Minireview

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Tracking the Light Environment by Cyanobacteria and the Dynamic Nature of Light Harvesting*

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In nature photosynthetic organisms cope with fluctuating light conditions. Light intensity and quality vary dramatically during the day or from one habitat to another. Photosynthetic organisms sense intensities and wavelengths of light both directly and indirectly. Because light fuels photosynthetic electron transport and CO_2 fixation, it is the primary determinant of levels of NADP/NADPH, ATP, and carbon metabolites, all of which can serve to modulate cellular processes. Light is also absorbed by photoreceptors that link light cues to cellular metabolism. However, light represents a single environmental cue, and other signals interact with light through a web of regulatory circuits that result in dynamic acclimatory responses. This review focuses on two specific aspects of light-influenced processes in Cyanobacteria; both concern changes in light harvesting structure and biosynthesis. The first part of this review discusses effects of changing wavelengths of light on the biosynthesis of the phycobilisomes (PBS),[1] dominant light harvesting complexes of Cyanobacteria. The other discusses how Cyanobacteria tune light harvesting and photosynthetic function to both light intensity and nutrient availability and how the two responses are integrated.

Light Harvesting and Fluctuating Light Signals

Phycobilisome Structure

PBS are peripheral membrane complexes in Cyanobacteria that efficiently harvest light energy and transfer the energy to photosynthetic reaction centers. PBS, which can comprise 30% of the cellular protein, are organized into two structural domains, the core and rods (Fig. 1). Each of these domains contains pigmented and nonpigmented polypeptides.

All PBS have the chromoproteins (phycobiliproteins) allophycocyanin (AP) and phycocyanin (PC), and many also contain phycoerythrin (PE) or phycoerythrocyanin. Phycobiliprotein colors are a consequence of light absorption by linear tetrapyrrole chromophores that covalently associate with the apoproteins (1, 2). Phycobiliproteins are composed of α and β subunits associated into heterodimers (termed "monomers" in the literature) that aggregate into trimers $(\alpha\beta)_3$ and hexamers $(\alpha\beta)_6$. Nonchromophorylated linker (L) polypeptides stabilize PBS, facilitate assembly of phycobiliprotein aggregates, and modulate the absorption characteristics of phycobiliproteins, promoting unidirectional energy flow to photosynthetic reaction centers (1).

PBS cores contain AP trimers along with pigmented and L polypeptides. A high molecular mass core polypeptide, or L_{CM}, has homology to both phycobiliproteins and L polypeptides (3). The phycobiliprotein-like domain of L_{CM} binds a tetrapyrrole chromophore and can serve as a PBS terminal energy acceptor. Generally, six rods, each composed of stacks of PC and PE hexamers, radiate from the core, giving PBS a fanlike appearance (Fig. 1) (see Ref. 2 for details).

Complementary Chromatic Adaptation

It was noted over a century ago that Cyanobacteria pigmentation changes with environmental light quality. This light control of pigmentation, shown in the *lower half* of Fig. 1, was termed complementary chromatic adaptation (CCA). Bennett and Bogorad (4) showed that CCA was the result of altered PBS pigment-protein composition. Development of molecular tools in the 1970s created new opportunities for elucidating PBS regulation, and by the end of the 1980s most genes encoding PBS structural polypeptides were characterized (5).

Genes of the PBS—In the Cyanobacterium *Fremyella diplosiphon* (similar to *Calothrix* PCC7601) the PE:PC ratio reflects the spectral distribution of light in the environment (6). In red light (RL) the organism has almost no PE, and each PBS rod can have three PC hexamers (and specific L polypeptides). If the Cyanobacterium is moved to green light (GL), new PBS are synthesized with rods having single PC hexamers (core proximal hexamer) and up to three PE hexamers. As the cells replicate in GL, blue-pigmented PBS of RL-grown cells are gradually diluted, and the cells begin to appear red. These light-responsive changes are reversible, and because PC absorbs RL (λ_{max} = 620 nm) and PE absorbs GL (λ_{max} = 560 nm), these changes facilitate efficient absorption of prevalent wavelengths of light in the environment.

Knowledge of genes encoding phycobiliprotein and linker polypeptide has been critical for understanding CCA (see Refs. 5 and 7)). Genes encoding α and β subunits of each phycobiliprotein are contiguous on the cyanobacterial genome and are cotranscribed. Often, polycistronic transcripts encode phycobiliprotein subunits and their associated L polypeptides. In *F. diplosiphon*, α and β AP subunits (α^{AP} and β^{AP}, respectively), encoded by the *apcA1B1* genes, are in an operon that also contains the *apcC1* and *apcE1* genes; the latter genes encode the core linker polypeptide and the L_{CM}, respectively (8).

