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▪ **Invited Review** ▪

Light Regulation of Gibberellins Metabolism in Seedling Development

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Abstract

Light affects many aspects of plant development, including seed germination, stem elongation, and floral initiation. How photoreceptors control photomorphogenic processes is not yet fully understood. Because phytohormones are chemical regulators of plant development, it may not be surprising that light affects, directly or indirectly, cellular levels and signaling processes of various phytohormones, such as auxin, gibberellins (GA), cytokinin, ethylene, abscisic acid (ABA), and brassinosteroids (BR). Among those phytohormones, light regulation of GA metabolism has probably attracted more attention among photobiologists and it is arguably the most extensively studied plant hormone at present with respect to its role in photomorphogenesis. It has become increasingly clear that phytochromes and cryptochromes are the major photoreceptors mediating light regulation of GA homeostasis. This short article attempts to examine some recent developments in our understanding of how light and photoreceptors regulate GA biosynthesis and catabolism during seedling development. It is not our intention to carry out a comprehensive review of the field, and readers are referred to recent review articles for a more complete view of this area of study (Kamiya and Garcia-Martinez 1999; Hedden and Phillips 2000; Garcia-Martinez and Gil 2001; Olszewski et al. 2002; Halliday and Fankhauser 2003; Sun and Gubler 2004).

Key words: phytochrome; cyrptochrone; gibberellins.

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Gibberellins (GA) Metabolism and Signaling

Gibberellins (GA) are tetracyclic diterpenoid hormones that regulate many aspects of plant development (Hedden and Phillips 2000; Olszewski et al. 2002; Sun and Gubler 2004). Unlike other plant hormones, GA are defined by their chemical structures that all contain the same ent-gibberellane ring, and only a few of the presently known 126 different GA, such as GA_1, GA_3, GA_4, and GA_7, are physiologically active. Different plant species appear to preferentially use different forms of bioactive GA. For example, GA_1 is believed to be the bioactive GA in lettuce, peas, and rice, whereas GA_4 is the bioactive GA in *Arabidopsis* and cucumber. GA metabolism takes place in three different cellular compartments, plastids, endoplasmic reticulum (ER), and cytosol (Figure 1) (Hedden and Phillips 2000; Olszewski et al. 2002; Sun and Gubler 2004). Multiple enzymes are involved in GA metabolism and catabolism, including *ent*-copalyl diphosphate synthase (CPS), ent-kaurene synthase (KS), P450 monooxygenases (e.g. KO), and dioxygenases. Two dioxygenases, GA 20-oxidase (GA20ox) and GA 3b-hydroxygenase (GA3ox), catalyze the last few steps in the synthesis of bioactive GA. Another dioxygenase, GA 2-oxidase (GA2ox), catalyzes GA catabolism of bioactive GA or their precursors (Figure 1) (Thomas et al. 1999; Hedden and

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Phillips 2000; Olszewski et al. 2002).

Two general experimental strategies often used at present to study light regulation of GA metabolism are the analysis of levels of various GAs, especially the bioactive GAs, and comparisons of the expression level of genes encoding GA metabolic/catabolic enzymes. In this regard, two features of GA metabolism and associated genes are particularly relevant in the interpretation of experimental results. First, the expression and activity of many GA metabolic/catabolic enzymes are controlled by cellular GA levels via complex feedback and feed-forward regulatory networks. For example, an increased level of GA usually suppresses expression of *GA20ox* and *GA3ox*, which controls the last a few steps in GA biosynthesis. Conversely, an elevated GA level stimulates the expression of *GA2ox*, which acts to inactivate physiologically active GAs or to remove their immediate precursors. Secondly, one type of a GA metabolic/catabolic enzyme is often encoded by multiple members of a gene family, and the expression of different member of the respective gene family varies with respect to the cell type, developmental timing, and response to light. For example, *Arabidopsis GA2ox* gene family has at least six members, and the expression of each member in response to blue light is not identical. These complexes presumably reflect a delicate homeostatic balance of GA metabolism in plants, but they would make it difficult to accurately interpret results of individual experiments with respect to physiological implications.

