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Phytochrome, Carbon Sensing, Metabolism, and Plant Growth Plasticity

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Phytochrome, Carbon Sensing, Metabolism, and Plant Growth Plasticity[1][CC-BY]

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Plants continuously monitor fluctuations in their environment and actively adjust their metabolism to cope with variations in light and carbon resource availability. However, the links between photoreceptor signaling pathways and central metabolism are poorly understood. Emerging evidence suggests that phytochrome photoreceptor signaling and carbon resource management are strongly coupled. In this review, we outline the current understanding of how phytochrome-dependent light signaling interfaces with metabolism and carbon resource management.

The ability to sense and react to the light environment enables plants to adapt to and thrive in a changing environment. Post germination, seedlings adopt either a skotomorphogenic or photomorphogenic developmental program, depending on whether light is available. The skotomorphogenic strategy is adopted by dark-grown seedlings, which exhibit elongated hypocotyls and closed cotyledons that are folded against the hypocotyl in a so-called apical hook. This growth program relies on seed reserves to seek light through rapid hypocotyl extension. By contrast, when exposed to light, seedlings undergo photomorphogenic growth, which typically prevents hypocotyl elongation and, instead, promotes cotyledon expansion and greening, processes that enable seedlings to begin photoautotrophic growth. To support photomorphogenic development, plants have evolved multiple families of photoreceptors that capture a wide range of the light spectrum. These include UVB-RESISTANCE8, which detects UV-B light, cryptochromes (crys), phototropins, and the ZEITLUPE/FLAVIN-BINDING, KELCH REPEAT, F BOX 1/LOV KELCH PROTEIN2 family of photoreceptors, which absorb UV-A and blue light, and the phytochromes (phys), which sense red (R) and far-red (FR) light (Galvão and Fankhauser, 2015). This review will focus on the phy photoreceptors, whose unique photosensory properties can profoundly influence plant growth and development.

The phys are a multigene family; for instance, the Arabidopsis (*Arabidopsis thaliana*) genome encodes five *PHY* genes, designated *PHYA* to *PHYE*. These photoreceptors are synthesized in the cytosol in their inactive Pr form. R light exposure drives Pr photoconversion to the biologically active Pfr form (Li et al., 2011). Pfr is then translocated into the nucleus, where it negatively regulates transcription through direct binding to PHYTOCHROME INTERACTING FACTORS (PIFs), basic helix-loop-helix (bHLH) transcription factors that are negative regulators of photomorphogenesis. Phy interaction with PIFs has the dual effect of sequestering PIFs from their cognate promoters and promoting PIF phosphorylation and proteolysis (Leivar and Quail, 2011; Park et al., 2012). In parallel, phys indirectly suppress the COP1 E3 ubiquitin ligase/SPA complex (Sheerin et al., 2015), which mediates the turnover of PHYA and PHYB and positive regulators of photomorphogenesis, such as HY5, HYH, LAF1, and LONG HYPOCOTYL IN FAR RED (HFR1; Wang and Wang, 2015). In darkness, these transcription factors are targets of 26S proteasome-mediated degradation by the COP1 E3 ligase component (Lau and Deng, 2012).

As light exposure elicits distinct and quantifiable growth and molecular changes post germination, the seedling system has proved to be invaluable in delineating the photoreceptor roles and signaling events.

www.plantphysiol.org/cgi/doi/10.1104/pp.17.01437

ADVANCES

- Phytochrome depletion targets a wide range of photosynthetic processes and transcriptionally regulates various photosynthetic components, such as chlorophyll and carotenoid biosynthetic genes.
- Phytochrome signaling affects the abundance of a wide range of primary metabolites, such as organic acids, amino acids, sugars, and starch.
- Photoreceptor-related signaling components help transduce metabolic signals.

Such experiments have established that phys are important sensors of irradiance quality and quantity in early development (Strasser et al., 2010; Hu et al., 2013). However, phys also operate postseedling establishment, ensuring that plant development continues to react to local and seasonal changes. Many of these light-driven responses elicit alterations in growth and architecture that may necessitate concomitant changes in carbon resource distribution and management. This review highlights studies that are beginning to uncover connections between phys and central metabolism. The new research is revealing that phy action is not confined to molecular signaling but also strongly impacts metabolism, while the plant's carbon status is relayed back to the phy pathway. This integrated system enables plants to simultaneously adjust growth, resources, and metabolism to a changing environment.