Three distinct operons encode α^{PC} and β^{PC} subunits (*cpcBA* genes) in *F. diplosiphon* (9–12). The *cpcB1A1* genes are constitutively transcribed and encode PC_c subunits (subscript indicates constitutive). This operon also contains *cpcE* and *cpcF*, which encode a lyase that attaches the tetrapyrrole chromophores to the α subunit of PC (13). The *cpcB2A2* operon is specifically active in RL (inactive in GL) and encodes PC_i (subscript indicates inducible), which is critical for CCA. Hexamers of PC_i comprise the majority of PBS rods when cyanobacterial cells are grown in RL. The genes *cpcH2*, *cpcI2,* and *cpcD2* (14), encoding L polypeptides associated with PC_i, are cotranscribed with *cpcB2A2*. Furthermore, the *cpcB2A2H2I2D2* operon is clustered on the *F. diplosiphon* genome with *cpcB1A1* and *apcE1A1B1C1* (10). A third PC operon, *cpcB3A3* plus genes encoding associated L polypeptides, is only active during sulfur-limited growth (12).

The *cpeBA* operon encodes α and β subunits of PE (15). In contrast to the situation for *cpc* and *apc* operons, genes encoding L polypeptides associated with PE are not contiguous on the genome to *cpeBA*; they are encoded by the *cpeCDE* operon (16). However, GL activation and RL suppression of *cpeBA* and *cpeCDE* are coordinated. Also, *cpeBA* genes are linked to *cpeY* and *cpeZ*, which

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[1] The abbreviations used are: PBS, phycobilisome(s); AP, allophycocyanin; PC, phycocyanin; PE, phycoerythrin; L polypeptide, linker polypeptide; CCA, complementary chromatic adaptation; RL, red light; GL, green light; HL, high light; PAS, PER/ARNT/SIM; DCMU, 3-(3,4-dichlorophenyl)-1, 1-dimethylurea; DBMIB, 2,5-Dibromo-3-methyl-6-isopropyl-p-benzoquinone.

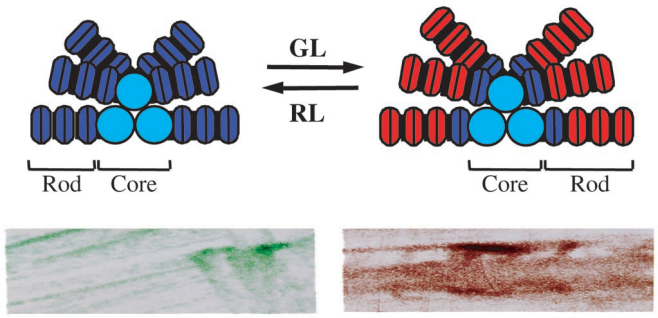

FIG. 1. **Cyanobacteria PBS.** *Top,* structure of PBS in RL and GL. Each disc in the rod substructure represents a phycobiliprotein hexamer; red-pigmented discs represent PE hexamers and blue-pigmented disks represent PC hexamers. *Bottom,* cyanobacterial cells on solid medium in RL (*left*) and GL (*right*).

encode the lyase that attaches tetrapyrrole chromophores to PE subunits (17).

Expression during CCA—Action spectra for the synthesis of PC and PE in the Cyanobacteria have been measured (18). Maximum PE synthesis and minimum PC synthesis occurred following exposure to 550 nm GL, and maximal PC synthesis and minimal PE synthesis occurred following exposure to 640 nm RL. Hence, photoreceptor(s) controlling CCA absorb RL and GL but elicit different responses in the two light qualities. PC synthesis dominates in RL whereas PE synthesis dominates in GL. Exposure of cells to natural sunlight, a mixture of RL and GL, results in the synthesis of PBS with intermediate PC and PE levels. Photocontrol of PE and PC_i levels is primarily a consequence of transcriptional regulation of *cpeBA* and *cpcB2A2* operons (19–21).

Mutants in CCA—The dissection of regulatory circuits involving CCA has exploited mutants abnormal for CCA. Several classes of CCA mutants have been isolated (5, 22–24), including the red (FdR), blue (FdB), green (FdG), and black (FdBk) strains. FdR mutants are red under all conditions of illumination and constitutively synthesize PE whereas PC_i is never synthesized. These mutants are fixed in a response normally exhibited only in GL, with aberrant regulation of both the *cpeBA* and *cpcB2A2* operons (22). FdB strains are bluer than wild-type cells in RL and require more GL to suppress PC_i synthesis (25). FdG mutants show normal PC_i expression, but the *cpeBA* genes never become active. FdBk mutants have moderate levels of both PE and PC_i, which remain the same in RL and GL (7, 24).

Initially, an FdR mutant was complemented by *rcaC*, which encodes a polypeptide of 651 amino acids with sequence similarities to response regulators of two-component regulatory systems (26). It is twice as large (73 kDa) as most response regulators and has two conserved, aspartate-containing receiver domains, one at the N terminus (Asp-51) and the other at the C terminus (Asp-576). The Asp-51 residue is likely phosphorylated in RL-grown cells, and the phosphorylation results in high level PC_i and little PE synthesis. In GL wild-type cells likely dephosphorylate Asp-51, which triggers elevated PE synthesis and depressed PC_i synthesis. Contiguous to the N-terminal receiver domain of RcaC is a sequence predicted to bind DNA. Between the putative DNA binding domain and the C-terminal receiver domain is a motif that resembles an H block of some unorthodox sensor proteins (27).

