Plant blue-light receptors

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Plants have several blue-light receptors, which regulate different aspects of growth and development. Recent studies have identified three such receptors: cryptochrome 1, cryptochrome 2 and phototropin. Cryptochromes 1 and 2 are photolyase-like receptors that regulate hypocotyl growth and flowering time; phototropin mediates phototropism in response to blue light. In addition, phytochrome A has also been found to mediate various blue-light responses. Although the signal-transduction mechanisms of blue-light receptors remain largely unclear, phototropin is probably a protein kinase that regulates cytoplasmic calcium concentrations, whereas the cryptochromes might regulate anion-channel activity and changes in gene expression.

Blue light affects many aspects of plant growth and development. Plant blue-light responses include inhibition of hypocotyl elongation, stimulation of cotyledon expansion, regulation of flowering time, phototropic curvature, stomatal opening, entrainment of the circadian clock and regulation of gene expression. During the past decade, molecular genetic studies using *Arabidopsis* as a model system have identified three blue-light receptors: cryptochrome 1, cryptochrome 2 and phototropin, which regulate primarily hypocotyl inhibition, flowering time and phototropism, respectively. This article focuses on some recent advances in our understanding of the function of these molecules in plant development. For comprehensive descriptions of these photoreceptors also see Refs 1,2.

Discovery of the cryptochromes

Plant blue-light receptors have long been recognized from their action spectra to have roles in mediating various developmental responses and they have been hypothesized to contain blue–UV-A-light-absorbing chromophores such as flavin, pterin and carotenoids[3]. However, unlike phytochrome, a blue-light-receptor protein has never been successfully purified from a plant. This was certainly not because of a lack of effort; rather, a lack of specific biochemical assays and the relatively low abundance of these proteins made the biochemical approach ineffective. A laboratory nickname, cryptochrome, was coined for the blue–UV-A light receptors, which reminds us of the pervasive blue-light responses found in cryptogamic plants (i.e. non-flowering plants such as ferns, mosses and algae) and the once-cryptic nature of this type of pigment[4].

Unlike phytochromes, plants have various blue-light receptors that appear to be derived from more than one evolutionary lineage and they could not be covered by the same nomenclature. One class of these pigments, the photolyase-like blue-light receptors, was named cryptochrome, simply because genes encoding this group of blue-light receptors were the first to be isolated[1]. Thus, a new name, phototropin, was invented for the blue-light receptor that mediates the phototropic response[2]. Nomenclature for the *Arabidopsis* cryptochromes is adapted from that for the phytochromes[5]; for example, the wild-type gene, mutant gene, holoprotein and apoprotein of cryptochromes 1 and 2 are designated *CRY1* and *CRY2*, *cry1* and *cry2*, cry1 and cry2, and CRY1 and CRY2, respectively[6].

Cryptochrome was first identified in *Arabidopsis thaliana*. In 1980, several *Arabidopsis* photomorphogenic mutants were isolated, one of which, *hy4*, had impaired blue-light-dependent inhibition of hypocotyl elongation, resulting in a long hypocotyl when grown in blue light[7]. The *HY4* gene (later renamed *CRY1*) was identified via the isolation and characterization of a T-DNA-tagged *hy4* allele[8]. DNA sequence analysis of the *HY4* locus revealed a 681-residue open-reading-frame, for which the N-terminal region (~500 amino acids) exhibited over 30% amino acid sequence identity to the microbial DNA-repairing enzyme DNA photolyase[9]. DNA photolyases are flavoenzymes that catalyze a blue–UV-A-light-dependent DNA-repair reaction through an electron-transfer mechanism[9].

Remarkably, photolyase had been previously suggested, before the isolation of the *CRY1* gene, to be a possible evolutionary precursor of the plant blue-light receptor[3,10]. The genetic evidence and the sequence similarity between CRY1 and photolyase suggested that cry1 was likely to be the blue-light receptor that mediates the light inhibition of hypocotyl elongation in *Arabidopsis*[8]. Analysis of cry1 protein purified from insect cells or *E. coli* expressing the *Arabidopsis CRY1* cDNA showed that cry1 associated non-covalently with a stoichiometric amount of flavin adenine dinucleotide (FAD), which primarily absorbs blue and UV-A light[11,12]. In addition to FAD, a pterin (5,10-methenyltetrahydrofolate) was found to bind to the recombinant CRY1 N-terminal photolyase-homology domain expressed in *E. coli*, suggesting that, like photolyase, cry1 might contain pterin as a second chromophore[12]. The cry1 protein showed no photolyase activity *in vitro* or in *E. coli* cells[11,12], which is consistent with it being a photosensory receptor rather than a DNA-repairing enzyme.

