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Interactions Between Light and Plant Hormones During De-etiolation

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Abstract

The transition from a dark-grown (etiolated) to a light-grown (de-etiolated) morphology is marked by a number of dramatic phenotypic changes such as a significant reduction in the rate of shoot elongation, opening of the apical hook, expansion of true leaves and the development of mature chloroplasts. Many of these developmental processes are also known to be regulated by plant hormones. In this review we discuss the interactions between light and plant hormones and their role in mediating phenotypic change during de-etiolation. Clear evidence exists for a light-mediated reduction in gibberellin A, GA levels and response in pea, which is thought to be responsible, at least in part, for the reduction of shoot elongation during de-etiolation. Indirect evidence from a number of species has been used to suggest that the reduction in shoot elongation could also be mediated by a reduction in brassinosteroid (BR) levels. However, direct evidence recently obtained from pea and rice demonstrates that de-etiolation is not mediated, or even accompanied, by a reduction in BR levels. Ethylene is known to play an integral role in apical hook formation and maintenance in plants. However, the physiological significance of light-induced changes in IAA and ABA levels found in some species is not clear. Recent molecular data provide evidence of interactions between light-and IAA/CK-signalling pathways. Potential mechanisms for these interactions are discussed.

Key words: Light; De-etiolation; Brassinosteroids; Gibberellins; Auxin; Cytokinin; Abscisic acid; Ethylene

Introduction

Light has a profound influence on virtually all aspects of plant growth and development, including seed germination, seedling development, morphology and physiology of the vegetative stage, the control of circadian rhythms and flowering (Kim and others 2002; Nemhauser and Chory 2002). The effect of light on plant growth and development is perhaps most obvious during the transition from a dark-grown (etiolated) to a light-grown (de-etiolated) morphology. Etiolated dicotyledonous seedlings exhibit a phenotype that includes a pronounced apical hook, elongated epicotyl/hypocotyl and undifferentiated chloroplast precursors (Chory and others 1996; Clouse 2001). Upon exposure to light, seedlings undergo a number of dramatic changes, including a significant reduction in the rate of elongation, opening of the apical hook, expansion of true leaves and the development of mature chloroplasts (Chory and others 1996; Clouse 2001; Figure 1).

While the perception of light through photoreceptors is well understood, the downstream components of light-signal transduction and the mechanisms by which light mediates phenotypic

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change are not clear (Fankhauser and Chory 1997; Fankhauser and Staiger 2002; Nemhauser and Chory 2002). However, many of the light-induced changes during de-etiolation, particularly the change in stem elongation, are also known to be regulated by plant hormones (Garcia-Martinez and Gil 2002). Thus the integration of light and hormone signalling pathways is also thought to be required for normal plant development (Clouse 2001). Indeed, plant hormones are thought to act as transducers of the light signal by mediating the effects of light on plant growth and development (Nemhauser and Chory 2002). A number of plant hormones have been implicated in the regulation of morphological change during de-etiolation, including gibberellins (GA), indole-3-acetic acid (IAA), abscisic acid (ABA), cytokinins (CK), brassinosteroids (BRs) and ethylene (Chory and Li 1997; Garcia-Martinez and Gil 2002; Kraepiel and Miginiac 1997; Neff and others 2000; Tian and Reed 2001).