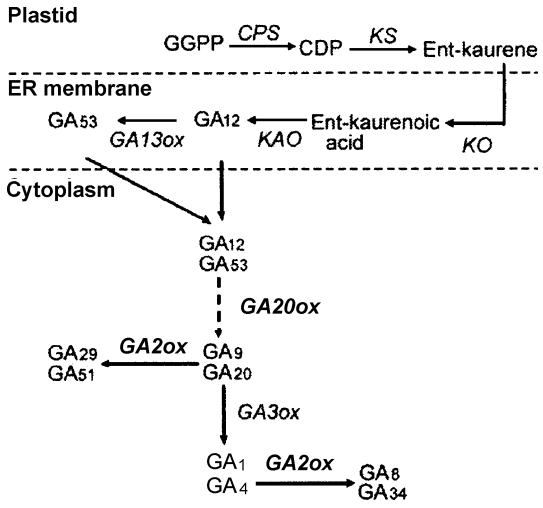

Figure 1. A brief view of the GA metabolic pathway in higher plants (modified from Hedden and Phillips, 2000).

GGDP, geranylgeranyl diphosphate; CDP, *ent*-copalyl diphosphate; CPS, *ent*-copalyl diphosphate synthase; KS, *ent*-kaurene synthase; KO, *ent*-kaurene oxidase; KAO, *ent*-kaurene acid oxidase; GA13ox, GA 13-hydroxylase; GA20ox, GA 20-oxidase; GA3ox, GA 3b-hydroxylase; GA2ox, GA 2-oxidase.

According to our present understanding, the GA signal transduction starts with the GA receptor(s), GID1 and related proteins. GID1 is a nuclear protein with structural similarities to the hormone-sensitive lipases (Ueguchi-Tanaka et al. 2005; Nakajima et al. 2006). GID1 interacts with the DELLA proteins, which are a subgroup of the GRAS family transcription regulators that contain a 27-residue conserved motif known as the DELLA domain. The DELLA proteins act as negative regulators of the GA signaling (Peng et al. 1997; Silverstone et al. 1997). It is believed that, when the cellular GA level is low, the DELLA proteins suppress the activity of positive regulators such as GAMYB or other transcription factors and transcription of GA-dependent gene expression. Association of GA to the GA receptors leads to ubiquitination and degradation of the DELLA proteins, resulting in de-repression of the expression of GA-induced genes (Olszewski et al. 2002; Peng and Harberd 2002; Sun and Gubler 2004; Chow and McCourt 2006). Therefore, similar to other plant hormones such as ethylene and auxin, GA appears to act by de-repression of its signal transduction (Bishopp et al. 2006). It seems intuitive that light may affect not only GA metabolism but also GA signaling processes, and some physiological and genetic analyses are consistent with this notion (Reed et al. 1996; Peng and Harberd 1997; Xu et al. 1997; O'Neill et al. 2000; Cao et al. 2005). However, in contrast to abundant reports on the light regulation of mRNA expression of GA metabolic/catabolic genes, how light affects the GA signaling processes remains largely unclear.

Genes associated with both GA metabolism and GA signaling are important to agriculture productivity. The remarkable increases in the yields of cereal crops such as wheat, rice, and corn during the 'Green Revolution' in the 1960s–1970s were made possible largely by breeding of dwarf traits into the new cultivars (Hedden 2003). Those new varieties have shorter stalks that allow plants to uphold increased weight of grains, resulting from improved agriculture practices such as applications of fertilizers and pesticides. Those semi-dwarf cultivars are also more resistant to winds and rains. It turns out that the semi-dominant mutations of wheat Rht-B1/Rht-D1 and maize dwarf-8 (d8), which have been widely bred into commercial cultivars, are functional orthologs of the *Arabidopsis GAI* gene that encodes the first DELLA protein identified (Peng et al. 1997; Peng et al. 1999). In contrast, the rice *sd1* recessive mutations, which are responsible for the semi-dwarf trait of many different rice cultivars, were found to affect the same *GA20ox* gene, *OsGA20ox2* (Sasaki et al. 2002; Spielmeyer et al. 2002). It is intriguing that, although rice genome encodes four *GA20ox* genes, mutant *OsGA20ox2* has been isolated and bred into various semi-dwarf rice cultivars grown in different regions of the world. This coincidence may be attributed to the fact that, compared to other *OsGA20ox* genes, *OsGA20ox2* is more abundantly expressed in rice stems (Hedden 2003; Sakamoto et al. 2004).