PHYTOCHROMES, MEDIATORS OF PLANT GROWTH PLASTICITY

The general body plan of plants is genetically encoded, but plant architecture can be modified to adjust to the environment that surrounds it. In this sense, external cues, such as light, have a profound effect on the way a plant grows and develops, ultimately affecting a plant's fitness, disease resistance, and productivity (Li et al., 2012). Phys are able to modulate plant plasticity because of their exquisite sensitivity to both fluence rate (light intensity) and light quality (spectral composition). The competing reactions of rapid light-induced photoreceptor activation to Pfr and slower inactivating thermal relaxation to Pr deliver a graded response to fluence rate (Rausenberger et al., 2010; Johansson et al., 2014; Jung et al., 2016; Legris et al., 2016).

This characteristic allows seedlings to calibrate deetiolation and subsequent vegetative growth with the available light levels. Sensitivity to spectral composition arises from the unique photochemical properties that allow phys to detect small changes in the R-FR ratio caused by nearby plants. The neighbor-sensing system allows plants to reprogram their growth and metabolism such that they can cope better with potential shading and competition for resources. This suite of changes in plant physiology and development is collectively referred to as the shade avoidance response (SAR). While several phys contribute to SAR, phyB plays a particularly prominent role. Indeed, *phyB* mutants display classical SAR phenotypic traits, but additional mutations increase the severity of the phenotype (Franklin and Whitelam, 2005; Martínez-García et al., 2010; Leivar and Quail, 2011; Casal, 2012). In Arabidopsis, SAR characteristics include perturbed seedling deetiolation (the switch to photomorphogenic growth), altered leaf architecture, typified by elongated petioles and small leaf blades, reduced biomass, and early flowering (Li et al., 2012; Galvão and Fankhauser, 2015; Yang et al., 2016; Fig. 1A). Phys, therefore, provide a versatile sensory system that can detect intrusive

vegetation, shading, or persistent cloud cover and elicit adaptive changes (Galvão and Fankhauser, 2015).

A series of studies have shown that PIFs operate antagonistically to phys, as positive regulators of the SAR response. Although seven members of the PIF family can interact with PHYs, only PIF4, PIF5, and PIF7 have unambiguously been shown to mediate this response, with PIF1 and PIF3 playing minor roles (Leivar and Quail, 2011; Casal, 2012; Leivar and Monte, 2014; de Wit et al., 2015, 2016). As PIFs are phosphorylated by phyB Pfr and targeted for degradation, under low R-FR ratio conditions that switch phyB Pfr to its inactive Pr form, PIF4 and PIF5 proteins accumulate (Lorrain et al., 2008). Interestingly, while phyB also induces PIF7 phosphorylation, this does not trigger PIF7 degradation; rather, it inactivates it, blocking the transcriptional regulation of target genes. A low R-FR ratio, therefore, strongly promotes PIF action by boosting PIF4 and PIF5 levels and by allowing for PIF7 action (Li et al., 2012).

PIF4, PIF5, and PIF7 are known to mediate SAR by directly targeting genes involved in auxin biosynthesis and other hormone signaling pathways, including GA, brassinosteroid (BR), jasmonate, and ethylene (Leivar and Monte, 2014).

The SAR is negatively regulated by the atypical bHLH transcription factors HFR1 and PHY RAPIDLY REGULATED1 (PAR1) and PAR2 (Galstyan et al., 2011). These proteins suppress PIF action by binding to their DNA-binding domain and affecting their biological activity (Hao et al., 2012; Zhou et al., 2014). Interestingly, PIFs induce the expression of these genes, which suggests that HFR1 and PARs operate in a negative feedback loop that may be important to moderate the SAR response. These studies have shown that the phy-PIF regulatory mechanism provides a means to directly couple light sensing with transcriptional regulation and growth. This system is a central driver of plastic growth responses enabling plants to adapt to changeable light conditions.

PHYTOCHROME CONTROL OF PHOTOSYNTHETIC CAPACITY

A very prominent feature of seedling establishment conditions is the development of chloroplasts and their preparation for photosynthetic function. This section will review the expanding body of evidence that links phy to photosynthetic competence.

Several studies have shown that phys are important regulators of photosynthetic pigment abundance. R light treatment of wild-type seedlings has been shown to induce the formation of chlorophyll within hours (Ghassemian et al., 2006), while sequential phy depletion in R-grown *phyB*, *phyABDE*, and *phyABCDE* mutant seedlings leads to concomitant reductions in chlorophyll levels (Hu et al., 2013). In older plants, severe phy deficiency or a low R-FR ratio also lowers chlorophyll levels per biomass unit, but not to the same

A

Ler phyB phyABD

≤ 0.6 (5th percentile) 1 (no change) ≥ 4.6 (95th percentile)