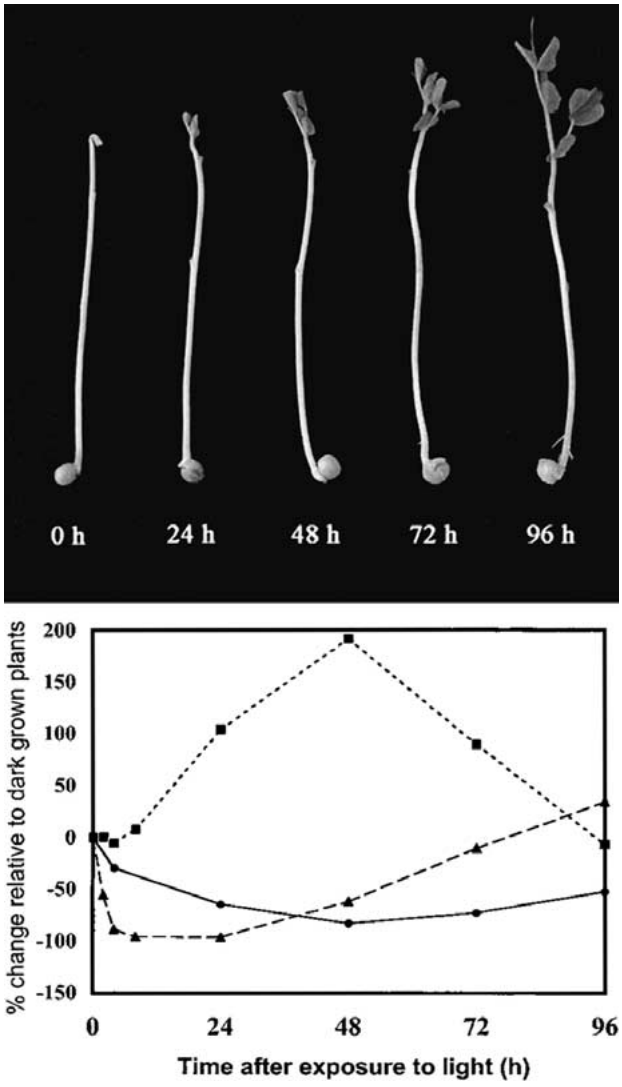

Figure 1. Top: Morphological changes in WT pea seedlings during de-etiolation. All plants were grown for 7 days at 20°C in continuous darkness before being transferred to continuous W light at an intensity of 150 μmol $m^{-2}s^{-1}$. **Bottom:** Percentage change in endogenous hormone levels in etiolated WT plants after exposure to light. All plants were grown for 7 days at 20°C in continuous darkness before being transferred into continuous W light at an intensity of 150 μmol $m^{-2}s^{-1}$. Values represent the percentage change in hormone levels (relative to the ng/g FW levels in the dark-grown controls) at various time-points (0, 2, 4, 8, 24, 48, 72, and 96 h) after exposure to light. Each value was calculated using the mean hormone levels, determined from three individual replicates, each containing either 5 or 6 plants. □ indicates the change in IAAlevels, ○ the change in ABA and Δ the change in GA_1 at each time point. After Symons and Reid (2003).

The aim of this review is to outline recent advances in our understanding of the way in which the effects of light on plant development during de-etiolation are mediated by changes in plant hormone levels and response. Each of the major groups of hormones thought to play a role in de-etiolation is covered, with a particular emphasis on the role/s played by GAs and the BRs.

Gibberellins

De-etiolation is Mediated by Changes in GA Levels and Response

The involvement of GAs in de-etiolation has been suggested to occur in a number of plant species, including pea, *Arabidopsis*, lettuce and rice (Garcia-Martinez and Gil 2002; Kraepiel and Miginiac 1997; Nemhauser and Chory 2002). Indeed, after some controversy (reviewed by Garcia-Martinez and Gil 2002) a role for GA in de-etiolation of pea (*Pisum sativum* L.) is now firmly established. It is clear that the level of the major bioactive GA, GA_1, drops during the first 24 h of exposure to light (Ait-Ali and others 1999; Gil and Garcia-Martinez 2000; O'Neill and others 2000; Reid and others 2002; Symons and Reid 2003) (Figure 1). Results suggest that these rapid changes in GA levels are controlled by phytochrome A (phyA), and a blue-light (B) receptor (Reid and others 2002). Red light appears to control GA_1 levels by down-regulating the expression of Mendel's *LE* (*PsGA3ox1*) gene that controls the conversion of GA_{20} to GA_1, and by up-regulating *PsGA2ox2* which codes for a GA 2-oxidase that converts GA_1 to the inactive GA_8. These changes in gene expression occur within 0.5 to 1 h of exposure to red light and precede changes in endogenous GA_1

levels. Similar responses occur in B light. In addition to the reduction in GA levels during de-etiolation, the ability of plants to respond to GA_1 has also been shown to decrease after exposure to light (O'Neill and others 2000). Indeed, this phytochrome B (phyB)-mediated reduction in GA response allows the continued inhibition of shoot elongation after exposure to light, even though GA levels return to homeostatic levels (O'Neill and others 2000; Reid and others 2002; Symons and Reid 2003) (Figure 1). Together these results provide a significant insight into the likely mechanism by which light mediates morphological change (particularly the reduction in shoot elongation) during de-etiolation in pea.

Although the effect of light on GA biosynthesis during de-etiolation in pea is well defined, the situation in other species is less clear (Nemhauser and Chory 2002). In *Arabidopsis*, studies show that *phyB* mutants exhibit increased responsiveness to GA, suggesting that light may act through phyB to negatively regulate GA responsiveness in the hypocotyl during de-etiolation (Chory and Li 1997; Nemhauser and Chory 2002; Reed and others 1996). However, it remains to be seen whether the phyA-mediated reduction in GA levels which occurs in pea (Reid and others 2002) also occurs in this species. This question is particularly relevant because in *Arabidopsis*, the red-light inhibition of growth during de-etiolation has been shown to be regulated by the sequential and coordinated actions of both phyA and phyB (Parks and Spalding 1999). Although analysis of hormone levels in de-etiolating *Arabidopsis* plants are inherently difficult due to the small size of dark-grown seedlings, such studies will be essential to gain a more complete understanding of the role of GA during de-etiolation in this species.

