A decrease in the active forms of photoreceptors in cells leads to the activation of PIFs and of PIF-based scotomorphogenetic reactions including accelerated elongation growth. The mediation of SAS involves almost all phytohormones [82]. In the shade, gibberellin levels rise [82], increasing the degradation rate of the DELLA proteins. In high light, DELLA proteins bind PIFs and interfere with the induction of SAS; however, DELLA proteins also interact with another group of transcription factor repressors, JAZ [72]. JAZ proteins inhibit the transcription factors MYC2/3/4, the activators of jasmonate-responsive genes including genes encoding enzymes involved in the biosynthesis of volatiles and responsible for growth arrest. In *Arabidopsis*, there are up to 10 JAZ proteins performing similar functions [83].

More and more studies reveal a tight interaction between phytochrome- and jasmonate-based regulatory systems [83–91]. The biosynthesis of the conjugate of jasmonate with isoleucine promotes the degradation of JAZ proteins via the E3-ubiquitin ligase complex

COI1 (CORONATINE-INSENSITIVE 1) [72]; however, recent studies have shown that this degradation requires a presence of PhyA [84]. Furthermore, the JAZ10 protein is required for the perception of the R : FR ratio and the inhibition of PhyB [83]. The active form of the JAZ10.4 protein is a product of alternative splicing [83]; it would be interesting to pinpoint the role of phytochromes in the regulation of splicing of the JAZ10.4 transcripts. In an *Arabidopsis* mutant, the absence of phytochromes resulted in an increase in the biosynthesis of jasmonate [76], while in a rice mutant, the inhibition of jasmonate biosynthesis changed the ratio of different spectral forms of PhyA [86, 87].

The identification of signals and mechanisms underlying the growth arrest under stress conditions is of special interest for agriculture [91–94]. The leading role in these phenomena belongs to jasmonate-regulated genes, and the biosynthesis of jasmonates is strongly induced under stress [89, 91, 93]. A recent study demonstrated that growth arrest and the onset of stress response reactions are two different genetic programs which function in close cooperation, but nevertheless can be completely separated in mutants of respective key regulators of these programs [89]. Such mutants of *A. thaliana* displayed high levels of stress resistance and growth vigor under stress conditions, similar to, or even exceeding (probably due to transgressive effects), that of wild-type plants in the absence of stress [89]. In this study, jasmonic acid was the inductor of stress responses and PhyB was the activator of the growth arrest in response to jasmonate signaling. Notably, manipulations of the levels of phytochrome activity influence not only the growth of shoots, but also of roots: the role of phytochromes (those occurring both in shoot and root cells) in the growth of roots was recently demonstrated [92]. The root phytochromes participate in root gravitropism and in the change of root growth patterns, including the perception of jasmonate signals [95]. The attempts to manipulate the expression levels of phytochrome genes in transgenic plants in order to shift the balance of biomass production to economically important plant organs had begun earlier and continue until now [96].

CONCLUSION

The research on the control of plant growth and development via photoreceptor systems, and their functions under stress conditions, currently experiences a boost. These studies provide a potential basis for breeding of crop varieties capable of high yield production under stress conditions.

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COMPLIANCE WITH ETHICAL STANDARDS

The authors declare that they have no conflict of interest. This article does not contain any studies involving animals or human participants performed by any of the authors.

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