Cryptochromes and blue-light regulation of hypocotyl growth

The major photoreceptor mediating blue-light inhibition of hypocotyl elongation is cry1

In nature, seeds are often buried under soil and germinate in the dark. Young seedlings of dicot plants germinated in the dark develop rapidly elongating hypocotyls to push unopened cotyledons above the soil surface. Upon exposure to light, hypocotyl elongation is inhibited and the cotyledons start to expand and to become photosynthetically competent. These developmental changes are collectively referred to as de-etiolation[13,14]. The photoreceptor-dependent hypocotyl inhibition response is the best-studied aspect of de-etiolation because it is easy to quantify. Isolation and characterization of *Arabidopsis* mutations impaired in this response have shown that phytochrome A (phyA) and phytochrome B (phyB) function in far-red and red light, respectively[15]. When tested with the hypocotyl-inhibition response, *Arabidopsis hy4–cry1* mutants are insensitive to blue light, especially high-intensity blue light[7,8]; by contrast, transgenic *Arabidopsis* plants overexpressing *CRY1* have enhanced blue-light sensitivity[16].

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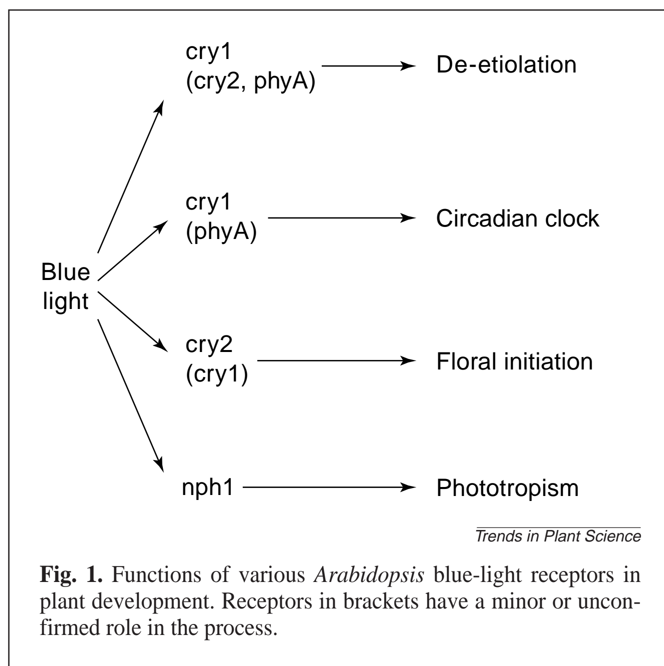

Fig. 1. Functions of various *Arabidopsis* blue-light receptors in plant development. Receptors in brackets have a minor or unconfirmed role in the process.

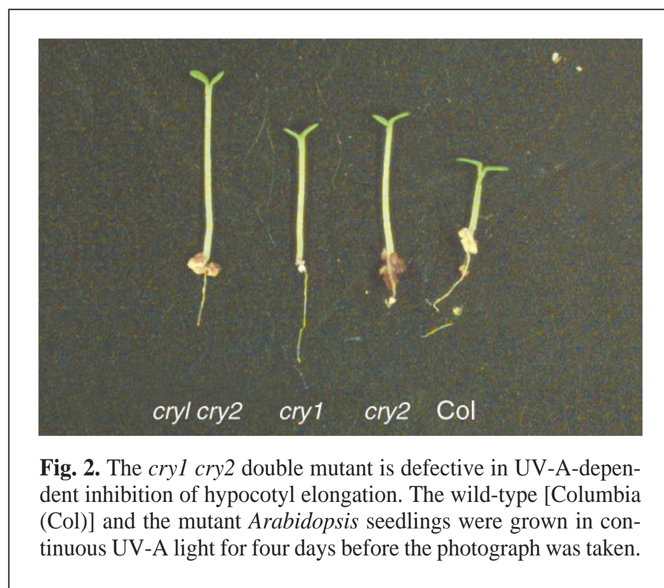

Fig. 2. The *cry1 cry2* double mutant is defective in UV-A-dependent inhibition of hypocotyl elongation. The wild-type [Columbia (Col)] and the mutant *Arabidopsis* seedlings were grown in continuous UV-A light for four days before the photograph was taken.