Light-Mediated Changes in GA Levels: A Widespread Phenomenon

Although evidence that de-etiolation is mediated by changes in GA levels is largely restricted to studies on pea, there are other instances where light clearly regulates growth and development by modulating the levels of bioactive GAs (reviewed by Garcia-Martinez and Gil 2002; Kamiya and Garcia-Martinez 1999; Yamaguchi and Kamiya 2002). Perhaps the best-studied examples are the light-regulated control of seed germination in lettuce and *Arabidopsis*. In lettuce, the *Ls3h1* gene is dramatically up-regulated by red light, which leads to increased GA_1 levels and the promotion of seed germination (Toyomasu and others 1993, 1998). Similarly, in *Arabidopsis* two genes encoding GA 3β-hydroxylases, *GA4* and *GA4H*, are also induced by red light (Yamaguchi and others, 1998). Through the use of a *phyB* mutant it was shown that *GA4H* was regulated by phyB but that some other member of the phytochrome gene family presumably regulates the *GA4* gene (Yamaguchi and others 1998). Although GA levels were not directly determined in this study, the results suggest that a light-induced, phytochrome-mediated increase in GA levels is also responsible for seed germination in *Arabidopsis* (Yamaguchi and others 1998). Regulation of GA levels by photoperiod has also been shown in long-day rosette plants such as spinach (Talon and others 1991) and during tuberization in potato (Xu and others 1998). GA 20-oxidase mRNA levels are regulated by light in spinach (Wu and others 1996) whereas in potato phyB mediates the tuberization response (Jackson and others 2000). These results suggest that phytochrome-mediated regulation of GA levels is a widespread phenomenon that controls many aspects of growth and development. However, the biosynthetic steps affected by light may vary among different species and different developmental processes.

Indole-3-Acetic Acid (IAA)

Light Modulates Auxin-signalling Pathways

Recent molecular studies of light- and auxin-signal transduction in *Arabidopsis* have produced strong evidence that light may modulate auxin-signalling pathways (reviewed by Swarup and others 2002; Tian and Reed 2001). This idea is supported by results which demonstrate that many auxin-regulated genes can also be regulated by light. Such genes include members of the *GH3*, *SAUR* and *Aux/IAA* families, which are up-regulated within minutes of auxin application and whose products are thought to play a central role in IAA signalling by acting as modulators of transcription (Hagen and Guilfoyle 2002). The Aux/IAA proteins provide an example of the proposed link between auxin signalling and light. Mutations in *Arabidopsis Aux/IAA* genes such as *AXR2*, *AXR3* and *SHY2* induce photomorphogenic characteristics in dark-grown seedlings (Colon-Carmona and others 2000 and references therein), suggesting that light may normally regulate these genes or proteins to induce morphological responses (Tian and Reed 2001). Consistent with this suggestion are results which show that light regulates the expression of the *SHY2* gene (Tian and others 2002). Furthermore, Aux/IAA proteins from *Arabidopsis* and pea can interact with, and are phosphorylated

by oat phyA *in vitro* (Colon-Carmona and others 2000). Together these results suggest that phytochrome-dependent phosphorylation of Aux/IAA proteins may provide a molecular mechanism for integrating light and auxin signalling in plant development (Colon-Carmona and others 2000; Tian and Reed 2001). In particular, it is thought that some phyA-mediated responses could be facilitated by changes in the phosphorylation status of Aux/IAA proteins, thereby fine-tuning the expression of auxin-regulated genes (Swarup and others 2002).

Additional evidence for a link between light and IAA signalling comes from the *Arabidopsis HY5* gene, which encodes a bZIP transcription factor that binds to the promotors of light-induced genes, and acts as a positive regulator of photomorphogenesis (Osterlund and others 2000; Oyama and others 1997). Loss-of-function *hy5* mutants exhibit an auxin-related phenotype (Oyama and others 1997) and increased hypocotyl length in the light (Koornneef and others 1980). Results suggest that light regulates the stability of the HY5 by controlling the nuclear abundance of the photomorphogenic repressor protein, COP1 (Osterlund and others 2000). In the dark COP1 is located in the nucleus, where it is thought to regulate HY5 abundance by targeting it for degradation. However, in the light COP1 is localized in the cytoplasm and cannot interact with HY5, therefore presumably allowing HY5-activated gene expression that is required for normal photomorphogenesis (Osterlund and others 2000; Swarup and others 2002).

Effect of Light on IAA Levels

Endogenous IAA levels have also been proposed to be an important determinant in phytochrome-mediated growth suppression during de-etiolation (Chory and others 1996). Studies using pea seedlings (Behringer and Davies 1992) and apical mesocotyl tissue of maize (Jones and others 1991) show that exposure of dark-grown plants to continuous R light causes a small reduction in IAA levels in the epidermis. This suggests that phytochrome may regulate stem elongation rates by depleting auxin within the epidermis, which in turn could constrain the growth of the entire stem (Behringer and Davies 1992; Jones and others 1991). Further support for this hypothesis was found in the pea *phyB* (previously *lv*) mutant (Weller and others 2001). The phyB-deficient plants exhibit increased stem elongation and contain slightly elevated levels of IAA in the epidermis (Behringer and others 1992). Thus, it was suggested that the *phyB* mutation might result in increased internode growth in part by blocking the ability of phytochrome to decrease epidermal IAA levels (Behringer and others 1992). Although the changes in IAA levels in these studies were relatively small, the results do suggest that light-induced decreases in IAA levels may contribute to the inhibition of plant cell elongation after exposure to light (Kraepiel and Miginiac 1997). Furthermore, results obtained from *Arabidopsis* suggest that light-regulated changes in IAA levels could be mediated by changes in IAA transport (reviewed by Nemhauser and Chory 2002; Tian and Reed 2002). However, if decreases in IAA levels do play a role in de-etiolation in pea, then these decreases must be highly localized, as endogenous-free IAA levels in the whole shoots of dark-grown pea seedlings were actually significantly increased after exposure to white light (Symons and Reid 2003) (Figure 1).