B

Publication	Jumtee et al 2008		Jumtee 2009		Yang et al 2016		Han et al 2017	Fukushima et al 2009							Publication
Species	*Arabidopsis*		rice		*Arabidopsis*		*Arabidopsis*	*Arabidopsis*							Species
Experiment	Etiolated seedlings FR or W treated at 0h, sampling after 24h. (*phyA*/D) / (WT/D)		Young and mature leaf		EOD and EON		EOD and EON, different lights	Time course in LD							Experiment
Genotypes	*phyA* vs WT		*phyABC* vs WT		*phyABDE* and *phyABCDE* vs WT		*phyAB* vs WT	*prr975* vs WT							Genotypes
Plant age	4 days		25 to 30 days		35 days		20 days	18 to 19 days							Plant age
Light regime	etiolated, then FR or W		LD 14.5:9.5		2 weeks LD8:16, 3 weeks LD12:12		LD16:8	LD12:12							Light regime
Temperature	21°C		28°C (day) 23°C (night)		18°C		20°C (day), 16°C (night)	22°C							Temperature
Samples	FR	W	YL	ML	EON	EOD	*phyAB*	7	9	11	13	15	17	19	Samples
Glucose	+		+	+		+			−	−	−	−	−	−	Glucose
Fructose	+		+	+				−	−	−	−	−	−	−	Fructose
Galactose	+		+	+			−	na	na	na	na	na	na	na	Galactose
Sucrose	+					+									Sucrose
Maltose	na	na	−	−				na	na	na	na	na	na	na	Maltose
Trehalose	na	na		−	+									−	Trehalose
Raffinose	na	na	+	+	+	+		+	+	+	+	+	+	+	Raffinose
Succinate			−	−	+	+	−	+	+	+	+	+	+	+	Succinate
Fumarate			+		+	+		+	+	+	+	+	+	+	Fumarate
Malate			+	+	+	+	na	+	+	+	+	+	+	+	Malate
Citrate			+		+	+	−	+	+	+	+	+	+	+	Citrate
2-Oxo-glutarate	na	na	+	−	na	na	na	+	+	+	+	+	+	−	2-Oxo-glutarate
Alanine			+	−	−				+		−	+	−		Alanine
Valine	+		+	−	+	+	−				+	+	+		Valine
Leucine	+		+		+		−								Leucine
Isoleucine	+		+	−	na	na	−	+	+	+	+		+		Isoleucine
Proline	+		+		+	+		+	+	+	+	+	+	+	Proline
Glycine	+		+		na			+	+	+		+	+		Glycine
Serine	+										−		+		Serine
Threonine	+		+	−		−	−				−	+		−	Threonine
Aspartate			−	−	+	+	−				+	+	+		Aspartate
Methionine	+	+	+		na	na		+	+		+		+		Methionine
Glutamate			+		+	+		+	+	+	+	+	+	+	Glutamate
Phenylalanine	+	+	+	−	+	+			−	−	−	+	+	+	Phenylalanine
Lysine	+		+	−	na	na	−	−	−						Lysine
Tyrosine	+		+		na	na									Tyrosine
Tryptophan			na	na	na	na		−	−	−			+		Tryptophan
Asparagine			+					+		+	+		+		Asparagine
Glutamine	na	na			+	+	na	+	+	+	+	+	+	+	Glutamine
Homoserine	na	na	na	na	na	na	−		+		+	−	+		Homoserine
Arginine	na	na	na	na	na	na		−	−	−	−		+		Arginine
Putrescine	+		na	na	na	na						+	+		Putrescine
Ornithine			na	na	na	na									Ornithine
Glycerate			−		na			+	+	+	+	+			Glycerate
Pyruvate			+		na	na									Pyruvate
Lactate			−	−			na		−						Lactate
Shikimate	na	na	−	−	na	na	na	+	+	+	+	+	+	+	Shikimate
Ascorbate	na	na	na	na	na	na		+	+	+	+	+	+	+	Ascorbate
Nicotinate	na	na	na	na	na	na		+	+	+	+	+	+	+	Nicotinate

Figure 1. Phytochrome affects biomass, plant architecture, and carbon metabolism. A, Images of Arabidopsis Landsberg *erecta* wild type (Ler) and the phytochrome mutants *phyB* and *phyABD*. B, Lack of phytochrome signaling leads to enhanced levels of several organic acids, sugars, and amino acids in most studies. The heat map visualizes fold changes in soluble metabolite content in phytochrome mutants and the *prr975* mutant compared with wild-type (WT) controls, or in response to low R-FR ratio, as seen in gas chromatography-mass spectrometry (GC-MS) data sets (Jumtee et al., 2008, 2009; Fukushima et al., 2009; Yang et al., 2016;

extent as in seedlings (Hu et al., 2013; Patel et al., 2013; Yang et al., 2016). In tomato (*Solanum lycopersicum*), phy effects on chlorophyll levels also are quite evident in adult plants (Kharshiing and Sinha, 2016).

