Understanding phototropism: from Darwin to today

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Abstract

Few individuals have had the lasting impact on such a breadth of science as Charles Darwin. While his writings about time aboard the HMS Beagle, his study of the Galapagos islands (geology, fauna, and flora), and his theories on evolution are well known, less appreciated are his studies on plant growth responses to a variety of environmental stimuli. In fact, Darwin, together with the help of his botanist son Francis, left us an entire book, ‘The power of movements in plants’, describing his many, varied, and insightful observations on this topic. Darwin’s findings have provided an impetus for an entire field of study, the study of plant tropic responses, or differential growth (curvature) of plant organs in response to directional stimuli. One tropic response that has received a great deal of attention is the phototropic response, or curvature response to directional light. This review summarizes many of the most significant advancements that have been made in our understanding of this response and place these recent findings in the context of Darwin’s initial observations.

Key words: Auxin, Chlodony–Went theory, Darwin, LOV domain, phototropin, phototropism, protein kinase.

‘The power of movements in plants’: Darwin’s lasting legacy to the field of phototropism research

Plants are sessile by nature, and thus to maximize energy production they must rely on their capacity to move directionally, or exhibit tropic responses, in response to directional environmental cues. The way plants respond to stimuli has fascinated humans since the time of Ancient Greece (Whippo and Hangarter, 2006). Although Charles (and son Francis) Darwin’s ‘The power of movements in plants’ dealt in large part with Darwin’s proposal that circumnutation could provide a unifying model to explain directional growth responses in plants (Darwin, 1880), an hypothesis that has been shown to be generally incorrect, this seminal book has provided the foundation for an entire field of study focused on the tropic responses of plants.

‘The power of movements in plants’ proposed several key elements that shape the current research on tropic responses. Darwin, although not the first to do so (for an excellent historical literature review on tropic response research, see Whippo and Hangarter, 2006), proposed that plants could grow differentially (thus directionally) in response to external stimuli such as light or gravity. Second, he demonstrated that the part of the plant that perceives the stimulus is separate and distinct from the part that responds to that stimulus. In the case of phototropism, directional light is perceived in the apical portion of a young seedling and ‘transduced’ to more basally localized portions of the shoot as a differential signal that informs the plant which side is the closest to and which is the furthest from the light source such that a bending response occurs (Fig. 1). Finally, Darwin proposed that an ‘influence’ (though he was unable to identify it) moves from the site of stimulus perception to the area of response where bending occurs (Fig. 1).

Darwin’s ‘influence’: auxin and its role in phototropism

Just after the turn of the century Boysen-Jensen (1911) was able to gain further insight into Darwin’s ‘influence’ in an experiment that used pieces of mica to disrupt the proposed influence’s flow, and the results of those experiments confirmed that the ‘influence’ does indeed participate in a plant’s response to directional stimuli, such as light.
Fig. 1. Stylized representation of phototropism. The diagrams depicted here are meant to represent the coleoptile of a dark-grown grass seedling, such as oat, which is a classic model for phototropism. On the left is a seedling soon after exposure to unidirectional blue light (half sun to the right of the seedling). The ‘eye’ is used to represent Darwin’s proposal that light sensing occurred within a specific region of the seedling (in this case the tip of the coleoptile), not that Darwin was ascribing any anthropomorphic properties to the plant per se. The downward pointing black arrows represent the downward flow of the ‘influence’ (we now know as auxin) that Darwin proposed moved from the site of light perception to the site of differential growth that results in curvature. The horizontal black arrows reflect the lateral movement of auxin occurring from the lit to the shaded side of the seedling that has been demonstrated to occur in a variety of plants. On the right is a seedling that has developed a curvature response after a refractory period ($t_x$) during which the differentially accumulated auxin (green shading) promotes localized growth (plus signs).

