REVIEWS

Role of Phytohormones and Light in De-etiolation

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Abstract—De-etiolation or transition from etiolated growth (skotomorphogenesis) to photomorphogenesis is one of the most intriguing and intricate stages of plant ontogenesis. It comprises reprogramming of plant cell metabolism, reorganizing the operation of the hormonal system, and altering plant morphology. Dark growth in the soil mainly depends on phytohormones with gibberellins and brassinosteroids playing the leading role; on the soil surface, light as a major exogenous agent starts operating. It inhibits activity of the main repressor of photomorphogenesis (COP1) and regulators of transcription, which govern realization of gibberellin (DELLA) and brassinosteroid (BZR1/BES1) signals and activates *trans*-factors initiating transition to autotrophic nutrition (for instance, HY5). The strategy of etiolated growth consists in achieving a quick exposure to sunlight at the expense of active elongation of the stem. For transition to autotrophic nutrition, a plant must form a photosynthetic apparatus and protect itself from possible light injury. This review deals with the role of the main regulatory components ensuring etiolated growth and transition to photomorphogenic development.

Keywords: photoreceptors, *trans*-factors, phytohormones, photomorphogenesis, etiolation

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INTRODUCTION

Depending on the absence or availability of light, young seedlings pursue one of the two programs of development: etiolated growth (skotomorphogenesis) or de-etiolation (photomorphogenesis), respectively [1–3]. Etiolated dicotyledons produce an apical hook for protection of the apical meristem; they lack leaves and have proplastids and etioplasts instead of chloroplasts. In the case of etiolation, the bulk of storage substances of the seed are spent on the quick growth of the hypocotyl. Prolonged stay in the dark may result in depletion of nutrients and irreversible etiolation, which may cause a loss of seedlings. Etiolation is achieved by inhibition of expression of the genes responsible for

photomorphogenic development. A leading regulatory role in etiolation is performed by phytohormones and, first of all, by gibberellins (GA), brassinosteroids (BR), auxins, and ethylene [4, 5]. *Trans*-factors of these hormones induce expression of the genes responsible for growth. In the dark, numerous regulatory proteins not only activate growth but also suppress the activity of other proteins that try to set the plant on the track of photomorphogenesis. Such inhibitors primarily comprise the main repressor of photomorphogenesis COP1 (CONstitutive Photomorphogenesis 1) and different components of the 26S proteasome complex as well as *trans*-factors of the bHLH (basic Helix-Loop-Helix) family of PIF (Phytochrome Interacting Factors).

As soon as the seedling is exposed to light, the process of de-etiolation starts (Fig. 1), and genome expression and plant metabolism are reorganized. This occurs owing to the participation of photoreceptors and positive regulators of photomorphogenesis (HY5—elongated HYpocotyl 5; GAI—Gibberellic Acid Insensitive; RGA—Repressor of GAI—3, et al.) and due to changes in the role of phytohormones; for instance, the role of GA and BR becomes considerably less important, but the role of cytokinins (CK) rises, at least in formation of the photosynthetic apparatus. Activity of proteins ensuring etiolated growth is also suppressed [6].

In order to avoid vagueness, we will use the term “etiolation” only in respect to plant growth under complete lack of light. The French word *étolier* stands

Abbreviations: BES1—BRI1-EMS-suppressor 1 or BZR2; bHLH—basic helix-loop-helix; BIN2—brassinosteroid insensitive 2; BR—brassinosteroids; BZR1—brassinazole-resistant 1; Chlide—chlorophyllide; ChlH—Mg-chelatase H subunit; CK—cytokinins; COP1—constitutive photomorphogenesis 1; CRY—cryptochrome; DELLA domain—Asp-Glu-Leu-Leu-Ala motif; FHL—FHY1-like protein; FHY—far-red elongated hypocotyl 1; EIL1—ethylene insensitive 3-like 1; EIN3—ethylene insensitive 3; GA—gibberellins; GID1—gibberellin insensitive dwarf 1; GNC (GATA, nitrate inducible, carbon-metabolism involved) and GNL/CGA1 (GNC-like/cytokinin-responsive gata factor 1)—*trans*-factors of GATA type; HFR1—long hypocotyl in far-red 1; HY5—elongated hypocotyl 5; HYH—HY5 homolog; JA—jasmonic acid; Pchlide—protochlorophyllide; PHYA (B, C, D, E)—phytochromes A (B, C, D, E); PIF—phytochrome-interacting factor; Pr—inactive form of phytochromes; Pfr—active form of phytochromes; PORA (B, C)—protochlorophyllide oxidoreductase A (B, C); SPA1—suppressor of phyA-105; TBP—tetrapyrrole biosynthesis pathway.

Fig. 1. Participation of phytohormones and light at the early stages of plant ontogenesis. Usually plant lifecycle comprises a period of etiolated growth and photomorphogenesis. The stage of etiolated development mainly depends on phytohormones and, first of all, on GA and BR with participation of PIF and BZR1/BES1 *trans*-factors that induce expression of the genes responsible for hypocotyl growth. In the dark, the central repressor of photomorphogenesis COP1 shows the greatest activity and suppresses any manifestations of de-etiolation. When the seedling gets to the soil surface, light inhibits GA synthesis. This leads to the accumulation of DELLA proteins that inactivate PIF and brassinosteroid-dependent *trans*-factors terminating the program of etiolation. Phytochromes inactivate and/or destroy COP1 and PIFs and cause accumulation of protein factors triggering photomorphogenesis, such as HY5. This train of events results in plant transition from etiolation to photomorphogenesis. The picture was taken from Jaillais and Vert [6] and partially modified.

for straw, which approximately corresponds to the color of etiolated plants. Some problems concerning photomorphogenesis have been discussed in a recent review by Armarego-Marrriott et al. [3]. De-etiolation is morphophysiological process associated with suppression of shoot growth, apical hook opening, emergence of leaves, and formation of the photosynthetic apparatus. De-etiolation and photomorphogenesis are synonyms. Greening applies only to some events associated with de-etiolation. There is a question without a single meaning: when does de-etiolation stop? Some researchers believe that de-etiolation comes to the end when the first photosynthetically active true leaves are formed; we share this point of view. Wei et al. [7] proposed an interesting hypothesis about the origin of etiolation. They assumed that all the plants originally developed along the path of photomorphogenesis, and etiolation arose later in the course of evolution as a suppressed photomorphogenesis. However, there are reports that cannot be accounted for by this interesting hypothesis.