These results established cry1 as the major blue-light receptor regulating de-etiolation (Fig. 1). It also appears to mediate hypocotyl inhibition in other plants. For example, overexpressing *Arabidopsis CRY1* in tobacco resulted in exaggerated hypocotyl inhibition in blue–UV-A light, suggesting that the signal-transduction mechanism for cry1 is conserved in different plants[17]. Recently, two cryptochrome genes have been isolated from the tomato[18], and tomato CRY1 has 78% amino acid sequence identity to *Arabidopsis* CRY1 (Ref. 18). Transgenic tomato plants expressing antisense tomato *CRY1* had long hypocotyls when grown in blue light[18]. This observation indicates that, like *Arabidopsis* cry1, the tomato cry1 also mediates blue-light inhibition of hypocotyl elongation.

Expression of cry2 is negatively regulated by blue light

The second *Arabidopsis* cryptochrome gene, *CRY2*, was isolated by screening a cDNA library using *CRY1* cDNA as the hybridization probe[6,19]. In contrast with *CRY1*, which is expressed more-or-less constitutively[8,16], *CRY2* expression was downregulated in blue light[6,20]: the CRY2 protein concentration decreased rapidly in etiolated seedlings exposed to blue light. However, no change in the *CRY2* mRNA could be detected. Two lines of evidence suggested that blue light triggered the degradation of the cry2 protein. First, the *CRY2* coding sequence contained all the information for blue-light-dependent regulation of cry2 protein level: the cry2 protein derived from a transgene containing no native untranslated sequence of the *CRY2* gene was regulated by blue light in a similar way to the endogenous cry2 protein[6]. Second, blue-light-dependent downregulation of cry2 was not affected by the translation inhibitor cycloheximide[20].

Furthermore, a GUS–CRY2 fusion protein expressed in transgenic plants also showed a blue-light-induced degradation[21]. Interestingly, a fusion protein between GUS and the C-terminal domain (residues 480–612) of CRY2 showed no light-induced degradation[21], whereas a fusion between a smaller fragment of the C terminal of CRY2 (residues 505–611) and the N-terminal domain of CRY1 was found to be degraded in blue light[20]. The structure of CRY2 responsible for its blue-light-induced degradation needs further investigation. The blue-light-induced cry2 degradation cannot be mediated by cry1 because it is not affected by a *cry1* mutation[21]. It is conceivable that the absorption of blue light by cry2 changes its conformation and triggers its own degradation.

The cry2 protein also takes part in the de-etiolation process

Transgenic *Arabidopsis* overexpressing *CRY2* showed exaggerated blue-light-inhibition of hypocotyl elongation, especially in low-fluence rate blue light[6]. Based on this observation, it was hypothesized that cry2 might also be involved in the de-etiolation response. A genetic screen was designed to screen for mutants impaired in blue-light-inhibition of hypocotyl elongation under low-intensity blue light, resulting in the isolation of two *cry2* deletion-mutant alleles[6,22]. In comparison with wild-type plants, these *cry2* mutant seedlings had long hypocotyls and small or unopened cotyledons when grown in low fluence rate blue light. These results confirmed that cry2 also plays a role in the blue-light regulation of hypocotyl growth (Fig. 1). In high-fluence-rate blue light, the long-hypocotyl phenotype of the *cry2* mutant was less apparent. The dependence of the *cry2* mutant phenotype on light intensity was interpreted as a consequence of blue-light-dependent changes of the cellular cry2 protein concentration[6].

Cryptochromes respond to both blue light and UV-A light

One interesting aspect of blue-light receptors is that they function not only in blue light but also, to varying degrees, in long-wavelength UV light (UV-A, ~320–390 nm). Therefore, cryptochrome was historically defined as a photoreceptor with a two-peak action spectrum, one in the blue-light region and the other in the UV-A region[23]. *Arabidopsis* cry1 is clearly the primary photoreceptor mediating both blue-light- and UV-A-dependent expression of the chalcone-synthase gene, because the *cry1* mutant was impaired in this response[8,26] in both blue and UV-A light[24,25].