In recent years, genetic studies in the model plant Arabidopsis thaliana have identified proteins that appear to function as auxin transport facilitators (Leyser, 2006). At least five auxin transport proteins have been associated with stem/shoot phototropism: AUX1 (AUXIN-RESISTANT 1; Stone et al., 2008), PIN1 (PIN-FORMED 1; Blakeslee et al., 2004), PIN3 (Friml et al., 2002), MDR1 (MULTI-DROP-RESISTANT 1), and PGP1 (P-GLYCOPROTEIN 1; Noh et al., 2003). Studies in Arabidopsis have also led to important findings about how a gradient of auxin established by such transport facilitators leads to differential growth. For example, semi-dominant loss-of-function mutations in the NPH4 (~NON-PHOTOTROPIC HYPOCOTYL 4)~/~ARF7 (~AUXIN RESPONSE FACTOR 7)~ locus, and dominant gain-of-function mutations in MSG2 (~MAS-SUGU 2)~/~IAA9 and AXR5 (AUXIN-RESISTANT 5)~/~IAA1 result in severely impaired phototropic and gravitropic responses (Liscum and Briggs, 1996; Watahiki and Yamamoto, 1997; Stowe-Evans et al., 1998; Harper et al., 2000; Park et al., 2002; Tatematsu et al., 2004; Yang et al., 2004). NPH4/ARF7 is a transcriptional activator whose theory is still the prominent one used to explain a plant’s response to tropic stimuli.

Following the initial proposal of the Cholodny–Went theory, a number of hypotheses have evolved regarding how the unequal distribution of auxin occurs, particularly in response to phototropic (directional light) stimulation. For example, Went and Thimann (1937) hypothesized that the unequal auxin accumulation occurs due to either light inactivation of auxin on the stimulated side, light-induced inhibition of the production of auxin, or light-induced transport of the auxin from the lit side to the shaded side. A study by Briggs et al. (1957) showed that introducing a physical barrier between the lit and the shaded side of corn coleoptiles disrupts the formation of an auxin gradient, providing evidence against the arguments that light induces the destruction, or the inactivation, of auxin. Subsequently, Briggs (1963) published additional data that provided support for a hypothesis that the unequal distribution of auxin was due to a lateral movement or transport of auxin. Specifically, these data showed that, in maize coleoptiles, an increase in the amount of curvature in response to light was correlated to an increase in the amount of auxin present on the ‘shaded’ side of the coleoptile (Fig. 1).

Pickard and Thimann (1964) applied radio-labelled auxin, indole-3-acetic acid (IAA) in particular, to maize coleoptiles to trace the path of auxin during phototropism. It was found that IAA moves laterally across the coleoptile from the lit to the shaded side under both the pulse (first positive) and extended (second positive) irradiation conditions (Fig. 1). Based on similar radio-tracer labelling studies, Shen-Miller and Gordon (1966) proposed that light promotes a lateral accumulation of auxin by inhibiting polar auxin transport. Gardner et al. (1974) obtained additional support for the notion that light stimulates the lateral movement of auxin, although their data did not support a role of light-mediated inhibition of polar auxin transport.

In particular, Boysen-Jensen’s experiments suggested that Darwin’s ‘influence’ flows from the tip of the plant toward the base in the unlit side of the plant, and that this directional and differential movement of the ‘influence’ is critical for the plant’s bending response.