In this review, we will consider a scenario of plant development when etiolated growth is subsequently followed by transition to photomorphogenesis. We will describe the major participants in the process of de-etiolation, including phytohormones and components of their signal networks, light receptors, and interaction between the components of light and hormonal signaling. In order to better understand the mechanism of transition from etiolation to photomorphogenesis, we will discuss peculiarities of etiolated plant development.

GROWTH AND DEVELOPMENT OF ETIOLATED SEEDLINGS IN THE SOIL

When the seeds are placed into the soil, development of the seedling starts in the dark and its growth primarily depends on phytohormones. GA and BR accelerate etiolated growth and promote etiolation, while CK, ethylene, and jasmonic acid (JA) in different ways suppress them by acting as positive regulators

of photomorphogenesis. Actual growth will reflect a resultant of joint regulatory action of these phytohormones.

In order to optimize the growth rate, the seedlings must somehow perceive the impact of the soil and determine how deep they are located in the soil and what soil (light or heavy) is around. It is assumed that they respond to soil properties by means of ethylene [8]. Mechanical impact of soil on the seedlings of *A. thaliana* brings about a rise in the content of ethylene and the main components of ethylene signaling EIN3 (Ethylene INsensitive 3) and EIL1 (Ethylene INsensitive 3-Like 1). Their production directly correlates with soil depth and density. *Arabidopsis* mutants *ein3/eil1* lose sensitivity to ethylene and cannot get through the soil layer of 3 mm, although they germinate and develop well without soil cover. EIN3/EIL1 activate ERF1 (Ethylene Response Factor 1) that decelerates hypocotyl growth [9]. This leads to the formation of the seedlings with shorter and thicker hypocotyls, which promotes their penetration through the soil layer.

MORPHOLOGICAL FEATURES OF ETIOLATED SEEDLING

In the course of etiolated growth, dicotyledons form an apical hook that protects the apical meristem of the shoot and cotyledons from damage upon getting through the soil layer. There exist three phases of hook development: formation, preservation, and opening [10]. Formation of the hook depends on differential growth of the cells located on the external and internal sides of hypocotyl caused by asymmetrical distribution of auxin. When auxin gradient dissipates, the hook is not produced [11, 12]. Ethylene and GA also participate in the development of the apical hook. Ethylene promotes the creation and maintenance of an auxin concentration gradient [13]. GA modifies the role of auxin and ethylene in hook development [14]. Gene *HLS1* (*HOOKLESS 1*) encoding N-acetyl transferase was identified; its inactivation made it impossible to produce the hook [11, 15]. An et al. [15] believe that ethylene and GA that modify expression of gene *HLS1* regulate formation of the apical hook. Thus, auxin, ethylene, and GA participate in the development and maintenance of the apical hook in the course of etiolated growth. In the light, auxin gradient dissipates in 4 h, and apical hook starts opening [10]. Along with light, the hook opening also depends on auxin, ethylene, and GA [11, 12, 15, 16].

GIBBERELLINS AND BRASSINOSTEROIDS ACTIVATE HYPOCOTYL GROWTH

In etiolated plants, the hypocotyl grows quickly and predominantly by means of extension. Extension of cells is realized via increased water uptake, cell volume, and turgor pressure. As we noted above, the

greatest effect on the growth of an etiolated hypocotyl was produced by GA and BR.

Gibberellins are diterpenoid plant hormones affecting many aspects of plant growth and development. GA promotes etiolated growth and suppresses photomorphogenesis. Inactivation of the genes for GA biosynthesis and signaling in the dark results in the formation of a deetiolated phenotype [17]. Key components of GA signaling in *A. thaliana* are soluble receptor GID1 (Gibberellin Insensitive Dwarf 1), growth inhibitors DELLA, and F-box proteins SLEEPY1 (SLY1). GA participates in growth regulation via degradation of DELLA proteins [18].

DELLA proteins are regulators of transcription and repressors of almost all GA-dependent processes [19]. Inactivation of DELLA genes (*dellaKO*) completely suppresses the GA-deficient phenotype. DELLA proteins have a highly conserved C-terminal GRAS domain participating in the regulation of transcription [19] and DELLA domain responsible for GA-dependent interaction with receptor GID1. *A. thaliana* has five DELLA proteins; however, we are particularly interested in GAI (Gibberellic Acid-Insensitive) and RGA (Repressor GA 1-3) suppressing growth.

How do gibberellins work? When GA is absent or present at a low concentration, DELLA proteins accumulate and suppress GA-regulated processes in plants. These proteins do not have a DNA binding site and cannot directly participate in the regulation of gene expression; however, they interact with key regulatory proteins responsible for numerous processes of growth and development, form heteroduplexes with them, and suppress the ability of these *trans*-factors to participate in the regulation of genome expression.

In the dark, GA concentration is usually higher and the complex of GA-GID1-DELLA is produced, which interacts with SLY1 bringing about ubiquitination and proteasome degradation of DELLA proteins [20]. In the absence of DELLA, GA can realize its regulatory programs. In this case, *trans*-factors, such as PIF3, PIF4, BZR1 (BrassinaZole Resistant 1), etc., will stimulate expression of the genes participating in cell elongation, including the genes encoding enzymes of biogenesis and modification of cell wall components and/or enzymes responsible for modification of cell wall structures [14]. Low levels of DELLA resulting in a high activity of PIF cause a rapid growth of hypocotyl. Since DELLA interact with numerous regulators of transcription [16, 19], GA controls the expression of a great number of genes involved in different metabolic and signal pathways in the course of etiolated growth. Therefore, GA activates growth in the dark in at least two ways: it maintains a low level of HY5 and causes the degradation of DELLA proteins.