Over the years, a solid body of work has strongly implicated phys in the transcriptional regulation of photosynthesis-related genes. *CAB* and *RBCS* were among the first known phy-regulated genes (Silverthorne and Tobin, 1984; Mösinger et al., 1985; Nagy et al., 1986; Otto et al., 1988; Dean et al., 1989; Wehmeyer et al., 1990; Thompson and White, 1991). These early studies implicated phy in the rapid accumulation of *CAB* and *RBCS* mRNA following exposure to R light. Later research provided genetic evidence for phy control of the chlorophyll biosynthesis gene *HEMA1* and the light-harvesting complex component *LHCB2* (McCormac and Terry, 2002). This role was confirmed by microarray studies illustrating that R light treatment of etiolated seedlings led to broad changes in the expression of genes involved in photosynthesis or chloroplast development (Leivar et al., 2009). Moreover, in darkness, about 60% of these genes also are significantly up-regulated in *pifQ* mutants, which lack PIF1, PIF3, PIF4, and PIF5. This indicates that, in the dark, PIFs have an important role in suppressing photosynthetic gene expression (Leivar et al., 2009).

In addition to these observations, the molecular mechanisms leading to the reduction in chlorophyll levels have been investigated. Two reports (Toledo-Ortiz et al., 2010, 2014) showed that PIFs and the bZip transcription factor HY5 antagonistically regulate chlorophyll and carotenoid biosynthesis. Carotenoids assist photosynthesis by acting as auxiliary antennae for light absorption. These studies demonstrated that both PIFs and HY5 can bind to and potentially compete for G-boxes on the promoters of carotenoid and chlorophyll biosynthetic genes, such as *PHYTOENE SYNTHASE, VIOLAXANTHIN DEEPOXIDASE* or *PROTOCHLOROPHYLLIDE OXIDOREDUCATASE C, GENOMES UNCOUPLED5 (GUN5),* and *LIGHT-HARVESTING CHLOROPHYLL-PROTEIN COMPLEX I SUBUNIT A4*. Pigment and transcript levels for these genes are accordingly low in the *hy5* mutant and elevated in the quadruple *pifQ* mutant. As phys negatively regulate PIFs but promote HY5 action, this dual-control transcriptional mechanism conveys exquisite light control of photosynthesis-related genes.

Interestingly, other studies have shown that chloroplast status can feedback to influence the photomorphogenic pathways through chloroplast retrograde signaling (for review, see Chan et al., 2016). Earlier studies hinted that this might be the case (McCormac and Terry, 2004; Nott et al., 2006). A recent study has shown that the *GOLDEN2-LIKE1 (GLK1)* transcription factor plays a critical role in this process in seedlings (Martín et al., 2016). In darkness, PIFs repress *GLK1* expression, while light inactivates this process, allowing gene regulation and photomorphogenic development to proceed. At high light intensities that may be damaging for the chloroplast, *GLK1* is repressed, this time through *GUN1*-mediated retrograde signaling from the chloroplast. This signal halts photomorphogenic gene expression and development, which serve to protect the seedlings from high-light damage.

In summary, there is ample evidence for transcriptional regulation of photosynthetic genes by phytochromes, especially genes involved in photosynthetic pigment synthesis, and we are now also beginning to understand the role of phytochrome in retrograde signaling and the interplay between both signaling systems.

FINE-TUNING OF PHOTOSYNTHETIC PROCESSES BY PHYTOCHROME

Molecular studies have hardwired the links between phy and photosynthetic gene expression, but photophysiological analysis is required to understand the advantages and disadvantages of altered photosynthetic capacity in phy-dependent adaptive responses (e.g. SAR). A recent study analyzed the contribution of phys and crys to the regulation of proteins involved in chloroplast metabolism and the Calvin cycle, including Rubisco and Rubisco activase (Fox et al., 2015). First, the quadruple *phyA;phyB;cry1;cry2* mutant was shown to have reduced chlorophyll levels, harvesting complex, and Calvin cycle proteins. Then, reduced maximum CO_2 fixation in this quadruple mutant confirmed the impaired activity of the Calvin cycle and electron transport components. These data suggest that phy and cry signaling may not affect simply photosynthetic capacity but the efficiency of the Calvin cycle. Notably, this study also showed that, despite the deficiencies of *phyA;phyB;cry1;cry2* mutants, these were not limiting