Although textbooks generally credit the Dutch plant physiologist Fritz Went with the identification on Darwin’s ‘influence’ as the now well-understood plant hormone auxin, the actual history of auxin’s chemical identification is a bit more complicated. First, it is important to give shared credit for the physiological identification of auxin to the Russian plant physiologist Nicolai Cholodny, who, while Went was working with grass coleoptiles (Went, 1926), was generating similar results with grass roots (Cholodny, 1927). It is also critical to note that, in actuality, it is Kogl and colleagues (Kogl and Haagen-Smits, 1931) at Utrecht University (where Went did his graduate work) that deserve credit for the first chemical identification of an ‘auxin’ from human urine. Cholodny (1928) and Went (1928) each independently proposed a similar mechanism by which auxin could mediate tropic responsiveness, which was later simply renamed the Cholodny–Went theory (Went and Thimann, 1937). In brief, the Cholodny–Went theory combines Darwin’s hypotheses with those of the auxin pioneers to propose that an asymmetric accumulation of auxin occurs in response to a tropic stimulus, and that this asymmetric gradient of auxin stimulates the differential growth response that results in tropic curvature. While other models have been proposed, the Cholodny–Went model is still the prominent one used to explain a plant’s response to tropic stimuli.
activity is repressed in the presence of the MSG2/IAA19 and AXR5/IAA1 (Liscum, 2002). In the presence of elevated levels of free auxin, MSG2/IAA19 and AXR5/IAA1 are rapidly degraded by a 26S proteasome that requires the SCF\textsuperscript{TIR1} complex containing the auxin receptor TIR1 to target these proteins for degradation (Tan et al., 2007). This, in turn, allows homodimerization of the NPH4/ARF7 protein and transcription of ‘auxin responsive genes’ (Tatematsu et al., 2004; Celaya et al., 2009). A recent transcript profiling study in Brassica oleracea has identified a number of genes that appear to represent targets of NPH4/ARF7 regulation in response to tropic stimulation; these include genes encoding proteins involved in the regulation of free auxin levels, additional transcriptional regulators, and proteins involved in the regulation of cell wall extensibility (Esmon et al., 2006).

Darwin’s vision: phototropin blue light receptors

In addition to proposing the existence of a mobile ‘influence’ that was necessary for tropic responses (we now know this ‘influence’ to be auxin; see above), Darwin made observations, again presented in ‘The power of movements in plants’, that indicated that tropic curvatures in response to light were not general light responses but specific with respect to light quality. In particular, Darwin was able to demonstrate that the blue region of the electromagnetic spectrum is the most effective portion of the spectrum with respect to the induction of phototropism. These findings have, like those of Darwin’s tropic ‘influence’, provided the impetus for a large number of studies over the past 100 or so years. Yet only within the past decade or so have the molecular details of how plants ‘see’ blue light cues (Fig. 1) to induce phototropism, ‘Darwin’s vision’ if you will, have become known.

As was the case with the elucidation of the molecular mechanisms underpinning the role of auxin in phototropism, Arabidopsis genetics was also a major factor in the identification of the photoreceptor molecules mediating phototropism in higher plants. The first of these photoreceptors identified at the molecular level is phototropin 1 (phot1) (Huala et al., 1997), originally designated NPH1 (for its non-phototropic hypocotyl mutant phenotype; Liscum and Briggs, 1995). The PHOT2 gene was subsequently identified based on its high degree of sequence homology to PHOT1 (Jarillo et al., 2001; Sakai et al., 2001). Phototropins regulate not just phototropism, but a number of additional blue light responses, including stomatal opening, chloroplast movements, leaf movements and expansion, and rapid inhibition of stem growth (Christie, 2007).

The functions of the phototropins in these responses are both overlapping and distinct. For example, in the case of phototropism, phot1 (see Briggs et al., 2001, for a description of nomenclature) is the dominant receptor, mediating response across a wide range of fluence rates (e.g. 0.01–100 \( \mu \)mol m\(^{-2} \) s\(^{-1} \)), whereas phot2 appears to operate only at higher fluence rates (>10 \( \mu \)mol m\(^{-2} \) s\(^{-1} \)) (Sakai et al., 2001). By contrast, with respect to stomatal regulation, both phot1 and phot2 contribute over the entire range of effective fluence rates (Kinoshita et al., 2001; Kinoshita and Shimazaki, 2002). The interplay between the phototropins is even more complex when one considers blue light-induced chloroplast movements. In high-light conditions, chloroplasts move away from the upper surface of the leaf to avoid photobleaching (Wada et al., 2003), a response that is mediated solely by phot2 (Jarillo et al., 2001; Kagawa et al., 2001). However, in low light, both phot1 and phot2 appear to contribute equally to the accumulation of chloroplasts along the upper surface of the leaf to maximize photosynthetic light capture (Wada et al., 2003).