Brassinosteroids (BRS)

There is a long-standing and widely cited view that BRs act as negative regulators of de-etiolation. However, recent direct evidence has been obtained that casts significant doubt over the validity of these claims.

Interaction Between Light and Brassinosteroids: An 'Historical' Perspective

The suggestion that BRs play a negative-regulatory role in de-etiolation is based largely on indirect evidence, such as the 'de-etiolated' phenotype of many dark-grown BR mutants (Chory and others 1996; Chory and Li 1997; Li and others 1996). For instance, when grown in the dark, *Arabidopsis* BR mutants such as *dim*/*dwf1* (Choe and others 1999a; Klahre and others 1998; Takahashi and others 1995), *det2* (Fujioka and others 1997; Li and others 1996; 1997; Noguchi and others 1999b), *dwf4* (Azpiroz and others 1988, Choe and others 1998), *cpd* (Szekeres and others 1996), *dwf5* (Choe and others 2000), *bri1* (Clouse and others 1996; Li and Chory 1997; Noguchi and others 1999a), *bin2* (Li and others 2001) and *dwf12* (Choe and others 2002) all reportedly exhibit a de-etiolated phenotype, characterized by short hypocotyls, expanded cotyledons and developing leaves. Similar de-etiolation characteristics have been reported in dark-grown tomato (d^x) and rice (*d61*, *brd1*) BR mutants (Bishop and others 1999; Hong and others 2002; Mori and others 2002; Yamamuro and others 2000). Furthermore, treatment of dark-grown *Arabidopsis* with the BR biosynthesis inhibitor, brassinazole (Brz),

induces some morphological characteristics of light-grown plants (Nagata and others 2000). In many cases the abnormal dark-grown phenotype of such BR-deficient plants can be, at least partially, restored to a normal WT etiolated phenotype, after application of exogenous BRs (Bishop and others 1999; Chory and others 1996; Chory and Li 1997; Li and others 1996). Furthermore, the expression of the light-regulated genes, *RBCS* and *CAB*, are up-regulated in dark-grown, BR-deficient mutants, *det2* and *cpd*, and in dark-grown WT plants treated with Brz (Asami and others 2000; Chory and others 1991; Li and others 1996; Szekeres and others 1996). Together this evidence has been interpreted as suggesting a negative regulatory role for BRs in de-etiolation (Chory and Li 1997; Li and others 1996). That is, high BR levels promote normal etiolated growth, whereas the development of a de-etiolated phenotype after exposure to light is mediated (at least in part) by a reduction in BR levels.

Molecular data provide additional support for the proposed negative regulatory role of BRs in de-etiolation. For instance, microarray analysis of gene expression in *Arabidopsis* plants that were exposed to different light treatments showed that four genes involved in the BR biosynthesis pathway were all down-regulated by light (Ma and others 2001). In addition, studies by Kang and others (2001) provide an insight into a potential mechanism by which light could regulate BR levels. This work demonstrated that in pea, a light-repressible small G protein, Pra2, regulates DDWF1, a cytochrome P450 C-2 hydroxylase involved in brassinosteroid biosynthesis (Kang and others 2001); (Figure 2). It has been suggested that this interaction between Pra2 and DDWF1 represents a link between light-signal transduction and endogenous BR levels in pea (Clouse 2001; Kang and others 2001). For instance, it is proposed that, on exposure to light, phytochrome and blue light photoreceptors signal the repression of Pra2 (and therefore DDWF1), which leads to a reduction in BR levels, and a slowing of shoot growth (Clouse 2001; Kang and others 2001).

The Problem with BRs as Negative Regulators of De-etiolation

Despite its wide acceptance and a diversity of indirect supporting evidence, the suggestion that BRs negatively regulate de-etiolation is not universally accepted because neither a fully de-etiolated phenotype, nor increased expression of light-regulated genes, is characteristic of all dark-grown BR mutants. For instance, in *Arabidopsis*, expression of the

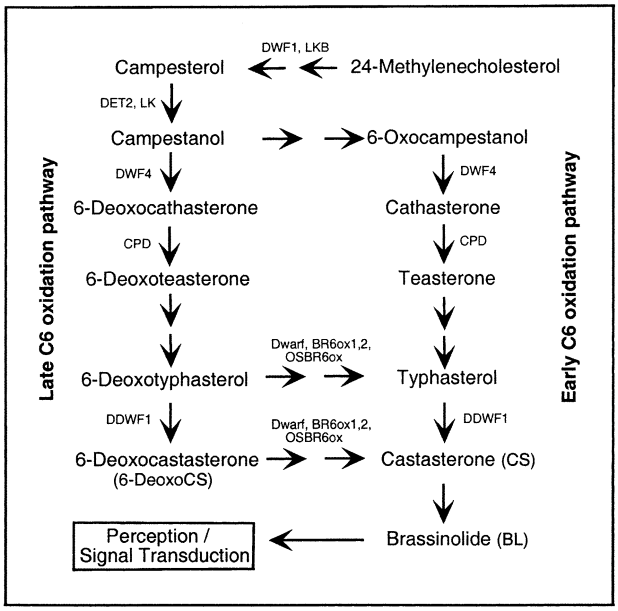

Figure 2. Proposed pathways and enzymes involved in the biosynthesis of BL from 24-methylenecholesterol (after Bishop and Yokota 2001; Kang and others 2001; Mori and others 2002; Shimada and others 2003).