Figure 1. (*Continued.*)
Han et al., 2017). Fold changes were calculated by dividing the metabolite content of the mutant (or low R-FR ratio-treated plants) by that of the wild type (or high R-FR-treated plants). For Jumtee et al. (2008), values at 24 h after the start of FR or white light treatment were first normalized by the respective dark control of each genotype at the same time point, and then fold change of *phyA* over the wild type was determined from these values. Experimental setup, samples, and conditions for each study are indicated above the heat map. Fold changes are indicated in colors, with dark blue representing the largest decrease and dark orange representing the largest increase in the mutant over the wild type, or low R-FR over high R-FR ratio. The values of the 5th and 95th percentiles were used as minimum and maximum values, respectively. + and − indicate statistically significant increase and decrease, respectively, according to the statistics employed in each study. EOD, End of day; EON, end of night; LD, light-dark; D, dark; YL, young leaf; ML, mature leaf; na, data are not available for this metabolite. Numbers for Fukushima et al. (2009) indicate time of sampling after light onset in the morning in hours.

for photosynthesis at low light levels but were very restrictive at high fluence rates. Other studies observed reductions in CO_2 uptake in *phyB* and multiallele *phy* mutants at medium and high light levels (Boccalandro et al., 2009; Yang et al., 2016). Conversely, cotton (*Gossypium hirsutum*) plants that overexpress *PHYB* were shown to have higher photosynthetic rates per unit of leaf area and higher biomass (Rao et al., 2011). These data collectively indicate that phy action is important for regulating carbon fixation and biomass production, particularly at high fluence rates. They also suggest that, even though the photosynthetic machinery may be impaired by phy deactivation, it is not necessarily limiting for photosynthesis in low-light environments.

An additional observation was made by Boccalandro et al. (2009), who showed that phyB also increases stomatal density, which is postulated to enhance photosynthetic rate at the expense of water use efficiency. Leaf thickness is another phy-controlled architectural trait that may affect photosynthetic performance. Thiele et al. (1999) showed that potato (*Solanum tuberosum*) plants overexpressing *PHYB* achieve higher photosynthetic rates per plant and per leaf area but have similar rates to wild-type plants when normalized to chlorophyll content. The authors suggested that the thicker palisade tissue and the resulting higher chlorophyll content per unit of leaf area allowed the plants to reach a higher overall photosynthetic rate. Thinner leaves are part of the shade avoidance syndrome (McLaren and Smith, 1978; Franklin and Whitelam, 2005), which necessarily entails a larger leaf area-to-biomass ratio. Therefore, carbon uptake should be normalized to the existing biomass to determine its contribution to growth, and this information is lacking in most studies.

Taken together, these mechanistic and physiological studies are uncovering an important role of phys in managing photosynthetic capacity. In SAR conditions that can be light limiting, reduced investment in the photosynthetic machinery does not appear to impair CO_2 uptake and may relieve the demand on energy reserves, liberating resources for other processes.

PHYS HAVE WIDE-RANGING EFFECTS ON CORE METABOLITES

Following assimilation by photosynthesis, the newly acquired carbon is distributed into different metabolic pathways to either provide energy for maintenance and growth or to biosynthesize metabolites for other components, such as amino acids for protein synthesis. Evidence is emerging that phy signaling has sizable effects on the majority of such primary carbon metabolic pathways and a subset of secondary metabolites. Studies published over the last 11 years reported metabolomics experiments comparing phy mutants with wild-type controls in Arabidopsis and rice (*Oryza sativa*; Ghassemian et al., 2006; Jumtee et al., 2008, 2009; Patel et al., 2013; Yang et al., 2016; Han et al., 2017). One of these studies analyzed metabolic changes during the

first 24 h of FR- or white-light induced deetiolation of dark-germinated wild-type and *phyA* seedlings (Jumtee et al., 2008), while Ghassemian et al. (2006) investigated changes due to deetiolation in R light. The other reports (Patel et al., 2013; Yang et al., 2016; Han et al., 2017) focused on metabolic changes in low R-FR ratio or *phy* single and multiallele mutant whole rosettes (more than 2.5 weeks old), while the rice study distinguished between young and mature leaves (Jumtee et al., 2009). Although the results differ, as expected for different experiments, species, and conditions, the general trend indicates that a large number of metabolites, especially sugars and tricarboxylic acid cycle components, accumulate to higher levels in *phy* mutants compared with wild-type plants (for cross-comparison of most of these studies, see Fig. 1B). Interestingly, a large subset of these changes are observed in the *prr975* mutant (Fig. 1B) that lacks the circadian clock genes PSEUDO RESPONSE REGULATOR9 (PRR9), PRR7, and PRR5 (Fukushima et al., 2009). This may not be surprising as, like the *phyB* mutant, *prr975* seedlings have a similar elongated hypocotyl phenotype in R light, suggesting that PRRs may be positive regulators of the phyB pathway (Kato et al., 2007). Indeed, epistasis analysis positions *PRR7* and *PRR5* downstream of phytochrome in this response (Ito et al., 2007). Furthermore, as TOC1 was shown to bind to and repress the activity of PIF3 and PIF4, it is possible that other PRRs suppress PIF signaling in a similar manner (Soy et al., 2016; Zhu et al., 2016). Because of the strong connections between phy signaling and PRRs, we have included the metabolic changes in the *prr975* mutant in this review (Fukushima et al., 2009). The following section will review phy control of sugars and starch, followed by tricarboxylic acid cycle intermediates and amino acids.