PHOT1 and PHOT2, being duplicate genes, encode proteins that are strikingly similar in their overall sequence and structure (Christie, 2007). Structurally, the phototropins consist of two major parts: (i) an amino-terminal photosensory domain, and (ii) a carboxyl-terminal Ser/Thr protein kinase signalling domain (Fig. 2). Both portions of the protein are necessary for phototropic signalling and much has been learned in recent years about how each portion functions and is regulated, as discussed below.

LOVing blue light: photosensory mechanism of phototropins

The photosensory domain of a phot contains two ~110 amino acid islands with homology to each other that are critical for photoreceptor activity (Christie, 2007): LOV1 (light, oxygen, voltage) and LOV2 (Fig. 2). The LOV domains are members of the larger PAS (Per, Arnt, Sim) domain superfamily (Huala et al., 1997; Crosson et al., 2003). Each of the LOV domains binds a single molecule of blue light-absorbing flavin mononucleotide (FMN) (Christie et al., 1998), imparting photoreceptor function to the phototropins (Christie et al., 1999; Salomon et al., 2000).

As shown in Fig. 3, photosensitive LOV domains undergo a unique photocycle in response to absorption of blue light (Celaya and Liscum, 2005; Christie, 2007; Matsuoka et al., 2007). In darkness, the FMN chromophore is bound non-covalently to the LOV domain as a singlet ground state molecule. This state, which is capable of absorbing blue light, is referred to as LOV\(^D\) (Salomon et al., 2000;
Crosson and Moffat, 2001; Swartz et al., 2001) and absorption of a single photon of blue light results in the generation of an excited singlet FMN, which is rapidly converted into a red light-absorbing (LOV\textsuperscript{L,660}) excited triplet state (T), which is hence converted into the near-UV-absorbing covalent adduct (LOV\textsuperscript{S,390}) that represents the active state. Both the singlet and active LOV\textsuperscript{S,390} states can be converted to the initial dark-state by incubation in darkness. Details of this photocycle are described in the text. Approximate half-times of reactions are given.

It is generally accepted that the LOV\textsuperscript{S,390} FMN-cysteinyl adduct represents the active signalling state of a phototropin (Crosson et al., 2001; Celaya and Liscum, 2005; Christie et al., 2008). Consistent with this model, replacement of the critical cysteine with either serine or alanine eliminates the formation of LOV\textsuperscript{S,390} (Salomon et al., 2000; Swartz et al., 2002; Kottke et al., 2003), and expression of a PHOT1 transgene containing the cysteine to alanine mutation in both LOV1 and LOV2, or LOV2 alone, fails to complement the aphototropic phenotype of a phot1 null mutant (Christie et al., 2002). It is interesting to note that a LOV1 cysteine to alanine single mutant transgene does complement the aphototropic phot1 mutant phenotype, indicating that the two LOV domains are not equal with respect to physiological function (Christie et al., 2002; Sullivan et al., 2008). Similarly, LOV1 is dispensable, whereas LOV2 is sufficient on its own to mediate function of phot2 in the chloroplast avoidance response (Kagawa et al., 2004).

The aforementioned findings raise an obvious question: what is the functional role of the LOV1 domain? Several independent studies (Salomon et al., 2004; Nakasako et al., 2004; Katsura et al., 2008), suggest that LOV1 may function as a dimerization motif; a finding certainly not at odds with the fact that LOV domains are a sub-class within the larger PAS domain superfamily (Crosson et al., 2003). It is also interesting to note that the quantum efficiency for
conversion of LOV$^{D,447}$ to LOV$^S_{8,390}$ is about 10-fold higher in LOV2 than LOV1 in phot1 (Salomon et al., 2000; Kasahara et al., 2002; Iwata et al., 2005), although once photoconverted the LOV1 domain is longer-lived than LOV2 (Kasahara et al., 2002; Iwata et al., 2005). These observations suggest that at least in the case of phot1, the predominant receptor mediating phototropism, the LOV2 domain is considerably more ‘photodynamic’ than LOV1, and that selective pressures in nature are stronger on LOV2 versus LOV1. It remains to be determined why, if it is not functioning to regulate phototropin activity, LOV1 remains photosensitive at all.