Phytochrome Regulation of GA Metabolism

The red/far-red light receptor phytochrome was first discovered over a half century ago in a study of lettuce seed germination (Borthwick et al. 1952). Soon after, it was found that phytochrome might be required for seed germination because it stimulates GA accumulation (Ikuma and Thiman 1960). However, those same researchers may have also been puzzled because they failed to detect in the germinating lettuce seeds a significant increase of GA_3, which was thought to be the active GA required for lettuce seed germination (Ikuma and Thiman 1960). This mystery was solved many years later when it was found that GA_1, instead of GA_3, was the bioactive GA in lettuce, and that phytochromes indeed mediate red light-induced accumulation of GA_1 in the germinating lettuce seeds (Toyomasu et al. 1993). Not only was an identification of the correct form of active GA important, but it is also critical to find out which specific member of a gene family is affected by light in order to understand how light regulates GA metabolism. In this regard, it may be interesting to revisit a study on the role of phytochrome and GA metabolic genes in *Arabidopsis* seed germination. It was known for a long time that seeds of *Arabidopsis* mutants such as *ga1*, *ga2*, and *ga3*, which are defective in genes encoding early GA biosynthesis enzymes CPS, KS, and KO, respectively (Figure 1), could not germinate without exogenous application of GAs, confirming that GA is essential for seed germination. Another *Arabidopsis* GA metabolic mutant, *ga4*, is impaired in the *GA3ox1* gene encoding a GA3ox catalyzing the last step of GA_4 biosynthesis (Chiang et al. 1995; Williams et al. 1998) (Figure 1). In contrast to the other GA metabolic mutants, *ga4* can germinate without exogenous application of GAs. This puzzle was resolved only when another *GA3ox* gene in the *Arabidopsis* genome, *GA3ox2* (previously referred to as *GA4H*) was isolated (Yamaguchi et al. 1998). *GA3ox1* and *GA3ox2* apparently act redundantly so that the monogenic mutation of neither one alone is insufficient to prevent seed germination. It was found that *GA3ox2* was predominantly expressed in germinating seeds and its expression was stimulated in response to red light. The red light-induced expression of *GA3ox2*, but not *GA3ox1*, is impaired in the *phyB* mutant, demonstrating that phyB stimulates seed germination at least partially by activating *GA3ox2* expression (Yamaguchi et al. 1998).

It seems clear now that phytochromes regulate many aspects of plant development via their direct physical interactions with phytochrome interacting factors (PIF) such as PIF3, PIF4, and PIF1/PIL5, which are bHLH (basic-helix-loop-helix)-type transcription factors (Ni et al. 1998, 1999; Martinez-Garcia et al. 2000; Huq et al. 2004)(Quail, article in this issue). Phytochromes may affect the expression of GA metabolic genes via a direct interaction and regulation of those PIF transcription factors. For example, one of the PIF factors, PIF1/PIL5, positively regulates transcription of *GA2ox2* but negatively regulates transcription of *GA3ox1* and *GA3ox2* (Oh et al. 2006). PIF1/PIL5 interacts preferentially with the Pfr form of phyA and phyB. Interaction with phytochromes leads to degradation of the PIF1/PIL5 protein, resulting in altered expression of the *GA3ox1, GA3ox2,* and *GA2ox2* genes, increased accumulation of bioactive GA, and stimulation of seed germination (Figure 2A) (Martinez-Garcia et al. 2000; Oh et al. 2006).

In addition to the regulation of GA metabolism, light may also affect GA signal transduction to promote seed germination (Cao et al. 2005). It has been reported recently that removal of the negative regulators of GA signal transduction pathway can rescue the none-germinating phenotype of the *ga1* mutant. *Arabidopsis* has at least five DELLA proteins, *GAI, RGA*, *RGL1*, *RGL2*, and *RGL3*, but the direct involvement of those proteins in photomorphogenesis has not been extensively studied. In the absence of exogenous application of GAs, the *ga1* mutant fails to germinate in either dark or light, the *ga1rgargl1rgl2* and *ga1gairgl1rgl2* triple mutants could germinate in light but not in dark, whereas the *ga1gairgargl2* triple mutant and *ga1gairgargl1rgl2* quadruple mutant could germinate in both light and dark. It appears that, at least in the seed germination process, light acts in a way similar to that of GA-triggering degradation or inactivation of DELLA proteins such as GAI and RGA to activate the GA signal transduction pathway. This hypothesis, however, is yet to be directly tested.