Brassinosteroids are steroid hormones that are found in all the plants and that possess a wide range of functional activities. Deetiolated phenotype of BR-deficient

or insensitive mutants (*bri1*) corroborates their negative role in photomorphogenesis. By now, all the main components of BR signal transduction from recognition by a receptor to activation of *trans*-factors that regulate the expression of thousands of BR-dependent genes have been identified [21–23]. Interaction between BR and membrane receptor kinase BRI1 (BRassinosteroid-Insensitive 1) is followed by interaction with kinase BAK1 (BRI1-Associated Kinase 1). A cascade of phosphorylation is then triggered resulting in activation of phosphatase BSU1 (BRI1 SUPpressor 1) of the PP1 type, which dephosphorylates and inactivates BIN2 (Brassinosteroid-INsensitive 2) kinase [24]. BIN2 plays a negative role in BR signaling. It phosphorylates two homologous *trans*-factors BZR1 and BES (BRI1-Ems Suppressor 1 also known as BZR2), which suppresses their transcriptional activity. BZR1/BES1 become incapable of binding to DNA and producing dimers with other *trans*-factors. Moreover, phosphorylated BZR1/BES1 migrate from the nucleus to cytoplasm.

At elevated levels of BR, BIN2 is inactivated by means of dephosphorylation. BES1 and BZR1 are dephosphorylated by cytoplasmic protein phosphatase 2A (PP2A). Dephosphorylated *trans*-factors are more stable, they accumulate in the nucleus where they interact with BRRE (5'-CGTG (T/C) G-3') and E-box (5'-CANNTG-3') elements of promoters of BR-dependent genes and regulate their expression. The examined data show that BRs exert regulation predominantly via kinase BIN2 and two *trans*-factors BZR1/BES1, and the nature of their regulation depends on the level of BR in the cell.

BRs regulate the expression of approximately 4000–5000 genes [25]. They suppress transcription of components of light signaling. Comparison of light-regulated genes with the genes regulated by BRs shows antagonistic relations between light and BRs. BZR1 and BES1 may act as activators or repressors of transcription. They regulate the expression of approximately 200 genes of *trans*-factors. Direct target genes for BZR1 and BES1 are the genes participating in biosynthesis or transduction of signals of GA, ABA, ethylene, CK, and JA; this means that, apart from BR-specific genes, BRs interact with different phytohormones in regulation of physiological processes [25]. BRs induce extension growth by regulating the expression of the genes participating in modification of the cell wall, water transport, and reorganization of the cytoskeleton. BZR1 interacts with PIF4 and activates growth in the course of etiolation [26]. BZR1 and PIF4 share approximately 2000 target genes, and many of them are regulated by both proteins. *Trans*-factor GLK (Golden2-Like) participating in the development of chloroplasts is a direct target for BES1 and is inhibited by it, which hampers chloroplast development [25]. Many genes for signal and metabolic pathways are direct targets for BZR1 and BES1, which causes the

regulation of a high number of genes and reprogramming of plant growth and development.

MAIN FACTORS GOVERNING ETIOLATION

Although the growth of etiolated seedlings highly depends on GA and BR, the most important role in plant development in the absence of light belongs to COP1 (the main repressor of photomorphogenesis) and a small family of PIF *trans*-factors.

COP1 is a chief repressor of photomorphogenesis; it possesses E3 ubiquitin ligase activity and, together with SPA (suppressor of PHYA-105) proteins, identifies substrate proteins for ubiquitination and subsequent degradation by 26S proteasome. COP1 is crucial for the maintenance of etiolation [27]. The molecule of COP1 has three specific domains that mediate dimerization, interaction with regulatory proteins, partner proteins, and substrates [28, 29]. SPA proteins (SPA1–SPA4) in the COP1-SPA1-E3 ligase complex are cofactors and perform a regulatory function, whereas COP1 plays a catalytic role. Modification of the set of SPA proteins within the complex affects COP1 function [29]. The plants bearing mutations in the genes *cop1*, *det1*, and *spa1234* or in the genes for other proteins of these complexes show a deetiolated phenotype.

In the dark, the COP1-SPA1 E3 ubiquitin ligase complex is located in the nucleus and with the assistance of 26S proteasome participates in degradation of *trans*-factors promoting photomorphogenesis, such as HY5, HYH (HY5 Homolog), HFR1 (long Hypocotyl in Far-Red 1), and LAF1 (Long After Far-red light 1) [29]. Their degradation brings about the accumulation of growth-stimulating *trans*-factors, such as PIF3 [30]. It was shown that PIF1 interacts in the dark with COP1 and elevates the activity of E3 ligase for degradation of HY5. Ling et al. [26] detected nonproteolytic stabilization of PIF3 in the dark with the participation of COP1/SPA. They showed that kinase BIN2 phosphorylates PIF3 with subsequent degradation via 26S proteasome in the course of skotomorphogenesis. However, COP1/SPA can interact with PIF3 via SPA1 and impair interrelation BIN2-PIF3, which inhibits BIN2-mediated phosphorylation and degradation of PIF3. It was also shown that ABI4 (ABA-Insensitive 4) activated expression of gene COP1, which contributed to suppression of de-etiolation [31]. COP1 directly interacts with phytochromes and cryptochromes [29].

One may conclude that COP1/SPA activity is necessary for the maintenance of etiolation. In the dark, this complex induces degradation of phytochromes and numerous *trans*-factors initiating transition to photomorphogenesis but stabilizes regulators of transcription promoting etiolated development.

PIF is a small family of transcription factors controlling the expression of thousands of genes responsible for etiolated growth and development [32]. Regu-

lation of photomorphogenesis is the most important function common to PIF members that is realized together with components of GA, BR, and light signaling; however, each of these *trans*-factors also performs its specific functions. In *Arabidopsis*, all eight members of the PIF family (PIF1–PIF8) bear an APB (active PHYB-binding) motif, while PIF1 and PIF3 also have an APA (active PHYA-binding) motif [33, 34]. In the dark, mutants deficient for the genes of four PIFs (*pifq*) show a photomorphogenic phenotype [2, 35].