Sharing the LOV: protein kinase domain activation

As already mentioned the phototropins contain a Ser/Thr protein kinase domain in their carboxyl-terminal regions (Christie, 2007). While no native substrates for the phot protein kinase domain, other than the phototropins themselves, are currently known (Christie, 2007; Matsuoka et al., 2007), mutational studies have demonstrated that the catalytic activity of this domain is apparently necessary for phototropic signal-output (Christie et al., 2002; Cho et al., 2007). Because of this latter fact much effort has been focused in recent years on understanding how the blue light-dependent formation of LOV$^S_{8,370}$ leads to activation of the protein kinase domain.

While initial X-ray crystallography studies suggested that only minimal changes occur in the tertiary structure of a LOV domain during photocycling (Crosson and Moffat, 2001, 2002; Fedorov et al., 2003), solution spectroscopy provided clear evidence that, in fact, the structural rearrangements associated with the formation of LOV$^S_{390}$ are fairly pronounced, especially if the polypeptides being examined also encompassed regions carboxyl-terminal to LOV2 (Swartz et al., 2002; Corchnoy et al., 2003; Eitoku et al., 2005; Iwata et al., 2005). Nuclear magnetic resonance (NMR) studies by Harper and colleagues (Harper et al., 2003, 2004) identified an alpha-helical region (designated the Jα-helix) that resides between LOV2 and the protein kinase domain, which, in darkness, associates with the solvent-exposed surface of the β-sheet portion of the LOV2 core region facing away from the FMN chromophore. Upon blue light-induced FMN-cysteinyl adduct formation the Jα-helix becomes disordered and dissociates from LOV2 (Harper et al., 2004). A number of mutations were identified that could mimic the aforementioned ‘dissociated state’ in the absence of light exposure, and, when introduced into a full-length phot1, these same mutations resulted in light-independent autophosphorylation of the phot1 protein, suggesting that the LOV2-Jα-helix interaction acts to repress the protein kinase activity of phot1 (Harper et al., 2004). In support of such a LOV2 domain ‘repression model’ (Fig. 4), in vitro studies have shown that an isolated protein kinase domain from phot2 is catalytically active against casein, a common in vitro substrate for protein kinase assays (Matsuoka and Tokutomi, 2005). These results further suggest that phototropins may target proteins other than themselves for phosphorylation in planta, although again no such substrates are currently known.

Moving distances with LOV: intracellular localization of phototropins is dynamic and light regulated

Movements of the phototropins are not limited to the angstrom-level intramolecular movements upon formation of LOV$^S_{390}$, rather the entire phototropin protein appears to move from one part of the cell to another in response to blue light. For example, while phot1 is normally tightly associated with the plasma membrane in dark-grown seedlings, probably through its carboxyl-terminal protein kinase domain (Kong et al., 2006), blue light induces the relatively rapid (within minutes) movement of some proportion of phot1 to intracellular locations (Sakamoto and Briggs, 2002; Wan et al., 2008). Similar relocalization properties have also been observed for phot2; although the
intracellular compartment to which phot2 moves appears to be the Golgi (Kong et al., 2006). At present it is unknown exactly how phototropin movement is linked to a particular physiological response, however, a recent study by Han and colleagues (Han et al., 2008) suggests that this dynamic response may be coupled with receptor adaptation/desensitization or signal attenuation. Specifically the authors found that blue light-induced relocation of phot1 can be largely, if not completely, prevented by prior exposure to red light (Han et al., 2008); light conditions that also lead to phytochrome A-mediated enhancement of phot1-dependent phototropism (Stowe-Evans et al., 2001; Han et al., 2008). Thus it would appear that plasma membrane-localized phot1 is more ‘active’ in terms of phototropic signalling than internalized phot1. Certainly the dynamic nature of phototropin localization represents fertile ground for future studies.