By contrast, analysis of various *cry1* mutant alleles showed that the *cry1* mutation had a relatively minor or no effect on the UV-A-dependent hypocotyl-inhibition response[8,26] (Fig. 2). Similarly, a *cry2* mutant did not show a dramatic long-hypocotyl phenotype when grown in UV-A light (Fig. 2). However, transgenic tobacco and *Arabidopsis* plants overexpressing the *Arabidopsis CRY1* gene had a hypocotyl-inhibition response that was significantly more sensitive to both blue and UV-A light, suggesting that cry1 could respond to UV-A light[16,17].

Why did overexpression, but not mutation, of the *CRY1* gene significantly affect the hypocotyl-elongation response in UV-A light? One possible interpretation is that cry1 and cry2 function

redundantly in mediating hypocotyl inhibition in UV-A light. Indeed, the *cry1 cry2* double mutant has a hypocotyl that is much longer than that of the wild type when grown in UV-A light (Fig. 2). It remains to be seen whether there are additional photoreceptors mediating hypocotyl inhibition in UV-A light.

Phytochrome and blue-light responses

Phytochrome is known to absorb blue light and it has long been suspected that it might function as a blue-light receptor[27]. An *Arabidopsis phyA* null mutant was found to develop a long hypocotyl in relatively low fluence rates of blue light, suggesting a possible role for phyA in mediating blue-light inhibition of hypocotyl elongation[28]. It was later proposed that phytochromes might participate in the transduction of the cry1 signal[29]. More-detailed genetic and physiological analysis of *phyA*, *phyB*, *cry1* and multiple-photoreceptor mutants has recently shown that the cry1-dependent hypocotyl inhibition could be independent of phytochrome and that at least phyA could indeed act as a blue-light receptor[30–32]. The function of phyA in blue light is not limited to hypocotyl inhibition. For example, phyA has been found to mediate blue-light-dependent cotyledon opening and expansion, as well as blue-light regulation of the circadian clock[30,33].

Cellular mechanisms underlying blue-light inhibition of hypocotyl elongation

Blue-light-induced hypocotyl inhibition has two kinetic phases: a rapid phase and a slow phase. The rapid response occurs transiently within a few minutes or even seconds of a blue-light pulse, and the slow response occurs hours later and lasts much longer[2,34]. Interestingly, it has recently been shown that these two kinetic responses are mediated by distinct photoreceptors in *Arabidopsis*[34].

The rapid growth inhibition induced by blue light is preceded by a transient plasma-membrane depolarization in hypocotyl cells of various plant species, including *Arabidopsis*[34,35]. This membrane depolarization might result from the opening of ion channels, because blue light has also been shown to trigger a rapid (within 1 min) activation of anion-channel opening[36]. However, the relationships between blue-light-induced membrane depolarization, anion-channel activation and rapid growth inhibition remain unclear at present. Although the *cry1* mutant was significantly impaired in blue-light-induced membrane depolarization, it showed no defect in the rapid growth inhibition and it is not clear whether the rapid anion-channel activation is affected[34]. It will be interesting to see whether the rapid growth-inhibition response is mediated by the redundant actions of cry1 and cry2 or by other photoreceptors such as phyA. It is also puzzling that, although the anion-channel inhibitor 5-nitro-2-(3-phenylpropylamino)-benzoic acid (NPPB) could suppress anion-channel activity as well as the blue-light-induced membrane depolarization[36], it appeared to have no effect on the rapid growth inhibition in response to blue light[34].

The slow phase of blue-light-dependent growth inhibition is primarily mediated by cry1 (Ref. 34). Moreover, it has been shown that NPPB had a similar effect to *cry1* mutation in suppressing the slow response[34]. Therefore, for at least the slow response, cry1 might somehow activate anion channels, resulting in elevated water potential in the cell and slowing down the cell's expansion.

Are changes in gene expression involved in blue-light inhibition of hypocotyl elongation?