In the dark, PIF1 and PIF3 inhibit photomorphogenesis down-regulating development of chloroplasts and synthesis of chlorophyll. PIF4 and PIF5 affect elongation of hypocotyl regulating the expression of the genes involved in biosynthesis of auxin and GA- and BR-dependent genes associated with growth. Other PIFs play an important role in transition to de-etiolation, and PIF2 and PIF6 are positive regulators of photomorphogenesis. Transcriptome analysis of the mutants of different orders (including *pifq*) showed both overlapping and specifically regulated genes. Preferable *cis*-elements for PIF proteins are *G-Box* (CACGTG) and *PBE* (CATGTG) (PIF-binding E-box, PBE-box) [36]. PIFs regulate the expression of *trans*-factors from different classes. A number of factors (DET1, HECATE2, and COP1/SPA complex) stabilizes PIFs in the dark in order to maintain etiolation, whereas others (BIN2, DELLA, and HFR1) promote degradation of PIFs in the dark. DELLAs induce degradation of PIFs both in the dark and in the light [37]. HFR1 suppresses PIF activity and reduces their content. The issue of PIF phosphorylation that precedes their degradation was discussed in the review [34]. PIFs can interact with DNA both as homo- and heterodimers. Certain agents strengthen or weaken the DNA-binding activity of PIFs. PIF activity is regulated at different levels, including the modification of protein stability, subcellular location, posttranslational modifications, affinity to cDNA, and activity of the DNA-bound complex.

PIFs are stable in the absence of light, active in the absence of DELLA proteins, and can regulate the expression of genes responsible for growth via E-box in their promoters. The expression of several target genes of PIF3 was reduced under GA deficit. In the course of germination, when the level of GA is high and the seedlings are located below the soil surface, PIF proteins free of DELLA repression and PHYB-dependent degradation activate hypocotyl growth [38]. PIFs act as a central unit of numerous signal pathways; therefore, components of these pathways (DELLA, BIN2, EBF1/2, etc.) interact with PIF both in the course of etiolation and photomorphogenesis.

NEGATIVE REGULATORS OF ETIOLATION

The agents reviewed above (GA, BR, COP1, and PIF) promote the development of seedlings in the course of etiolation and suppress any manifestation of

photomorphogenesis. However, each function even an elementary one in the plant is controlled by numerous regulatory factors; most of them are probably still unknown. For instance, GA and BR can ensure rapid growth of hypocotyl; however, hypocotyl will be too weak and incapable of breaking through the soil without the participation of ethylene or CK. Growth regulation in etiolated plants involves at least three hormones that are positive regulators of de-etiolation: CK, ethylene, and JA.

Treatment of *A. thaliana* plants with CK in the dark causes changes in their morphology [39]. Extended cotyledons emerge, true leaves develop, photosynthetic genes are expressed, and etioplasts with the elements of thylakoid membranes arise; that is many features of de-etiolation appears. The role of CK in activation of de-etiolation is corroborated by the results obtained in mutant *ampl* (*altered meristem program*) with an elevated content of CK [40] and in transgenic plants of *A. thaliana* expressing the gene for CK biosynthesis (*ipt*) [41].

This effect of CK resembles triple response to ethylene in *Arabidopsis* plants grown in the dark; therefore, it was assumed that the effect of CK depends on its influence on metabolism and/or signaling of ethylene [42]. However, having examined this effect of CK, Cortleven et al. [43] showed that the inhibitory effect remained both in the presence of inhibitor of ethylene signaling (AgNO₃) and in ethylene-insensitive mutants (*ein2-1*). It was shown that, in the presence of AgNO₃ in the dark, CK altered the level of transcripts of 2463 genes and identical changes occurred in the light. Many CK-regulated genes are related to photosynthesis, synthesis of pigments, light signaling, and components of the cell wall. Ethylene-independent inhibition of hypocotyl elongation by cytokinin occurs with the participation of AHK3 receptor (Arabidopsis Histidine Kinase 3) and type B response regulators ARR1 (Arabidopsis Response Regulator 1) and ARR12 (Arabidopsis Response Regulator 12). This means that retardation of hypocotyl growth in etiolated seedlings by CK is independent of ethylene. It is interesting that inactivation of the major negative regulators of photomorphogenesis COP1, DET1, and CIN4/COP10 (cytokinin insensitive 4/cop10) in the mutants results in the loss of their sensitivity to CK; that is, these inhibitors of de-etiolation are necessary for the suppression of hypocotyl elongation with CK in the dark.

As we have already mentioned, ethylene hampers growth of etiolated seedlings of *A. thaliana* in the dark [9]. JA is an even stronger negative regulator of etiolation [44]; in the seedlings of *Arabidopsis* in the dark, it suppresses the elongation of hypocotyl and stimulates the opening of cotyledons. It stabilizes HY5 owing to inhibiting the activity of COP1 E3 ubiquitin ligase, hampering the accumulation of COP1 protein in the nucleus, and weakening the direct interaction between

COP1 and suppressor *PhyA-105* (*SPA1*), which reduces the activity of COP1. Moreover, in etiolated seedlings, JA regulates the expression of 862 light-sensitive genes.

The cited data suggest that the growth of etiolated seedlings depends on positive and negative regulators of etiolation, which ensure optimization of growth rate depending on numerous factors, including soil structure and the depth of seed accommodation. The seedling must grow quickly but it should be strong enough to get through the soil layer. Specifically these properties of a plant develop upon interaction between many factors.

DE-ETIOLATION

Having got to the surface, plants respond to the quality of light, its intensity and duration of exposure, and can optimize their growth and development in accordance with growth conditions (Fig. 1). Regulatory systems of plant are transformed to ensure the suppression of hypocotyl growth, formation of leaves, development of photosynthetic apparatus, and transition to autotrophic nutrition. Light is added to hormonal regulation primarily with participation of phytochromes and cryptochromes. This process involves positive regulators of photomorphogenesis, such as HY5 and DELLA proteins. Concurrently, the activity of the central repressor of photomorphogenesis COP1 and PIF proteins is suppressed, as well as the activity of major BR *trans*-factors (*BZR1/BES1*), GA biosynthesis is inhibited, and the role of CK becomes more important (especially in chloroplast development). During this period of ontogenesis, the most important aim is the formation of the photosynthetic apparatus crucial for transition to autotrophic nutrition.