CAB and *RBCS* genes is not increased (as is the case in *cpd* and *det2*) in *dim* mutant plants (Takahashi and others 1995). Furthermore, although dark-grown *dim*/*dwf1*, *det2*, *dwf4*, *cpd*, *bri1* and *bin2* BR mutants are reported to exhibit a de-etiolated phenotype (see above), the phenotypes of dark-grown, BR-deficient *sax1* and *dwf7* mutants are, at most, only partially de-etiolated (Choe and others 1999b; Ephritikhine and others 1999a; 1999b). Indeed, the dark-grown phenotype of *dwf7* plants consists of closed cotyledons and an intact apical hook (Choe and others 1999b), whereas dark-grown *sax1* seedlings display an etiolated phenotype close to that of the wild type (Ephritikhine and others 1999a). Some controversy exists as to whether the de-etiolated phenotype exhibited by some dark-grown, *Arabidopsis* BR mutants is in fact a secondary consequence of the retarded cell elongation in these mutants, rather than an interruption in the normal light signal transduction pathway (see Altmann 1998; Bishop and Yokota 2001; Goda and others 2002; Nagata and others 2000). For instance, Azpiroz and others (1998) suggested that the apparent de-etiolated phenotype of dark-grown *dwf4* plants might be due to their dwarfed stature and growth on agar plates. However, Nemhauser and Chory (2002) argue that the up-regulation of light-regulated genes seen in dark-grown *det2* and *cpd* mutants can only be a true 'misreading' of the light conditions rather than a consequence of growth inhibition.

A similar degree of inconsistency exists in the dark-grown phenotypes of tomato (*Lycopersicon esculentum* L.) BR mutants. For example, though the extreme d^x mutant shows a de-etiolated phenotype when grown in the dark (Bishop and others 1999), the *dpy* mutant (also thought to be defective in BR biosynthesis) retains a pronounced apical hook and closed cotyledons and therefore is not truly de-etiolated (Koka and others 2000). Furthermore, a mutation in the *Curl3* gene (the tomato homolog of the *Arabidopsis BRI1* gene, which encodes the BR receptor) results in a partially de-etiolated, dark-grown phenotype that includes a partial apical hook and some opening of the cotyledons (Koka and others 2000; Montoya and others 2002).

The situation is perhaps clearest in pea (*Pisum sativum* L.) because pea BR-deficient mutants *lk* and *lkb* are not de-etiolated at the morphological or molecular level, as they exhibit neither a de-etiolated phenotype or altered expression of light-regulated genes when grown in the dark (Symons and others 2002). Similarly, dark-grown WT plants treated with the BR biosynthesis inhibitor, Brz, do not exhibit a de-etiolated phenotype (Symons and others 2002). Indeed, such evidence suggests that BR levels do not play a negative regulatory role in de-etiolation in this species (see Symons and others 2002).

The major problem with the suggestion that BRs negatively regulate de-etiolation is that, until recently, there were no reports of actual measurements of BR levels in light- and dark-grown plants of any species. A reduction in BR levels in light-grown plants is implicit in the argument that BRs play a negative regulatory role in de-etiolation (Clouse 2001). Therefore, direct evidence of endogenous BR levels is crucial in order to substantiate these claims.

Direct Evidence Provides a New Perspective

To address the need for direct evidence, Symons and others (2002) recently reported the first measurements of endogenous BR levels in light- and dark-grown pea seedlings. Significantly, the results show that BR levels were actually increased, not decreased, in light-grown pea seedlings compared with those grown in the dark (Symons and others 2002). The levels of brassinolide (BL) and that of its direct precursor castasterone (CS) were up to 17-fold and 4-fold higher, respectively, in light-grown plants than in comparable dark-grown seedlings (Symons and others 2002, Figure 2). These results are clearly inconsistent with the idea that BRs negatively regulate de-etiolation. Indeed, they suggest that de-etiolation in pea is not regulated or even accompanied by a decrease in endogenous BR levels (Symons and others 2002).