In nature, germinating seeds are often buried under soil in the dark and they need to push the embryonic leaves (cotyledons) out of soil by rapid elongation of the embryonic stem (hypocotyls). When the cotyledons emerge above the soil surface to light, there seems little need to keep pushing anymore; the energy and recourses would be better spent on other developmental processes, such as leaf expansion and chloroplast formation. Next to greening, inhibition of hypocotyl elongation is probably the most visible response to light in young seedlings. Indeed, light inhibition of hypocotyl elongation has been arguably the most widely used read-out in the study of plant photoreceptors. How is GA involved in the photomorphogenic processes, such as light inhibition of hypocotyl elongation? Alternatively, this question may be asked in a different way: how is GA involved in skotomorphogenesis or seedling development in the absence of light? In a series of elegant experiments, it has recently been shown that GA is essential for skotomorphogenesis (Alabadi et al. 2004). GA is not only a positive regulator promoting hypocotyl elongation in etiolated *Arabidopsis* seedlings, it also acts as a negative regulator of other photomorphogenesis responses, such as light-induced gene expression and cotyledon opening. In dark-grown or etiolated seedlings, application of paclobutrazol, which prevents GA biosynthesis by inhibiting KO, suppresses hypocotyl elongation but stimulates cotyledon opening. Surprisingly, the application of paclobutrazol also enabled seedlings to express

light-induced genes such as *CAB2* and *Rbcs* in the absence of light (Alabadi et al. 2004). Consistent with the role of GA in skotomorphogenesis, *ga1* mutant seedlings grown in the dark also had short hypocotyl, opened cotyledons, and ectopic expression of mRNA of the *CAB2* and *Rbcs* genes (Alabadi et al. 2004). Interestingly, the phenotype of *ga1* resembles that of *cop* (constitutive photomorphogenesis) and *det* (de-etiolated) mutants (Chory et al. 1989; Deng et al. 1989). The *cop/det* phenotypes of the *ga1* mutant is suppressed in the presence of both *gai* and *rga* mutations. Based on these observations, one might argue that suppression of GA accumulation would be all phytochromes need to do to bring about photomorphogenic development in a seedling. Although this scenario is apparently oversimplified, results of numerous studies have demonstrated that light regulation of GA metabolism/catabolism does play an important, and probably indispensable role, in photomorphogenesis of young seedlings (Kamiya and Garcia-Martinez 1999; Hedden and Phillips 2000; Garcia-Martinez and Gil 2001; Halliday and Fankhauser 2003).

In contrast to light stimulation of the accumulation of bioactive GA in germinating seeds, light appears to suppress the accumulation of bioactive GAs in young seedlings (Figure 2B). Many of the studies on the light control of GA metabolism in seedlings have been carried out in the garden pea (*Pisum sativum*), partially because it is easier to collect more tissues from pea seedlings for the GA analysis (Ait-Ali et al. 1999; Gil and García-Martinez 2000; O'Neill et al. 2000; Weller et al. 2001; Reid et al. 2002; Symons and Reid 2003). For example, the bioactive GA_4 in *Arabidopsis* was hardly detectable in some early studies, because of the difficulty in collecting sufficient *Arabidopsis* seedling tissues (Reed et al. 1996). This appears to not be a problem for peas. It has been shown that the level of bioactive GA_1 decreased to the trace amount soon after etiolated pea seedlings were exposed to light (Ait-Ali et al. 1999; Gil and García-Martinez 2000; Symons and Reid 2003). In a systematic analysis of how different hormones in pea seedlings change their levels in response to light, it was found recently that the GA_1 level decreased approximately 10-fold during the first 4 hours of light treatment. In contrast, the levels of IAA and ABA changed much later after light exposure (Symons and Reid 2003).

Given that the amount of GA_1 decreases in response to light in young pea seedlings, one may expect that the expression of genes required for the synthesis of bioactive GAs, such as *GA20ox* and *GA3ox* would decrease in response to light. Contrary to this expectation, it has been reported that the expression level of two major GA biosynthesis genes, *GA20ox* and *GA3ox*, increased by about 5-fold within the first 4 hours of light treatment (Ait-Ali et al. 1999; Gil and García-Martinez 2000). This perplex observation was interpreted by the feedback inhibition of GA on the expression of GA biosynthetic genes. It was shown that the light induction of GA biosynthetic genes is controlled by phyA and phyB (Ait-Ali et al. 1999), but it remains unclear whether the phytochrome-dependent upregulation of *GA20ox* and *GA3ox* is a direct or indirect consequence of the phytochrome activity. Given this, could phytochromes mediate light suppression of GA_1 accumulation via activation of GA catabolism? Indeed, the mRNA expression of a GA-inactivating gene, *GA2ox*, increased approximately 5-fold in response to light, and that the light-induced *GA2ox* expression was significantly impaired in the pea *phyA* (*fun1*) or *phyB* (*lv*) mutant (Weller et al. 1995; Weller et al. 1997; Ait-Ali et al. 1999). Therefore, it is most likely that phytochromes mediate light-induced expression of *GA2ox* to suppress the accumulation of bioactive GA in response to light, resulting in reduced hypocotyl elongation. However, it has also been noticed that the amount of bioactive GA appears to climb back to "normal" after a prolonged light exposure, probably as a result of the feedback upregulation of *GA20ox* and *GA3ox* expression (Ait-Ali et al. 1999; Symons and Reid 2003). It remains unclear how the inhibition of seedling elongation under prolonged illumination is sustained. But it has been suggested that, at least in peas, the light effect in the relatively later stages of seedling photomorphogenesis may be accomplished by light regulation of the GA signaling process (O'Neill et al. 2000).