WAYS TO OVERCOME PHOTOOXIDATIVE INJURY

An etiolated plant must prepare itself for life under illumination. In light, it can rapidly become autotrophic or quickly perish as a result of photooxidative injury. In the course of de-etiolation, regulatory signals of a different nature must ensure development of the photosynthetic apparatus with simultaneous protection against light injury.

Intermediate metabolites of the tetrapyrrole biosynthesis pathway (TBP) may be a source of reactive oxygen species. In plants, biosynthesis of tetrapyrroles occurs in plastids and all the enzymes of this pathway are encoded in the nucleus [45]. A chlorophyll precursor protochlorophyllide (Pchlde) is dangerous to plants; it accumulates in the dark but may be quickly eliminated in the light owing to NADPH protochlorophyllide oxidoreductases (*PORA-PORC*) that convert Pchlde into chlorophyllide (Chlide) [46]. The quantity of Pchlde must be stoichiometrically related to the level of POR since free Pchlde in the light acts as a

photosensitizer and generates ROS, thereby inducing photooxidative injury.

In order to protect themselves against light injury, plants have developed a complicated system of defense accomplished in two ways: via suppression of TBP enzymes in the dark and/or by activation of POR at the moment when the seedling is exposed to light. It is known that heme inhibits the conversion of glutamate to aminolevulinic acid [47]. GUN1 (Genomes UNcoupled 1) inhibits the expression of gene *HEMA1* (Glutamyl-tRNA reductase) in the dark. A negative regulator of chlorophyll biosynthesis FLU (FLUorescent in blue light) was identified [48]; it directly interacts with glutamyl-tRNA reductase (HEMA) and inhibits its activity. The leading role in suppression of TBP genes belongs to PIFs [34, 35]. Inactivation of four PIF genes (*pifq*) activated the expression of *HEMA1*, *CHLH* (H subunit of magnesium-chelatase), and *GUN4* (Genomes UNcoupled 4), which led to excessive accumulation of Pchlde in the dark and caused the photodestruction of seedlings upon illumination [35]. It was shown that PIF3 interacts with the promoter of *CHLH*, while PIF1 interacts with promoters of genes *CHLC*, *PORC*, and *CAO* (Chlide A Oxygenase), with PIF and HY5 competing for the same binding sites on promoters [49]. PIF1 and PIF3 suppress the accumulation of ROS by producing heterodimers with *trans*-factors HY5 and HYH thus preventing activation of ROS-sensitive genes [50]. PIF1 activates the expression of gene *PORC*, which is beneficial for de-etiolation. In addition, PIF1 often suppresses transcription of factors FHY3 (Far-red elongated HYpocotyl 3)/FAR1 (FAr-red impaired Response 1) activating the expression of gene *HEMB1* gene that encodes ALA-dehydratase [51].

It was shown that ethylene activates the expression of photoprotection genes *PORA* and *PORB* in the cotyledons of *A. thaliana* owing to direct binding of ethylene-dependent *trans*-factor EIN3 to promoters of these genes [8, 52]. AgNO₃ suppresses the expression of genes *PORA* and *PORB*. Mutant plants with inactivated *EIN3/EIL1* genes accumulated more Pchlde than wild type plants. Ethylene boosted the accumulation of chlorophyll after transfer to the light of *flu* mutants grown in the dark (probably because of reduction in Pchlde level). Zhong et al. [8, 52] suggest that EIN3/EIL1 should be looked upon as a new class of chlorophyll biosynthesis regulators. Thus, ethylene participates together with PIF3 in inhibition of expression of chlorophyll biosynthesis genes in the dark, and activation of *PORA* and *PORB* genes promotes a quick reduction of Pchlde, which protects the seedlings against photooxidation.

It is interesting that plants deficient in GA accumulate more Pchlde than wild type plants but they are more resistant to photostress [53]. In the dark, such plants contain more DELLA proteins that probably remove repression from the genes of chlorophyll bio-

synthesis owing to interaction with PIFs. DELLAs are positive regulators of *POR* expression, which reduces the accumulation of ROS upon de-etiolation. The examined results show that plants employ numerous ways of protection against oxidative damage upon transition from etiolation to photomorphogenesis.

LIGHT TRIGGERS THE PROCESS OF DE-ETIOLATION

De-etiolation of plants starts from the perception of light by photoreceptors. Plants respond to light of different wavelengths by means of photoreceptors which, upon activation by light, trigger certain pathways of signal transduction modifying growth and development. UV-B (280–315 nm) is perceived by UVR-8 (UV resistance locus 8) receptor. Phototropins, cryptochromes, and family Zeitlupe respond to UV-A (315–400 nm) and blue (400–495 nm) light, and phytochromes recognize red (600 nm) and far red (730 nm) light [54–57]. *A. thaliana* has five phytochromes designated as PHYA–PHYE. The N-terminal end of the PHY molecule is responsible for light perception, while the C-terminal area participates in dimerization. PHYA is stable only in the dark, whereas all other phytochromes are stable in the light. In the dark, phytochromes are predominantly located in the cytosol as inactive form Pr. Upon exposure to red light, PHYB modifies its conformation, converting from inactive Pr (660 nm) into active Pfr (730 nm) form, and moves to the nucleus. Migration of PHYA to the nucleus is initiated by a short pulse of red, far red, or blue light. Translocation of PHYA takes several minutes and depends on the participation of small proteins FHY1 (Far red elongated HYpocotyl 1) and FHL (FHY1 Like). Translocation of PHYB is much slower and does not demand participation of FHY1/FHL. In the nucleus, PHYA and PHYB are located in the speckles or nuclear photobodies [58]. PHYB forms two types of nuclear photobodies: early photobodies that are formed within 15 min after exposure to light and located together with PIF and late photobodies that are larger, more stable, and arise after 2–3-h-long exposure to red light [30]. PHYA is located together with COP1 in early nuclear bodies and quickly degrades in red light. In the presence of activated phytochromes or cryptochromes, COP1 migrates from the nucleus to cytosol, which results in spatial separation of COP1 from its substrates [59]. Light can induce degradation of SPA proteins or weaken interaction between COP1 and SPA1, and this inhibits the activity of COP1 [60]. Interaction of CRY1 and CRY2 with SPA proteins leads to a reduction in the binding of SPA1 with COP1 and causes inactivation of the COP1/SPA complex. UV-B receptor UVR8 (UV resistance locus 8) inactivates COP1 differently. Prolonged inactivation of COP1/SPA is achieved by the exclusion of COP1 protein from the nucleus.