These initial findings are further supported by results from a comprehensive time-course investigation of CS and 6-deoxocastasterone (6-deoxoCS) levels in etiolated, WT pea seedlings after exposure to light (Symons and Reid 2003). Kang and others (2001; see above) have previously suggested that the light-mediated suppression of the pea *Pra2* gene causes a reduction in the levels of the DDWF1 enzyme, which catalyzes the formation of 6-deoxoCS and CS (see Figure 2). However, no substantial decrease in endogenous 6-deoxoCS or CS levels was evident in WT pea seedlings after exposure to light (Symons and Reid 2003). Indeed, this was the situation throughout a detailed time-course study, which demonstrated that there is not even a transitory decrease in BR levels, as previously shown for GA_1 (O'Neill and others 2000; Reid and others 2002; Symons and Reid 2003). Furthermore, 6-deoxoCS levels actually increased (approximately 3-fold) by 96 h after exposure to light, suggesting an up-regulation of BR biosynthesis, via the late C-6 oxidation pathway, during de-etiolation (Symons and Reid 2003, Figure 2). This is consistent with results showing endogenous CS and BL levels are higher in plants grown in continuous light than in dark-grown plants (Symons and others 2002). Together these findings suggest that in pea, BR biosynthesis is not down-regulated by light, as was suggested by Kang and others (2001). Indeed, when we also consider that dark-grown, pea BR mutants are not de-etiolated at either the morphological or molecular level (Symons and others 2002), it is reasonable to conclude that BRs do not negatively regulate de-etiolation in pea.

Tamaki and others (2002) have shown that BR levels are also higher in rice shoots grown under white light than those grown in the dark. As was the case in pea (Symons and others 2002; Symons and Reid 2003), the levels of CS and 6-deoxoCS levels were increased markedly in light-grown rice shoots (Tamaki and others 2002). Further analysis indicates that this increase in BR levels may be due to a blue-light-mediated increase in *DWARF* transcript levels, resulting in increases in C-6 oxidation and the formation of 6-deoxoCS (Tamaki and others 2002). These results strongly suggest that BRs do not play a negative-regulatory role in de-etiolation in rice.

In *Arabidopsis*, Shimada and others (2000) have also shown that the expression of *BR6ox1* (the homologue of the rice *DWARF* gene) is up-regulated by light. Similarly, the expression of some BR-bio-

synthetic genes was increased when *Arabidopsis* seedlings were transferred from the dark to the light (Shimada and others 2001). Furthermore, preliminary data indicate that BR levels in light-grown *Arabidopsis* seedlings are increased (not decreased) compared with dark-grown plants (Y. Shimada and S. Fujioka unpublished). These studies are particularly important given the widely cited suggestion that BRs play a negative-regulatory role in de-etiolation in *Arabidopsis* (Chory and Li 1997; Li and others 1996). Further direct measurements of BR levels in *Arabidopsis* are now required to clarify the situation in this species.

In light of the results obtained from pea and rice and the preliminary data from *Arabidopsis*, we must now ask exactly what role (if any) do BRs play in de-etiolation? A recent microarray analysis of BR-regulated genes has shown that BRs down-regulate the expression of a gene encoding PIF3, a transcription factor that functions at the upstream end of the light-signalling pathway (Goda and others 2002). As a consequence, these authors suggest that BRs may act as regulators of the light-signalling pathway, in addition to or rather than functioning as down-stream mediators of light-signal transduction.

Abscisic Acid (ABA)

Several studies have also implicated ABA levels in the control of de-etiolation and light-regulated development (also reviewed by Kraepiel and Miginiac 1997). For instance, Kraepiel and others (1994) showed that the phytochrome A-deficient tobacco mutant, *pew1*, has higher levels of ABA relative to WT. Further analysis of *pew1*, using the ABA-deficient *aba1* mutant, led to the suggestion that light induces a phytochrome-mediated activation of ABA degradation (Kraepiel and others 1994). Similarly, Weatherwax and others (1996) have shown that brief red-light treatment resulted in substantial decreases in ABA concentrations in dark-grown *Lemna gibba* plants. This effect was reversible by a far-red light treatment, suggesting that phytochrome action can negatively regulate ABA levels. A light-induced decrease in ABA levels has also been reported in pea (Symons and Reid 2003) (Figure 1). In this case ABA levels in etiolated WT plants gradually decreased and reached a minimum (approximately 6-fold lower than in dark grown controls) 48 h after exposure to light. Together these results certainly suggest a negative regulation of ABA levels by light. However, as was previously pointed out by Kraepiel and Miginiac (1997), both the sequential relationship between these two signals and the physiological relevance of this relationship are not clear. Indeed, the timing of the decrease in ABA levels in de-etiolating pea seedlings suggests that these changes could be a consequence (rather than the cause) of the changing morphology after exposure to light (Symons and Reid 2003).

Cytokinins (CKs)

The similar effects of both light and CKs on a range of developmental processes has often been cited as evidence for a link between these two signals (Krapiel and Miginiac 1997; Su and Howell 1995). Exogenous application of CKs promotes de-etiolation in dark-grown *Arabidopsis* plants (Chory and others 1994), consistent with the suggestion that light could mediate changes during de-etiolation by positively regulating CK levels. Similarly, the *Arabidopsis amp1* mutant, which has increased CK content, also develops a light-grown phenotype in the dark (Chaudhury and others 1993; Chin-Atkins and others 1996). However, in one of the few studies of light and CK levels, Chory and others (1994) concluded that the de-etiolated dark-grown phenotype of the *Arabidopsis det1* mutant was unlikely to be caused by altered levels of CKs. In addition, there is no detectable regulation of CK levels in WT *Arabidopsis* plants exposed to different light regimes, suggesting that de-etiolation is not regulated by changes in CK levels under normal conditions (Chory and others 1994; Nemhauser and Chory 2002). Thus it remains unclear whether light and CKs act independently to affect developmental responses, or whether changes in CKs levels act as downstream transducers of the light signal.