In addition to the role of anion channels, blue-light-dependent changes in gene expression might also be involved in the slow phase of hypocotyl inhibition. Although the regulation of gene expression has not been directly shown to affect this response, several lines of evidence seem to suggest that photoreceptor-regulated changes of gene expression might play a role. First, the slow response of hypocotyl inhibition does not take place until at least an hour after blue-light treatment[2,34], which allows enough time for changes in gene expression to take effect. Second, many of the photoreceptors, including *Arabidopsis* phyA, phyB, cry1 and cry2, are nuclear proteins[1,21,37–39], which suggests that these photoreceptors might regulate gene expression via a short signaling path. Furthermore, genes encoding transcription regulators, including *COP1*, *DET1*, *HY5*, *CCA1*, *LHY*, *SPA1* and *PIF3*, have been shown to play roles in the light inhibition of hypocotyl elongation[40–46]. For example, mutation of the *HY5* gene causes an elongated hypocotyl in red–far-red and blue light[7]. Transgenic plants overexpressing *COP1* or *CCA1* also had long hypocotyls when grown in light[42,46]. It is not clear how many other nuclear proteins participate in blue-light-inhibition of hypocotyl elongation, nor is it clear how a transcription regulator might be involved in light-dependent growth inhibition.

Cryptochromes and the circadian clock

Animal cryptochromes

Since the discovery of plant cryptochromes, this type of photolyase-like pigment has also been found in animals. For example, human and mouse each have two cryptochromes (hCRY1 and hCRY2, and mCry1 and mCry2)[47,48], and *Drosophila* has one cryptochrome (dCRY)[49]. Animal cryptochromes are also flavoproteins with no photolyase activity[48]. In contrast with the plant cryptochromes, which are more closely related to the microbial type-I photolyase, the animal cryptochromes resemble the 6–4 photolyase, and there seems to be little sequence relatedness between the C-terminal domains of cryptochromes in different organisms[1,47–49]. It has been hypothesized that plant and animal cryptochromes arose independently during evolution[1].

Interestingly, the expression of the mammal and *Drosophila* cryptochrome genes shows a circadian rhythm[49,50], an activity that has not been reported for the plant cryptochromes. The *Drosophila* cryptochrome mutant *cry*^{b}, when coupled to a mutation impaired in the rhodopsin signal-transduction pathway, responded poorly to phase-shifting light pulses[51]. Transgenic flies overexpressing dCRY had clock-regulated genes whose expression was more sensitive to light[49]. Moreover, dCRY showed light-dependent interaction with a clock protein (TIM) and suppressed the PER–TIM functions, which are key clock components in *Drosophila*[52]. These observations indicate that dCRY is one of the photoreceptors that regulates the circadian clock in *Drosophila*[49,51,52].

It is not clear whether mammalian cryptochromes are also photoreceptors. Knockout mice lacking mCry1 or mCry2 showed a shorter or longer period length, respectively[53,54]. It was also reported that light-induced *mPer1* expression was impaired in *mCry1 mCry2* double-mutant mice, although the photic induction of *mPer2* expression was not affected[55]. These results are consistent with the suggestion that mammalian cryptochromes might also be photoreceptors regulating the circadian clock[53]. The knockout mice lacking both mCry1 and mCry2 completely lost free-running-behavior rhythmicity, suggesting that the mammalian cryptochromes are themselves an essential part of the clock apparatus[54]. However, this complete loss of free-running rhythmicity made it difficult, if not impossible, to study further the possible role of mouse cryptochromes in the light regulation of the circadian clock.

It has been shown that the expression of hCRY1 and hCRY2 in a cell-culture system affected gene expression in a light-independent manner, and the human cryptochromes showed

light-independent interaction with other clock proteins including CLOCK, BMAL1, PER1, PER2 and TIM (Ref. 56). Moreover, it was reported recently that the light-induced expression of neither *mPer1* nor *mPer2* was affected by the *mCry1 mCry2* double mutation[57], suggesting that another photoreceptor is involved in the light regulation of *mPer* genes.