Starch and Sugars

Suc is the major sugar used to transport excess carbon from source leaves to sink tissues (Kölling et al., 2013), where it is broken down into Glc and Fru, which are then used by different pathways including respiration. Starch is synthesized during the day and is used as a carbon resource during the night to sustain maintenance and growth in the absence of light (Zeeman et al., 2010).

Jumtee et al. (2008) reported that exposure of etiolated seedlings to FR light led to a phyA-dependent fall in the levels of sugars (including Suc, Glc, Fru, and Gal). Interestingly, white light treatment appeared to be less effective in depleting sugars. As FR light activates deetiolation, but not the greening of cotyledons, under these conditions, the carbon resources may be used for growth but not replenished through photosynthesis.

An intriguing finding is that, even though phy depletion tends to impair photosynthesis, particularly at higher light levels (see above), Arabidopsis phyBD and phyABDE mutants sampled at day 35 overaccumulate daytime sugars and starch (Yang et al., 2016). The rice phyABC triple mutant also has higher daytime sugar

levels compared with the wild type, and this is particularly evident in younger triple mutant leaves that contain excessive levels of the reducing sugars Glc, Fru, and Gal. Like the Arabidopsis phyBD and phyABDE mutants, the rice phyABC mutant has altered starch levels compared with the wild type. However, in rice phyABC, starch is more abundant at night, indicating incomplete usage during the dark period.

Two studies (Fukushima et al., 2009; Han et al., 2017) that sampled around 18 to 20 d (as opposed to 35 d in Yang et al., 2016) reported reduced sugar levels in prr975, phyA, phyB, and phyAB when compared with the wild type. Reflecting this, the phy mutants in the study by Han et al. (2017) also have lower starch. This is interesting, as it illustrates that the impact of phy and PRR signaling on sugar and starch abundance may be dependent on the developmental stage and/or experimental conditions. In fact, Patel et al. (2013) show that rosettes accumulate significantly more soluble sugars (especially Suc) at low R-FR ratios compared with high R-FR ratios at 16°C but not at 22°C. This is a possible explanation for the different results reported by Han et al. (2017; 20°C daytime) and Yang et al. (2016; 18°C), in addition to age, photoperiod, choice of mutant, and light intensity. A consistent finding across studies is that phy deficiency alters sugar levels. Whether this leads to a rise or fall in sugars will potentially depend on the collective effects of phy on carbon uptake and resource use in different conditions or phases of development. More in-depth metabolic flux analysis will be required to decipher the regulatory processes that underlie the sometimes dramatic phy-controlled changes in sugar metabolite levels. Transcriptome studies showed that ~30% of R-induced genes are involved in cellular metabolism (Leivar et al., 2009); therefore, it appears that metabolic changes may result at least in part from transcriptional regulation.

Tricarboxylic Acid Cycle Components and Amino Acids

Photosynthetic carbon is used either for growth and biosynthetic processes or to create ATP, involving glycolysis and the tricarboxylic acid cycle. The intermediates of these processes are used not only in respiration but also in the biosynthesis of other metabolites, such as amino acids (for review, see Fernie et al., 2004). It appears that the majority of tricarboxylic acid cycle organic acids and amino acids are regulated by phy. During FR-induced deetiolation, amino acid concentrations drop in wild-type but not in phyA seedlings (Liu et al., 2012). The authors hypothesize that this phyA-mediated effect may arise from an increase in protein synthesis to support growth, which would deplete the amino acid pool. Likewise, the abundance of amino acids drops in response to deetiolation in constant red light within only a few hours (Ghassemian et al., 2006). Han et al. (2017) also record reductions in amino acid and tricarboxylic acid cycle intermediates in phyA, phyB, and phyAB plants long after deetiolation.

However, other studies report elevated levels of amino acids and tricarboxylic acid cycle components in adult multiallele phy mutants, prr975, and young phyABC rice leaves (Fukushima et al., 2009; Jumtee et al., 2009; Yang et al., 2016). Among the metabolites measured by Patel et al. (2013), Gly exhibited the largest increase under low R-FR ratio compared with high R-FR ratio at 16°C, but no change was seen at 22°C. Therefore, as for sugars, temperature (but also photoperiod, age, choice of mutants, and light intensity) provides a possible explanation for the differences seen by Yang et al. (2016) and Han et al. (2017).