In the nucleus, phytochromes interact with *trans*-factors and regulate the expression of more than 10% of genes in *Arabidopsis*. The most important partners of phytochromes are PIFs that, along with COP1, play a key role in the maintenance of etiolation in plants [33]. In the light, phytochromes activate phosphorylation, ubiquitination, and proteasome-mediated degradation of negative regulators of photomorphogenesis, and this initiates transition to de-etiolation [61]. Light-sensitive degradation of PIF3 residing together with active phytochromes is most active in large photobodies [62]. Protein HEMERA is necessary for the production of large photobodies and degradation of PIFs. PHYA induces the degradation of PIF1 and PIF3 [30]. PIF4 and PIF5 are targets for PHYB. PHYA affects PIF4 and PIF5 indirectly elevating the content of HFR1 that forms heterodimers incapable of binding with DNA.

We can conclude that phytochromes relieve the repression of photomorphogenesis induced by PIFs. It is interesting that, in contrast to other members of the family, PIF2 and PIF6 are positive regulators of de-etiolation. COP1 causes the degradation of PIF2 in the dark, whereas PHYB stabilizes PIF2 in the light. PIF2 interacts with PIF1, 3, 4, and 5 and deprives them of the ability to regulate the expression of target genes.

Having got to the soil surface, the seedlings surrounded by neighboring plants often find themselves exposed to low illumination rich in far red light. PHYA is a chief photoreceptor ensuring transition of development from etiolation to photomorphogenesis, since PHYA mediates very low fluence responses; other photoreceptors are incapable of such responses. Moreover, etiolated tissue contains dozens of times more PHYA than other phytochromes [63]. In such a situation, even weak far red light may activate at least several molecules of PHYA, which will trigger photomorphogenesis. And what is more, photomorphogenesis triggered by PHYA cannot be cancelled by far red light [64]. In a short time, light causes expression of the genes many of which are *trans*-factors initiating transduction of signals leading to de-etiolation.

Two *trans*-factors, FHY3 and FAR1, are positive signal transduction regulators of PHYA and participate in control over different physiological processes [51]. In *Arabidopsis*, FHY3 activates the transcription of gene *HEMB1* in response to red and far red light and genes *GUN4* and *CHLH* after exposure to far red light. Upon inactivation of *FHY3* and *FAR1*, the level of Pchlide in the dark decreased and photodestruction in the light was diminished [51]. In the dark, PIF1 interacts with the DNA-binding domain of FHY3 and suppresses its activity. FAR1 and FHY3 interact with HY5 via DNA-binding domains, which affects the transduction of PHYA signals [65]. The available results show that PHYA performs its function in conditions when there is no other effective photoreceptor: under very low illuminance and at a low ratio between red and far red

light. As plants grow and the light spectrum changes, other photoreceptors become involved in the regulation of plant development.

HY5 ENSURES INTERACTION BETWEEN LIGHT AND DIFFERENT HORMONAL SIGNAL PATHWAYS

The most important component of light signaling common to signal pathways of GA, CK, auxin, and ABA is a positive regulator of photomorphogenesis *trans*-factor HY5 [66, 67]. Content of HY5 directly correlates with the extent of photomorphogenic development. *hy5* mutants are notable for elongated hypocotyls and reduced level of chlorophyll and anthocyanins. HY5 exists in two forms: phosphorylated (inactive) and nonphosphorylated (active). Light promotes accumulation of HY5 protein in the nucleus by means of inactivation of COP1 E3 ligase. HY5 regulates the expression of almost one third of *A. thaliana* genes; out of them, 3000 are controlled by direct binding of HY5 to their promoters [68, 69]. Genes regulated by HY5 often have a G-box sequence (CACGTG). CKs, as positive regulators of de-etiolation [39], induce accumulation of protein HY5 in the nucleus, probably via influence on COP1 [66]. GA suppresses photomorphogenesis reducing the content of DELLA proteins and HY5 [70]. HY5 interacts with PIF1 and PIF3 in vivo competing for binding with G-box sequences of the genes for photosynthesis proteins and regulates their expression [50]. Moreover, HY5 induces its own expression directly binding to its promoter [71]. It plays an important role in regulation of *HEMA1* transcription under different light conditions. Many genes of tetrapyrrole biosynthesis are direct targets for HY5. Loss of HY5 function partially inhibits the expression of *CHLH* and accumulation of chlorophyll in cotyledons in the course of photomorphogenesis.

HY5 promotes photomorphogenesis modifying transduction of auxin, GA, and ABA signals. For instance, HY5 and its homolog HYH may suppress transduction of auxin signals via activation of its negative regulators [67]. Investigation of HY5 target genes in the genome of *Arabidopsis* revealed potential genes of auxin signal proteins, including *AUX/IAAs* and *ARFs* [72], genes encoding signal proteins of ethylene (ERFs, ethylene responsive factors), and GA (*DELLA*) as well as the enzymes of ABA (*NCEDs* and *CYP707A3*), ethylene (*ACS8*), GA (*GA2ox1*), and JA (*LOX3*) metabolism. It was shown that HY5 integrates pathways of transduction of light signal and ABA at the early stage of growth of *A. thaliana* seedlings via direct activation of *ABI5* gene [73]. HY5 interacts with the promoter of *ABI5* gene affecting its expression and expression of the genes it regulates. Levels of transcripts of *ABI5* and its target genes were considerably reduced in the *hy5* mutant. HY5 suppresses the transduction of the BR signal owing to activation of the genes for proteins blocking the activity of BZR1. HY5

was shown to be a point of convergence for signal pathways of cryptochrome and cytokinin [66]. The cited results make it possible to conclude that HY5 occupies a central position within transcription networks of light and hormonal signaling, which jointly participate in the regulation of photomorphogenesis.