Arabidopsis cry1 and phyA are involved in regulating the circadian clock

The circadian clock regulates the expression of many plant genes. The clock-regulated expression of *Arabidopsis* genes encoding chlorophyll-*a*/*b*-binding protein (*CAB2*) and catalases (*CAT2* and *CAT3*) have been most extensively studied[58,59]. Analysis of the circadian rhythms of expression of a CAB2–luc (luciferase) fusion gene showed that blue light and red light could accelerate the pace of the circadian clock[60]. For example, *CAB2*-promoter activity had a 24–25 h period length in continuous blue light or red light, compared with a period length of ~30–36 h in continuous dark, suggesting that photoreceptors might shorten the period length[60]. It might be expected that the mutation of a photoreceptor would cause the circadian clock to run more slowly in the relevant wavelength of light. Indeed, the *CAB2*-promoter activity was found to oscillate with a longer period length in *phyA* and *cry1* mutant plants under appropriate fluence rates of blue light[33]. Detailed analysis of *CAB2*-promoter activity in photoreceptor mutants in response to blue light indicated that cry1 regulated the circadian clock in a wide range of blue-light intensities. However, phyA is also involved in blue-light regulation of the circadian clock but its function seems to be limited to low-light intensities[33]. The functions of phyA and cry1 have also been implicated in the clock-regulated expression of the *CAT3* gene[61].

Flowering time is regulated by cry2

In addition to its function in de-etiolation, cry2 also plays a role in the regulation of flowering time. The *Arabidopsis cry2* mutant (in a Columbia background) is allelic to the previously identified photoperiod-hyposensitive late-flowering mutant *fha* (in a Landsberg *erecta* background)[22,62]. A mutation in the *CRY2* gene has been found in all three *fha* alleles[22]. Plants with a mutated *cry2* gene flowered late in long day (LD) but not in short day (SD), and transgenic plants overexpressing *CRY2* flowered slightly early in SD but not in LD. Therefore, both the mutation and the overexpression of the *CRY2* gene caused reduced sensitivity to photoperiods. Surprisingly, in contrast with *cry1*, the mutation of *CRY2* does not seem to affect the circadian-clock-regulated expression of the *CAB2* promoter in blue light with fluence rates higher than 1 μmole m^{-2} s^{-1} (Ref. 33). It is not known whether cry2 regulates the circadian clock in response to blue light with a fluence rate of less than 1 μmole m^{-2} s^{-1}. However, an activity under such low-intensity light might not account for the dramatic flowering-time phenotype found in the *cry2* mutants grown in relatively high light conditions. The cry2 and cry1 proteins could regulate certain aspects of the clock function in a redundant manner; an analysis of the *cry1 cry2* double mutant should clarify this possibility. Alternatively, the function of cry2 in photoperiodic flowering might not be directly involved in the regulation of the circadian clock; rather, cry2 might regulate the expression of flowering-time genes and the transduction of the cry2 signal might be regulated, or gated, by the circadian clock[33,63].

Phototropin and phototropism

Phototropin nph1

Phototropism is probably the first blue-light response discovered in plants[2]. In many plants, including *Arabidopsis*, phototropism is largely controlled by blue-light receptors, although phytochromes also contribute, to various degrees, to the overall response[2]. *Arabidopsis* phototropism-deficient mutants (*nph1* to *nph4*) have been isolated and characterized[2,64], and the *NPH1* and *NPH3* genes have recently been identified[65,66]. *NPH1* encodes a 120 kDa protein previously shown to be associated with the plasma membrane and to undergo blue-light-induced phosphorylation[67]. There are at least three recognizable domains in the apoprotein NPH1: a serine–threonine-kinase domain at the C terminal and two LOV (for light, oxygen and voltage) domains at the N terminal. Consistent with the proposition that the holoprotein nph1 is the photoreceptor that was later referred to as phototropin, recombinant NPH1 was shown to be a flavoprotein and to undergo blue-light-dependent autophosphorylation[68]. *Arabidopsis* NPH1 expressed and purified from insect cells is a soluble protein that binds flavin mononucleotide[68] (FMN); the LOV domains have recently been shown to be the flavin-binding domains of nph1 (Ref. 69).

Interestingly, a fern gene (*Phy3*) has been found to encode a protein with sequence similarity to the chromophore-binding domain of phytochrome at its N terminal and to the entire NPH1 at its C terminal[70]. The PHY3 protein expressed and purified from yeast, after reconstitution with phycocyanobilin, showed the typical phytochrome photochromic reaction as assayed by the red–far-red differential spectrum[70]. The LOV domain of PHY3 expressed in *E. coli* bound to FMN (Ref. 69). Therefore, PHY3 might be a dual photoreceptor that can mediate red–far-red-light and blue-light responses.