Getting from Darwin’s vision to his ‘influence’: early phototropin signalling components

One of the biggest questions currently facing the community of researchers who study phototropism at the molecular level is: how does phototropin activation lead to auxin-regulated differential growth (curvature)? While the details of this process still remain largely unknown, several components of this ‘black box’ have been identified, most notably three phot1-interacting proteins: NPH3 (NON-PHOTOTROPIC HYPOCOTYL 3; Motchoulski and Liscum, 1999), RPT2 (ROOT PHOTOTROPISM 2; Inada et al., 2004), and PKS1 (PYTOCHROME KINASE SUBSTRATE 1; Lariquet et al., 2006).

NPH3 and RPT2 are paralogous proteins that represent the founding members of the moderately sized NRL (NPH3/RPT2-Like) protein family (33 members in total) in Arabidopsis (Celaya and Liscum, 2005; Celaya et al., 2009). Members of the NRL family, including NPH3 and RPT2, share five regions of primary sequence conservation (Fig. 5): Dla (Domain 1a) and Dib, together comprising an amino-terminal BTB (Broad-Complex/Tramtrack/Bric-à-Brac) domain (Aravind and Koonin, 1999; Stogios et al., 2005); DII, resembling no known structural or functional motif; and the remaining two regions, DIII and DIV, together representing the Pfam ‘NPH3 domain (PF03000)’ of unknown function (Finn et al., 2008). The NRL family also exhibits several regions of conserved predicted secondary structure that have diverged in sequence (Fig. 3); most notably a carboxyl-terminal coiled-coil (Lupas and Gruber, 2005) that is present in approximately half of the family members (Celaya and Liscum, 2005; Pedmale and Liscum, 2007). Although the functional roles of each of the aforementioned ‘domains’ are currently not fully understood, the coiled-coil has been shown to represent the phot1-interaction domain of NPH3 (Motchoulski and Liscum, 1999; Pedmale and Liscum, 2007), while the BTB domain can mediate interaction between NPH3 and RPT2, at least in yeast (Inada et al., 2004). Recent studies have shown that the BTB domain of NPH3 can also mediate interaction with CULLIN 3 (CUL3) (Pedmale and Liscum, 2007). These latter results suggest that NPH3 may represent the substrate adapter component of a CUL3-based E3 ubiquitin ligase, a recently recognized role for many BTB-containing proteins (Krek, 2003; Pintard et al., 2004; van den Heuvel, 2004; Willems et al., 2004; Stogios et al., 2005; Perez-Torrado et al., 2006).

While no target for ubiquitination by an NPH3-CUL3 complex has yet been reported, one can imagine that such a target might function in the regulation of auxin transport. Findings that phototropic stimulation fails to induce an asymmetric distribution of auxin across the coleoptile in the rice mutant cpt1 (coleoptile phototropism 1) (Haga et al., 2005) is consistent with this hypothesis. CPT1 encodes the rice orthologue of Arabidopsis NPH3 (Haga et al., 2005), thus placing NPH3/CPT1 downstream of phot1 and upstream of the regulation of auxin redistribution. Recent studies suggest that regulation of auxin transport may represent a common function of NRL family members. For example, mutations in the NPY/ENP/MAB4 (NAKED PINS IN YUC MUTANTS/ENHANCER OF PINOID/MACCHIBOU 4) subfamily of the NRL superfamily appear to influence auxin-mediated organogenesis through alterations in auxin movement (Cheng et al., 2007; Furutani et al., 2007), probably through genetic interactions between the NPY proteins and the AGC kinases PID (PINOID), PID2, WAG1 (denotes the ‘wagging’ root growth it mediates), and WAG2 (Cheng et al., 2008). This latter observation is particularly intriguing as phot1 is also an AGC kinase (Bögre et al., 2003; Galván-Ampudia and Offringa, 2007).