LIGHT AND PHYTOHORMONES IN THE COURSE OF DE-ETIOLATION

Gibberellins. Light quickly inhibits the expression of the genes encoding key enzymes of GA biosynthesis (*AtGA20ox1*, 2, and 3 and *AtGA3ox1*) and stimulates expression of the genes encoding enzymes inactivating GA, which brings about a reduction in GA content [4, 16]. DELLA proteins also participate in regulating the expression of the genes of GA metabolism [14]. In the light at a low content of GA, DELLA proteins accumulate and suppress the transcription activity of PIFs; this promotes accumulation of HY5 [4]. DELLAs repress elongation of the hypocotyl stimulated by GA and BR via interaction with PIFs and/or BZR1/BES1 [5, 33, 74]. DELLAs also participate in the regulation of biosynthesis of carotenoids that are important light-protective pigments [75]. In the dark, PIFs inhibit expression of the genes of carotenoid biosynthesis; however, they degrade in the light and carotenoids are produced along with chlorophylls. A considerable part of genes responsible for oxidative stress are also controlled by DELLA proteins. Light regulates the content of PIF3 and PIF4 on the protein level, and GA regulates the transcriptional activity of PIFs via DELLA proteins. Therefore, light reduces the content of GA, which results in accumulation of DELLA proteins that inhibit GA and BR action owing to inactivation of transcription factors PIF and BZR1/BES1.

Brassinosteroids. Light does not produce a profound effect on BR content. Mutants deficient in BR have dark-green leaves as a result of a rise in the content of chlorophyll and photosynthetic proteins. Light and BR antagonistically regulate the expression of the majority of genes [76]. BZR1 inhibits the expression of positive components of light signaling, such as PHYB and phototropin 1, but it stimulates the expression of negative regulators of photomorphogenesis, including COP1 and its partners (for instance, SPA1) [25, 77]. BZR1 shares target genes with *trans*-factors regulating photomorphogenesis (PIF and HY5) [77]. Thus, BRs inhibit components of light signaling on the level of transcription.

It was also shown that UVR8 interacts with BES1 and suppresses its binding to DNA, which causes inhibition of hypocotyl elongation [26]. Interacting with BIN2, CRY1 induces phosphorylation of BZR1, which suppresses its activity. BZR1 binds to the promoter of *GATA2* and represses it [76]. Heterodimer BZR1-PIF4 induces expression of the genes participating in responses to auxin and GA and ensuring

extension growth but indirectly represses the genes encoding chloroplast proteins. BES1 and BZR1 also interact with DELLAs that block their DNA-binding ability [74]. Moreover, melatonin also participates in the regulation of hypocotyl elongation probably via its influence on expression of the genes for BR biosynthesis and signaling [78]. All the cited data agree with a functional role of BR in light signaling and photomorphogenesis.

Cytokinins. In the stage of transition from etiolation to photomorphogenesis, a special place among phytohormones belongs to CKs that are positive regulators of photomorphogenesis [79–81]. The mechanisms of reception and realization of CK signal are well known [82]. CKs stimulate the accumulation of chlorophyll and accelerate the development of etioplasts and chloroplasts [80]. CKs induce differential accumulation of proteins of all the major protein complexes of chloroplast thylakoid membranes [83]. CKs were shown to activate the assembly of polysomes [84] and stimulate the transcription of a number of plastid genes [85]. CKs participate in the regulation of expression of more than 100 genes encoding proteins located in plastids [86]. They regulate all the main stages of chlorophyll biosynthesis governing expression of the genes and activity of the enzymes they encode [87]. In cucumber cotyledons, CKs activate the synthesis of aminolevulinic acid [88], and kinetin increases the activity of Mg-chelatase and Mg-protoporphyrin-IX-methyl transferase, as well as the content of CHLH subunit of Mg-protoporphyrin-IX-chelatase, in etiolated seedlings of barley exposed to light [89]. In the dark and within the first hours of exposure of isolated lupine cotyledons to the light, BAP increased the level of mRNA and the content of POR protein enzyme [90].

German researchers [80, 87] showed that components of cytokinin signaling participate in expression of the genes encoding the main enzymes of chlorophyll biosynthesis. Investigations of *Arabidopsis* mutants have shown that AHK2 and AHK3 receptors and type B response regulators ARR1, ARR10, and ARR12 play an important role in hormonal regulation of transition of etioplasts to chloroplasts. Moreover, it was shown that response regulators ARR10 and ARR12 bind to promoters of genes *HEMA1* and *LHCB6* (Light Harvesting Complex photosystem II subunit 6) performing a transcriptional regulation of their expression [87].

In the course of de-etiolation, *trans*-factors GNC/GNL (Gata Nitrate-inducible Carbon metabolism involved/GNC-like cytokinin-responsive GATA transcription factor1) from the GATA family participate in different processes of development associated with light and phytohormones [76, 91]. In mutants *phyA phyB*, CKs induce GNL showing that red light and CKs operate independently in regulation of its expression. Double mutant *gnc gnl* showed a reduced expression of TBP genes (*HEMA1*, *GUN4*, *PORB*, and

PORC); as a result, chlorophyll content was also reduced. However, overexpression of genes *GNC/GNL* brought about a rise in these parameters [92]. It is assumed that proteins GNC and GNL (in contrast to HY5, PIF, and GLK) only indirectly regulate the expression of TBP genes. GNC/GNL proteins are not essential for CK regulation of chloroplast biogenesis since both single and double mutants (*gnc gnl*) responded to CK in the course of de-etiolation [87]. GNL and GNC are involved in GA and IAA signaling [93].