Interactions Between Light and CK Signalling Pathways

The identification of a CK receptor, together with several elements that act in the CK signalling pathway, have provided insights into the possible interaction between light- and CK-signalling pathways (Fankhauser 2002). Evidence suggests that an integral component of CK signalling is the *Arabidopsis* response regulators (ARR), which act downstream of the CK receptor (reviewed by Schmülling 2002). Sweere and others (2001) have shown that one such ARR, ARR4, which is induced by cytokinin (see Schmülling 2002 and references therein), is also expressed in response to phyB action. Significantly, it was shown that ARR4 specifically interacts with the extreme amino-terminus of phyB, which

stabilizes the Pfr form of this photoreceptor and therefore increases the levels of active phyB (Sweere and others 2001). This has led to the suggestion that ARR4 may act as a signal module at which cytokinin- and light-signal transduction pathways converge to integrate information from these two signals (Fankhauser 2002; Sweere and others 2001). Thus it appears that, rather than acting as a downstream component of the light-signal-transduction pathway, CKs may actually modulate the light response via an ARR4-mediated control of phyB action.

Ethylene

Ethylene Mediates Apical-hook Formation

A prominent aspect of the etiolated phenotype is the presence of the apical hook. This hook-like structure is formed at the apical end of the epicotyl/hypocotyl of dicot seedlings to protect the delicate shoot meristem as the seedling makes its way through to the soil surface (Raz and Ecker 1999 and references therein). Formation of the apical hook is facilitated by differential cell growth in the epicotyl/hypocotyl. As the cells exit the apical meristem, those on the inner side of the hook elongate more slowly than do those on the outer side, resulting in curvature of the stem (Peck and others 1988 and references therein). However, upon exposure to light this differential growth ceases and the apical hook opens, a change that is irreversible (Raz and Ecker 1999).

Studies of the formation, maintenance and light-induced opening of the apical hook have provided strong evidence for the involvement of two different plant hormones, IAA and ethylene, in the regulation of these processes (see Lehman and others 1996; Raz and Ecker 1999; Swarup and others 2002). It is clear that apical hook formation is an ethylene-dependent process because both ethylene-treated *Arabidopsis* seedlings and ethylene over-producing mutants exhibit exaggerated hook curvature, whereas ethylene-insensitive mutants exhibit a hookless phenotype (Swarup and others 2002 and references therein). Similarly, IAA-treated *Arabidopsis* seedlings or IAA-overproducing mutants also disrupt hook formation, therefore indicating that IAA may also be involved in this process (Swarup and others 2002 and references therein). However, understanding the relative roles of IAA and ethylene in apical-hook formation has proven difficult, largely because of the functional overlap between the biosynthetic and response pathways of these two substances (Harper and others 2000; Swarup and others 2002 and references therein).

Studies conducted by Lehman and others (1996) indicate that the effect of ethylene on apical hook formation may be mediated by auxin. This suggestion arose out of studies on the *Arabidopsis hookless* (*hls*) mutants such as *hls1*, which does not form an apical hook in the dark. Ethylene was shown to up-regulate *HSL1* expression, and *HLS1* over-expressing plants have an exaggerated apical hook. Furthermore, endogenous IAA levels and the spatial patterns of expression of two intermediate early auxin-responsive genes are altered in the *hls1* mutants. Together these results are thought to suggest that ethylene-regulated expression of *HLS1* mediates apical hook formation by controlling IAA activity (Lehman and others 1996). However, Swarup and others (2002) question the link among ethylene, *HLS1* expression, IAA and asymmetric growth because Lehman and others (1996) reported a uniform pattern of *HLS1* expression across the apical hook.

Other studies into the factors controlling apical-hook formation suggest that ethylene may not act by regulating IAA activity. For instance, it has been shown that a gene involved in ethylene biosynthesis in *Arabidopsis* is expressed differentially in outer and inner apical hook tissues (Raz and Ecker 1999), indicating that apical hook formation in *Arabidopsis* may be a direct result of asymmetric ethylene biosynthesis (Raz and Ecker 1999; Swarup and others 2002). Similarly, Du and Kende (2001) propose that ethylene may also be the primary factor in apical hook formation in pea. In this species apical hook formation is thought to be mediated by an asymmetrically distributed component of the ethylene signal-transduction pathway (Peck and others 1988). However, neither author has ruled out the possibility that IAA also has a role in apical hook formation in pea (Du and Kende 2001; Peck and others 1988).

Indeed, Harper and others (2000) highlight the intimate connection between IAA and ethylene in the control of growth. These authors have shown that the *Arabidopsis NPH4* gene, which is conditionally required for differential growth responses (including apical-hook formation), encodes the auxin-regulated transcriptional activator ARF7. Interestingly the phenotypes of loss-of-function *nph4* mutants, which include multiple differential growth defects, were shown to be suppressed by application of ethylene (Harper and others 2000). This suggests that ethylene acts as a modulator of auxin-dependent differential growth (Harper and others 2000) and supports the outcomes of earlier studies by Lehman and others (1996). Thus it seems likely that with further analysis, both ethylene and IAA

will both be shown to have at least some level of involvement in apical hook formation. Obtaining direct evidence of IAA concentration and activity across the apical hook region will be an important step in elucidating the roles of IAA and ethylene in this process.