In cases where organic and amino acids accumulate, it is currently unclear whether this is because of increased production or slower consumption. Transcript profiles of enzymes catalyzing the synthesis of fumarate and citrate reveal that these genes are down-regulated in phyABDE mutants (Yang et al., 2016), suggesting that these metabolites are not elevated through transcriptional up-regulation. An alternative hypothesis is that tricarboxylic acid cycle intermediates accumulate due to decreased synthetic processes that use these metabolites. For example, reduced throughput to chlorophyll (which can be low in phy mutants) would increase the pool of chlorophyll biosynthesis precursors, including Glu (Tanaka and Tanaka, 2007). Another possibility is that high levels of amino acids may arise from reduced rates of protein synthesis. The challenge ahead will be to establish whether the metabolic changes observed in phy-deficient plants are an accidental consequence of misregulated growth or whether they are adaptive for light-limiting or FR-rich canopy shade conditions. The apparent conditionality of the phy-dependent metabolic profile suggests that phys may have an important role in ensuring that the metabolic response is aligned with the plant growth strategy (Jumtee et al., 2008, 2009; Yang et al., 2016; Han et al., 2017).

PHY CONTROL OF GROWTH AND BIOMASS

Another open question is whether and how the changes in cellular metabolic processes by phy depletion are linked to biomass accumulation. There is evidence that phy can have a positive effect on biomass accumulation in some conditions. For example, phyA tomato mutants, as well as 5-week-old Arabidopsis phyBD and phyABDE mutants, have substantially less biomass than the respective wild-type controls (Kharshiing and Sinha, 2016; Yang et al., 2016). As discussed above, reduction in photosynthetic rates often is accompanied with a reduction in biomass, and this would be a plausible mechanism leading to a reduction in biomass in phy mutants. Alternatively, inefficient metabolism in phy-deficient plants could contribute to their decreased biomass. To resolve these questions, more quantitative data are needed in order to estimate the contribution of the photosynthetic machinery or metabolic misregulation to biomass accumulation.

CARBON SENSING BY PHY SIGNALING COMPONENTS

Recent studies have shown that, as well as adjusting metabolism, phytochrome signaling responds to endogenous carbon status. In fact, if phytochrome signaling is involved in sensing carbon availability, this opens the question of whether the changes in metabolism and growth in phytochrome mutants and *prr975* arise at least in part from altered carbon reserve sensing. Several studies have delineated close links between central light signaling components, including HY5 and PIFs, and carbon-activated signaling.

HY5 has emerged as a key phytochrome signaling component that links light signal transduction to carbon resource management. Earlier chromatinimmunoprecipitation (ChIP)-chip analysis identified more than 3,500 direct HY5 target genes with a significant enrichment in metabolic, nutrient signaling, and photosynthetic genes (Lee et al., 2007). HY5 was subsequently shown to regulate the expression of chlorophyll biosynthesis and photosynthesis-related genes through direct binding to G-box promoter elements (Toledo-Ortiz et al., 2014). More recently, HY5 was shown to directly enhance the expression of TREHALOSE-6-PHOSPHATE SYNTHASE1 (TPS1) and the Suc efflux transporters SWEET11 and SWEET12. TPS1 elevates levels of trehalose-6-phosphate, a metabolic signaling molecule that controls growth, flowering, and shoot-to-root transport of Suc (Chen et al., 2016). However, HY5 action is not confined to the shoot; it translocates to the root and induces the expression of root-located *HY5*, which, in turn, activates *NRT2.1* transcription and root nitrate uptake. Furthermore, the HY5-induced *NRT2.1* expression in the root appears to be dependent on the shoot metabolic carbon status. Thus, HY5 appears to play a pivotal role in coordinating carbon uptake and growth in the shoot with nitrogen uptake in roots (Chen et al., 2016). Interestingly, COP1-mediated proteolysis of PHYA was shown to be impaired by Suc application, indicating that COP1 could be Suc regulated (Debrieux et al., 2013). It will be interesting to establish if other COP1 targets such as HY5 are regulated by internal carbon status.