Transcription factor GLK (Golden2-Like) in *A. thaliana* is encoded by two functionally identical genes *GLK1* and *GLK2* and participates in the regulation of chloroplast biogenesis in many plants [21]. In *glk1 glk2* mutant, biogenesis of chloroplasts in the leaves is impaired [21], and overexpression results in accelerated development of chloroplasts not only in the leaves but also in nonphotosynthetic organs, such as roots. GLK is induced by CKs. Direct binding of GLK1 to promoters of TBP key genes was corroborated [94]. A putative GLK recognition *cis*-element (CCAATC) was detected [94]. It is quite possible that GLK interacts with HY5 in regulating the expression of photosynthetic genes. However, GLK activity is more closely associated with regulation of chloroplast biogenesis in nonphotosynthesizing organs.

It has been known for a long time that biogenesis of chloroplasts depends on the interaction between CKs and light. Under certain conditions, CKs can partially substitute for light. Development of photomorphogenic phenotype of *A. thaliana* in the dark in the presence of cytokinins is one of such examples [39]. Interaction between components of cytokinin signaling (ARR4) and light receptor (PHYB) was shown in [95]. We found that light and CKs while regulating expression of the *ATPC* (gamma subunit of ATPase complex) gene operate via common *cis*-element and probably via the same *trans*-factor [96]. Vandenbusschte et al. [66] concluded that HY5 is a central component of signaling shared by CKs and blue light (CRY1), with both agents causing elevation of protein HY5 stability.

Concluding the discussion on the role of CK in chloroplast biogenesis, we would like to address the question posed by Cortleven et al. [87]. What is the role of CK in biogenesis of chloroplasts? On the one hand, CKs affect almost all the stages of chloroplast development at all the levels of their action [80, 81]; on the other hand, they are not essential for chloroplast development since almost total disturbance of cytokinin signaling in a mutant for the genes of all three membrane receptors [97] and in a mutant for three response regulators *arr1 arr10 arr12*, which hardly respond to exogenous CKs, did not prevent green leaves from emerging [87]. For the time being, we cannot rule out the existence of other pathways of CK signal transduction, especially when the known mechanism of signal transduction involving membrane receptors is impaired but there is no strong evidence

that alternative pathways exist. So far, we find promising the hypothesis proposed by Cortleven et al. [87] in which CKs may act as coordinators of all the endogenous and exogenous cellular signals ensuring the accumulation of chlorophyll quantity optimal for a specific stage of plant ontogenesis. Moreover, they suggested that unknown compensatory mechanisms regulating biosynthesis of chlorophyll and biogenesis of chloroplasts are triggered when cytokinin signaling is impaired. We cherish a hope that the search for the compensatory mechanism will result in the discovery of a new pathway of CK signal transduction.

CONCLUSIONS

Although we expect that much more is to be found out about the molecular mechanisms of transition from etiolation to photomorphogenic plant development, great progress has been made in this area in spite of baffling complexity of the early stage of ontogenesis. The etiolated state depends on phytohormones and inhibitors of photomorphogenesis. A leading role in regulation of etiolated growth belongs to GA and BR, and each case depends heavily on the content of hormones in the tissue. In the dark, the content of GA is usually high and it causes degradation of DELLA proteins that on a transcriptional level inhibit essentially all the programs triggered by GA. These proteins interact with numerous *trans*-factors, mainly with those that maintain etiolation. Produced heteroduplexes of *trans*-factors and DELLAs are incapable of binding to DNA and cannot regulate gene expression. In the dark, DELLAs are scarce; therefore, GA causes active growth. In the course of etiolation, GA does not allow the accumulation of DELLAs inducing their degradation and so Daviere and Achard [18] wittily called GA a suppressor of repressor. BRs operate in the same direction. Their influence on gene expression is almost entirely realized via *trans*-factors BZR1/BES1 that, just like GA, trigger expression of the genes maintaining etiolation. At a high concentration of BRs, BIN2 are inactivated, BZR1/BES1 are dephosphorylated and become active, and the program of etiolated development is realized. This is also promoted by *trans*-factors of the PIF family whose major task is to maintain etiolation. The most important role in etiolated development belongs to a chief repressor of photomorphogenesis COP1 that inhibits any processes promoting de-etiolation, for instance, it destroys HY5, a major regulator of photomorphogenesis. It is interesting that the production of an apical hook involves many regulators. It is striking that the plant receives information via ethylene about soil density and the depth of location of the germinating seed. Depending on soil characteristics, ethylene can retard the growth of the hypocotyl but make it thicker and stronger. It is possible that CKs can also participate in suppression of hypocotyl growth.

In the light, a plant may quickly proceed to autotrophic nutrition or experience a light injury. In order to avoid damage, a complicated system of protection against accumulation in the dark of excess Pchl_{ide} was created.

As soon as the plant emerges on the soil surface, hormonal regulation is added by light. Light suppresses the biosynthesis of GA, and this means that many DELLA proteins will be produced, which block all the programs regulated by GA; DELLAs will impair BR action as well. In addition, they will considerably inactivate PIF *trans*-factors. Light receptors will cause the inactivation of the COP1/SPA1 complex and initiate destruction or inactivation of PIF proteins. Instead of destroyed regulators of transcription, there will arise *trans*-factors triggering photomorphogenesis, and, first of all, HY5 that participates in numerous signal pathways and actually governs the process of de-etiolation. In the light, an important role in chloroplast development and formation of photosynthetic apparatus belongs to CKs.

Subsequently, plant ontogenesis will proceed depending on changing environmental conditions. In each stage of development, hormones and other regulatory agents will continue performing their important roles. One should note that inactivation and/or destruction by phytochromes of PIF and COP1/SPA1 proteins does not signify that they will completely disappear. They will remain in the plant and will fulfill necessary functions, for instance, regulate growth and other processes associated with transition from day to night.

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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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