Light and Hormones: an Integrated Approach

In a recent review of this subject Nemhauser and Chory (2002) outlined an increasingly complex model for the hormonal regulation of photomorphogenesis, which involves changes in the levels of and response to multiple hormonal signals. Although previous reports provide evidence (often indirect) suggesting that light-regulated changes in hormone levels and response may regulate de-etiolation, the direct evidence required to substantiate these claims is, in many cases, lacking (see above). To address this issue, Symons and Reid (2003) recently undertook a detailed time-course investigation of IAA, GA, ABA and BR levels during deetiolation in pea. This direct, simultaneous quantification of a range of plant hormones has provided an important insight into the relative importance of these compounds in regulating de-etiolation.

Althouth BR levels remain relatively unchanged after exposure to light (Symons and Reid 2003), the simultaneous quantification of IAA, GA, and ABA levels revealed a clear pattern of changes in the levels of these hormones during de-etiolation (Figure 1). The first and most dramatic change observed was a reduction in endogenous GA_1 levels, which was detected as early as 2 h after exposure to light. Importantly, the timing of the reduction in GA_1 levels coincides with the reduction in stem elongation after exposure to light (Behringer and Davies 1992; Symons and Reid 2003). Although there was a significant reduction in the level of GA_1, the levels of BRs and IAA remained relatively unchanged during the first 8 h after exposure to light (Symons and Reid 2003). Thus, it appears likely that the reduction in GA_1 levels may be the primary factor that regulates the reduction in stem elongation during de-etiolation in this species.

It has been established that IAA positively regulates GA_1 levels in pea internodes (see Ross and others, this issue). However, the fact that IAA levels remained relatively stable in the first 8 h after exposure to light, whereas GA levels dropped markedly (Figure 1), suggests that the light-regulated reduction in GA_1 levels is unlikely to be mediated via a reduction in IAA levels. This provides further support for the suggestion that the decrease in GA_1 levels is directly mediated by light via phytochrome A and a blue light receptor (Kamiya and Garcia-Martinez 1999; Reid and others 2002). Although IAA levels were initially unchanged, long-term exposure to light resulted in a significant increase (2-fold by 24 h and 3-fold by 48 h) in IAA levels compared to the dark-grown controls (Figure 1; Symons and Reid 2003). The physiological significance of this increase is not entirely clear. However, it may reflect the rapid development of the apical bud (see Figure 1), which is the presumed site of IAA biosynthesis in plants (Davies 1995).

As was the case for GA_1 and IAA, ABA levels also changed after exposure to light (Figure 1). In this case a reduction in ABA levels was detected as early as 4 h and ABA levels reached a minimum (approximately 6-fold lower than in dark grown controls) 48 h after exposure to light (Symons and Reid 2003). This decrease in ABA levels after exposure to light is consistent with the suggestion that phytochrome action negatively regulates ABA levels (Kraepiel and others 1994; Kraepiel and Miginiac 1997; Weatherwax and others 1996). However, as is the case for the increase in IAA, the physiological relevance of this decrease in ABA levels during de-etiolation is not known. Clearly the next challenge is to understand the physiological relevance of these changes in hormone levels during de-etiolation. In doing so it is important to acknowledge that de-etiolation is a multifaceted developmental process consisting of a number of independent processes as diverse as shoot elongation and leaf development. It is likely therefore, that changes in plant hormones may play a specific role in some aspects of de-etiolation but not others. The challenge will be to dissect out which hormone signals regulate each specific aspect of the de-etiolation process.

Future Perspectives

The availability of improving technologies and new research tools provides an exciting opportunity to advance our understanding of the interaction between plant hormones and light during de-etiolation. For instance, microarray-based gene-expression analysis allows us to gain an expression profile of the genes involved in multiple hormone biosynthesis and response pathways under different light regimes. Such information will enable us to rapidly assess the relative importance of changes in levels of and response to different hormone signals during de-etiolation. However, it is crucial that such

analysis is interpreted in conjunction with results of parallel studies that provide direct evidence of actual hormone levels. Indeed, a complete understanding of the hormonal regulation of de-etiolation can only be obtained by a comprehensive approach, which integrates biochemical, molecular and genetic data to answer the questions that confront researchers in this field.

In attempting to understand the hormonal regulation of de-etiolation, we must also question whether the underlying mechanisms that mediate light-induced changes in hormone levels and response are highly conserved and similar in different species, or if they vary between species? The occurrence of similar phytochrome-mediated mechanisms for the light-regulation of GA levels and response in a diverse range of species and developmental processes indicates conservation of these mechanisms throughout evolution of different plant species. However, the situation regarding other hormones, particularly the proposed interaction between light and BRs, is less clear. Understanding those mechanisms that are highly conserved, and those that are specific to certain species, is important to our understanding of light-regulated plant development, and provides a clear incentive for the continued use of a range of different model species to study this process.

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