Cryptochrome Regulation of GA Metabolism

The blue light-receptor cryptochrome was first discovered in a study of *Arabidopsis* seedling development in response to blue light, although it has since been found in animals and bacteria as well (Ahmad and Cashmore 1993; Lin et al. 1995).

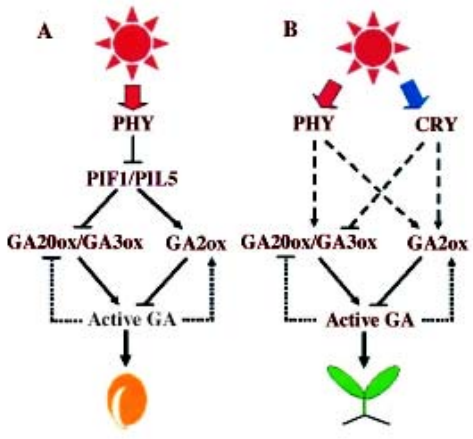

Figure 2. Working models depicting effects of light and GA on seed germination **(A)** and seedling growth **(B)**.

Arrows and T-bars represent positive or negative effect, respectively. Dashed lines indicate that more than one step may be involved between the two points.

Cryptochromes mediate blue light inhibition of hypocotyl elongation in *Arabidopsis* and other plants (Cashmore 2003; Lin and Shalitin 2003). Similar to phytochromes, cryptochromes may also regulate seedling development via a control of GA metabolism/catabolism (Figure 2B). Indeed, it has been shown that etiolated pea seedlings exposed to blue light exhibited a 5-fold decline in the level of GA_1, which was accompanied by an increase in the level of GA_8; a physiologically inactive catabolic product of GA_1 (Foo et al. 2006). This blue light-induced decrease of GA_1 is significantly compromised in the pea *cry1phyA* double mutant, demonstrating that cry1 and phyA act redundantly in mediating blue light suppression of GA_1 accumulation in pea seedlings. The redundant function of cry1 and phyA may explain why the pea monogenic *cry1* mutant showed little phenotypic alternation (Platten et al. 2005). cry1 and phyA also act redundantly to mediate blue light-induced down- and upregulation of mRNA expression of the *PsGA3ox1* and *PsGA2ox2* gene, respectively (Foo et al. 2006). It is interesting that, in contrast to the phyB- and phyA-mediated upregulation of the expression of a *GA3ox* gene discussed in the previous section, the blue light-induced downregulation of *GA3ox* appears consistent with the decreased GA_1 level in response to blue light. Why the decreased level of GA_1 did not cause a feedback upregulation of the *GA3ox* expression in blue light is unclear.