The PIF transcription factors were recently implicated in sugar signaling. Suc-induced hypocotyl elongation appears to be PIF dependent, and this response is abolished in the *pifQ* mutant (Stewart et al., 2011). Although Suc only moderately alters *PIF* transcription, PIF5 protein was shown to accumulate in response to Suc (Stewart et al., 2011). A different study by Shor et al. (2017) did not observe Suc effects on the protein stability of PIF1, PIF3, PIF4, and PIF5 but demonstrated through ChIP-quantitative PCR (qPCR) that Suc enhances PIF enrichment at the promoters of the clock genes *LHY* and *CCA1*. This Suc-dependent regulation appears to enhance the peak of *LHY* and *CCA1* expression at dawn. The authors propose that this mechanism may allow PIFs to participate in Suc entrainment of the oscillator. A number of earlier studies

demonstrated that PIFs target promoter elements of multiple auxin biosynthetic and signal transduction genes (Franklin et al., 2011; Nozue et al., 2011; Hornitschek et al., 2012). PIFs also have been shown to be required for the Suc regulation of several of these auxin-related rates (Lilley et al., 2012; Sairanen et al., 2012).

The PIF-interacting proteins DELLA and BZR1, master regulators in the GA and BR pathways, respectively, also have been implicated in sugar responses. DELLAs are potent growth suppressors that are known to operate, in part, by directly sequestering PIFs and BZR1 from target promoters (Davière and Achard, 2016). The binding of GA to GID receptors increases GID affinity for DELLAs and initiates their degradation by the 26S proteasome (Davière and Achard, 2016). Recently, Suc (but not Glc) was shown to stabilize the DELLA RGA protein and inhibit its GA-mediated turnover. This stabilized DELLA is necessary for both the Suc-induced up-regulation of the anthocyanin biosynthetic genes and the Suc-induced hypocotyl growth repression in dark-grown seedlings (Li et al., 2014). A separate study using paclobutrazol (PAC) treatment (a GA biosynthesis inhibitor) implicated GA in the Suc induction of hypocotyl elongation in dark-adapted seedlings (Zhang et al., 2010). The BR-regulated transcription factor BZR1 also has been implicated in this dark-dependent Suc response (Zhang et al., 2016). Interestingly, BZR1 has been shown to complex with PIF4 to coregulate light- and hormone-responsive genes (Oh et al., 2012). Zhang et al. (2016) demonstrated that Suc increased the stability of BZR1 in a mechanism proposed to involve Target of Rapamycin (TOR) kinase. TOR is a central component in energy sensing and the regulation of biosynthetic processes such as ribose biogenesis and protein synthesis. Inhibiting TOR activity results in growth arrest and reduced expression of BR-responsive genes (Zhang et al., 2016). Hence, carbon availability controls a growth program through a TOR-dependent BZR1 pathway. Together, this analysis indicates that the PIF-BZR1-DELLA regulatory hub integrates light, carbon, and hormonal signals.

TOR also has been implicated in the integration of carbon and light signaling in the control of leaf initiation at the shoot apical meristem. For some time, phytochrome signaling has been known to control the rate at which leaves develop in Arabidopsis (Halliday et al., 2003). Light was shown to promote leaf initiation and meristematic activity by triggering the localization of the polar auxin transporter PIN1 and cytokinin signaling (Yoshida et al., 2011). More recently, an elegant study in seedlings demonstrated that light signals are relayed to shoot apical meristem cells through a long-distance cytokinin, most likely from the cotyledons (Pfeiffer et al., 2016). Furthermore, TOR kinase conveys both light signaling and energy information to control the expression of *WUSCHEL*, a gene that keeps stem cells in an active state. This finding has advanced our

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OUTSTANDING QUESTIONS

- What are the regulatory processes targeted by phytochrome signaling that lead to the changes in metabolite abundance?
- What causes the severe biomass deficiency observed in multi-allele phytochrome mutant plants and how does this tie in with photosynthetic capacity?
- What are the mechanisms of metabolic signal sensing by phytochrome signaling components?

thinking on how light signaling and carbon availability jointly coordinate growth.

CONCLUSION AND FUTURE PERSPECTIVES

We have given an overview of the interrelation of carbon resource management and metabolism and phy signaling. Owing to considerable scientific efforts, a picture is emerging where phys play important roles in driving growth plasticity and biomass production as well as controlling photosynthetic capacity and metabolite levels. However, we are far from fully understanding most of the underlying mechanisms. It is unclear how the known phy signaling mechanisms are connected to the metabolite profiles observed in phy mutants (see Outstanding Questions). Systems or modeling approaches could be used to help delineate these links and to understand the interplay between light signaling, carbon signaling, metabolism, and growth. The Arabidopsis Framework model integrates information from external light inputs, carbon resource production, and allocation to leaves and growth (Chew et al., 2014). This type of modular model could be used to predict the dual action of phytochrome and photosynthesis on resource management and biomass production. A long-term goal will be to understand how light-induced changes in molecular signaling and metabolism control plant plasticity.

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