Signal transduction from phototropin

Plant nph1 is tightly associated with the plasma membrane[67]. However, the photochemically active NPH1 protein expressed and purified from heterologous systems was soluble[68], suggesting that NPH1 might undergo lipid modification or need a protein partner(s) to bind to the plasma membrane. One of the partners of nph1 is NPH3. Like *nph1*, the *Arabidopsis nph3* mutant also has defective blue-light-induced phototropism[64,71]. Genetic studies indicate that NPH3 is probably a downstream signaling partner of NPH1 (Ref. 64). The *NPH3* gene encodes a protein with protein–protein-interaction motifs[66]. Plant NPH3 also associates with the plasma membrane and interacts with nph1. Because NPH3 contains no obvious membrane-spanning region, an additional anchor protein or a lipid modification of either nph1 or NPH3 might still be needed for the membrane attachment of the nph1–NPH3 complex.

The nph1 protein is a kinase that catalyzes a blue-light-dependent autophosphorylation. Although it is not clear what other substrates might be phosphorylated by nph1, the plasma-membrane Ca^{2+} channel appears to be a good candidate. Using transgenic *Arabidopsis* and tobacco plants expressing aequorin as a calcium indicator, it was found that blue (but not red) light induced a transient increase of the cytoplasmic calcium concentration, which occurred within a few seconds of blue-light treatment and lasted for 1–2 min (Ref. 72). The blue-light-induced increase in the cytoplasmic calcium concentration was drastically reduced in the *nph1* mutant, whereas this response was not affected in either the *cry1* or *cry2* monogenic mutants. These observations indicate that the regulation of cytoplasmic calcium concentration might be part of the signaling mechanism of nph1 (Ref. 72).

Other photoreceptors involved in the phototropism

It is known that phytochrome is involved in the responsiveness of plants to blue light in phototropism[2]. It has also been reported that a *cry1 cry2* double mutant failed to show the first positive phototropism and it was suggested that cry1 and cry2 acted redundantly to mediate phototropism[73]. However, detailed analysis of

phototropism in *cry1 cry2* and *phyA phyB* double mutants suggested that these photoreceptors might not play direct roles in mediating phototropism, although both types of receptors could modulate the phototropic response under various conditions[2,74].

However, nph1 might not be the only photoreceptor that directly mediates phototropism in response to blue light. It has recently been found that, although the *nph1* mutant shows no phototropism in response to a 12 h exposure to blue light with a fluence rate of less than 1 μmole m^{-2} s^{-1}, it has almost normal phototropism when plants are exposed to 12 h of blue light with a fluence rate of 100 μmole m^{-2} s^{-1} (Ref. 75). This observation suggests that there are probably additional photoreceptor(s) that mediate phototropism in response to high-intensity blue light.

Future prospects

Plants, including *Arabidopsis*, are likely to have blue-light receptors other than cry1, cry2 and nph1. For example, blue-light-induced stomatal opening in *Arabidopsis* appears to be regulated by an as yet unidentified photoreceptor[74] and, as described above, *Arabidopsis* might have another photoreceptor controlling phototropism. There is little doubt that the *Arabidopsis*-genetics approach would play a vital role once again in the identification of these blue-light receptors.

We still know little about the signal-transduction mechanism of blue-light receptors, especially for the cryptochromes. If DNA photolyase, the likely evolutionary ancestor of the cryptochromes, is any guide, we might expect that the signal transduction of cryptochromes involves an electron-transfer reaction and/or an action on DNA. Although we have no direct biochemical evidence that a cryptochrome catalyzes an electron-transfer reaction, a recent pharmacological study suggests that a redox reaction might be involved in blue-light-induced gene expression[76]. There is also no evidence supporting the direct regulation of transcription by plant cryptochromes. However, plant cryptochromes are nuclear proteins that are known to regulate gene expression. It remains an intriguing possibility that, like their animal counterparts, plant cryptochromes might be part of the transcription-regulation complex and that they might mediate light regulation of development by direct interactions with DNA or DNA-binding proteins.

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