The PKS1 protein was originally identified as a negative regulator of phytochrome signalling and to serve as a substrate for phytochrome’s protein kinase activity (Fankhauser et al., 1999), but has since been shown to function as a positive regulator of phototropism as well and physically to interact with both phot1 and NPH3 (Lariquet et al., 2006). At present, it is not understood how PKS1 (or PKS2 and PKS4; Lariquet et al., 2006) influences phototropism at a molecular level, but it is tempting to speculate that the PKS proteins may bridge the enhancing influences of phytochrome on phot1-dependent phototropism (Liscum and Briggs, 1996; Parks et al., 1996; Janoudi et al., 1997; Stowe-Evans et al., 2001; Liscum, 2002), possibly through
influences on phot1 localization (Han et al., 2008). It is also worth noting that NPH3, like PKS1 (Fankhauser et al., 1999), is a phosphoprotein whose phosphorylation state and functional activity is light-dependent; whereas red light stimulates the phosphorylation of PKS1 in a phytochrome-dependent fashion (Fankhauser et al., 1999), blue light results in the dephosphorylation of NPH3 that is dependent upon the presence of phot1 (Pedmale and Liscum, 2007). Thus it would appear that the signalling capacity of both NPH3 and PKS1 are regulated by similar post-translational mechanisms linked to the photoreceptors through which the former molecules signal.

Power of movement meets origin: phototropism in the field

Though Darwin (1880) hypothesized that phototropic responses are adaptive to a plant, and this proposal has been reiterated many times over the past 100 plus years in one form or another (Iino, 1990; Liscum and Stowe-Evans, 2000; Christie, 2007), it has only been within the last few years that this hypothesis has actually been experimentally tested. Galen et al. (2004) have shown that the fitness of field-grown Arabidopsis plants carrying loss-of-function mutations in PHOT1 are significantly lower than that of wild-type plants grown in the same plots. Somewhat surprisingly, in contrast to previous proposals that stem phototropism would represent the adaptive response in nature (Iino, 1990), this study found that root phototropism was the trait coupled to fitness, and only under high light conditions (Galen et al., 2004). A subsequent study demonstrated that negative root phototropism (bending away from directional blue light) enhances the ability of the plant to access water, which under high light conditions is more abundant deeper in the soil because of increased evaporation near the surface (Galen et al., 2007a). Three life history traits in particular were shown to be influenced dramatically by the ability of a root to access water in arid conditions: (i) seedling establishment, (ii) accumulation of biomass in established plants, and (iii) fecundity of plants reaching adulthood (Galen et al., 2007a). These studies provide an exciting potential avenue to develop plants capable of growing in more arid environment that maintain, or even increase, their production value through genetic engineering of phot1 signalling (Galen et al., 2007b).

From Darwin to the future: final thoughts

Darwin’s ‘The power of movements in plants’ undoubtedly stimulated an entire field of study on plant responses to the environment. Since publication of this seminal work our understanding of phototropism in higher plants has expanded tremendously. Several significant findings have been made: identification and characterization of the photoreceptors controlling phototropism; identification of auxin as the major growth regulator involved in the development of phototropic responses, and elucidation of its mechanistic basis of action; identification of several signalling components functioning between photoperception and auxin responsiveness; and characterization of an adaptive significance for phototropism under natural growth conditions. However, the goal remains fully to elucidate all of the molecular components, from the reception of light to movement, that contribute to the phototropic response, as well as the ecological variables that have provided the selective pressures for the evolution of this response in nature. In all these regards, there is much work left to do. The next century, like the last, is likely to bring many answers to such questions, leading to an even greater appreciation of just how important ‘The power of movements in plants’ really is!

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