Although direct measurement of GA content in *Arabidopsis* has not been easy as discussed previously, recent improvement in the methodology of GA measurement has made it possible. And possible changes in the level of bioactive GA_4 in different *Arabidopsis* genotypes in response to blue light have been recently investigated by James Reid's laboratory in Australia (J. Reid, personal communication). It was found that the level of bioactive GA_4 decreased approximately 4-fold in *Arabidopsis* seedlings exposed to blue light for 4 h. As expected, the blue light-induced decline of GA_4 was significantly impaired in the *cry1cry2* mutant. Cryptochrome-dependent changes in the expression of GA metabolic/catabolic genes have been shown in a number of recent DNA microarray analyses (Ma et al. 2001; Folta et al. 2003; Ohgishi et al. 2004). Although these studies are invaluable in offering the first glance of genome expression profiles, the dynamic nature of expression changes of GA metabolic/catabolic genes may require more detailed kinetic analyses. Such an analysis of the expression of six members of the *GA2ox* gene family and three members of the *GA20ox* gene family in *Arabidopsis* seedlings under different blue-light conditions have revealed some of those details (Zhao et al., unpublished data). The results of this study indicate that the cryptochrome-dependent decline of GA_4 in response to blue light is at least partially accounted for by an increased expression of several members of the *GA2ox* gene family, especially *GA2ox1* (Zhao, unpublished data). For example, when etiolated seedlings were exposed to blue light (100 $\mu mol \cdot m^{-2} \cdot s^{-1}$) for 24 h, all six members of the *GA2ox* gene family showed transiently increased expression. The blue light-induced expression for four of the six *GA2ox* genes, again *GA2ox1* in particular, was impaired in the *cry1* or *cry1cry2* mutant. When etiolated seedlings were compared with seedlings grown in continuous blue light, it was found that the expression of *GA2ox1* increased significantly in wild-type seedlings grown in blue light but not in the *cry1cry2* mutant seedlings. Among the six *Arabidopsis GA2ox* genes, only *GA2ox1* and *GA2ox2* showed robust circadian rhythm that peaks in the light phases, but the circadian rhythmic expression of only *GA2ox1*, not *GA2ox2*, showed significantly reduced amplitude in the *cry1* or *cry1cry2* mutant. These results demonstrated that the expression of *GA2ox1* is most dramatically affected by blue light, although the expression of other members of the *GA2ox* gene family also increased in response to blue light. This study also showed that *Arabidopsis* cry1 and cry2 are the major photoreceptors mediating blue light induction of the expression of *GA2ox* genes, especially *GA2ox1*. In contrast to the blue light-induced expression of *GA2ox* genes, the expression of the *GA20ox* genes appear to be suppressed by blue light. All three members of *GA20ox* gene family showed transient and moderate decreases in their mRNA expression during the first hours of blue-light treatment. Interestingly, those expression changes seemed not significantly affected in the *cry1cry2* double mutant, implying the involvement of phytochromes. However, when arabidopsis seedlings grown under continuous blue-light were examined, the *GA20ox1* expression decreased dramatically in blue light-grown wild-type seedlings, but not in the *cry1cry2* mutants. Therefore, cryptochromes mediate not only blue-light induction of the expression of *GA2ox1*, but also blue-light suppression of the expression of *GA20ox1*. Both would contribute to a decreased level of GA_4 in young seedlings exposed to blue light (Figure 2B).

Conclusions

Light regulation of GA levels and GA signal transduction are important mechanisms underlying photomorphogenesis in plants. Light modulation of GA accumulation is accomplished, at least partially, by regulation of the expression of GA metabolic genes encoding GA20ox and GA3ox, and GA catabolic genes encoding GA2ox. Two complexities associated with the GA metabolic/catabolic genes are: (1) they are prone to feedback regulation by the cellular level of GAs, and (2) different members of the same gene families may be differentially regulated. Systematic analyses of the level of bioactive GAs and the expression of each members of *GA20ox*, *GA3ox*, and *GA2ox* gene families in different genetic backgrounds under different kinetic conditions would further our understanding of photomorphogenesis.

Phytochromes are the major photoreceptors that mediate light promotion of seed germination. Phytochromes stimulate rapid accumulation of bioactive GA in germinating seeds via modification of the expression of members of *GA20ox*, *GA3ox*, and *GA2ox* gene families. In *Arabidopsis*, phytochromes interact with bHLH transcription factors, such as PIF1/PIL5, which is a negative regulator of *GA3ox* and a positive regulator of *GA2ox*, to regulate GA homeostasis. Phytochromes mediate light-induced degradation of PIF1/PIL5 protein, resulting in an increased accumulation of bioactive GA and stimulation of germination. It is clear that phytochromes also regulate GA homeostasis in seedling development, and that phytochromes affect seedling growth via, at least partially, stimulation of *GA2ox* expression. How phytochromes regulate expression of GA metabolic/catabolic genes in developing seedlings is not completely clear, but modification of protein stability and/or activity of transcription factors are apparently the possible mechanisms. Control of GA homeostasis is also an important mechanism underlying the function of cryptochromes. Similar to phytochromes, cryptochromes suppress accumulation of bioactive GA in developing seedlings in response to blue light. It appears that cryptochromes suppress the expression of *GA20ox*, but stimulate the expression of *GA2ox*. However, the molecular mechanism associated with cryptochrome-regulated gene expression in general remains to be elucidated.

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