The FdBk class of mutants was complemented by *rcaE*, which encodes a polypeptide of 74 kDa (24). The C-terminal region of RcaE has motifs typical of bacterial sensor kinases (with a typical H block). The N-terminal half of the polypeptide has a domain of about 140 amino acids with similarity to the tetrapyrrole chromophore attachment domain of phytochromes. The central region of the protein contains a PAS domain (28), which may be involved in protein-protein interactions or binding of a redox-active prosthetic group. Recently RcaE was shown to covalently bind a linear tetrapyrrole chromophore at a cysteine within the phytochrome-like domain.[2] The phenotype of the FdBk mutant and similarity of RcaE to sensor kinases and eukaryotic phytochrome photoreceptors are consistent with a photoreceptor role for RcaE (7, 24).

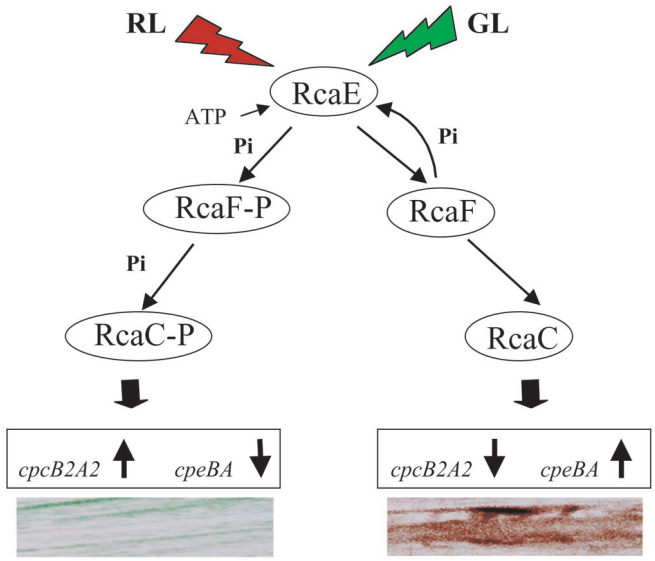

FIG. 2. **Phosphorelay regulation of CCA.** The *left side* depicts RL-stimulated phosphorylation of components of the signal transduction pathway, activation of *cpcB2A2,* and suppression of *cpeBA*. The *right side* depicts dephosphorylation of signal transduction components in GL and the suppression of *cpcB2A2* and activation of *cpeBA*. Pigmentation of cells under the different light conditions is shown.

Two FdR mutants were not complemented by *rcaC* (29). One was complemented by the putative photoreceptor gene *rcaE*, and the second by *rcaF*, which is immediately downstream of *rcaE* on the *F. diplosiphon* genome and encodes a small response regulator. RcaF may act as an intermediate in the phosphorelay pathway controlling CCA and facilitate phosphate transfer from its cognate sensor (presumably RcaE) to other response regulators such as RcaC.

Because *rcaE* is sufficient for complementing both FdBk (24) and FdR mutants, different lesions in *rcaE* can generate different phenotypes. Furthermore, an FdR phenotype can result from lesions in at least three distinct genes (*rcaC*, *rcaE*, and *rcaF*). The lesions that caused the mutant phenotype in these strains were the result of gene disruption by *in vivo* transposition (22). Each of the FdBk (*rcaE*-FdBk) and FdR (*rcaE*-FdR**,** *rcaF*-FdR) mutants characterized contained insertion sequences in the *rcaE/rcaF* operon (29). In *rcaE*-FdBk mutants the inserts were located within 200 base pairs of the putative translation start site, and no RcaE protein was detected in mutant cells.[2] The *rcaE*-FdR mutants contained insertions positioned between the H block and the four conserved motifs critical for histidine kinase activity; this strain appears to synthesize truncated RcaE. The *rcaF*-FdR mutants contained insertions located ~200 base pairs downstream of the *rcaF* translation initiation codon.

Constitutive PE and PC synthesis in *rcaE*-FdBk mutants reflect an intermediate activation state of the system as a consequence of low level phosphorylation of regulatory elements that are no longer under RcaE control. Hence, RcaF likely undergoes low level phosphorylation in the absence of RcaE; this phosphorylation is not controlled by light quality and may result from cross-talk between RcaF and other sensors or phosphoryl transfer from small molecule phosphodonors. It is not unusual that a null mutation in a sensor kinase leads to an intermediate, constitutive activation of the phosphorelay system. In the *rcaE*-FdR mutants, although the truncated RcaE cannot undergo autophosphorylation, it may bind RcaF and block its phosphorylation by other molecules and/or retain phosphatase activity, which would maintain RcaF in a dephosphorylated state.

Model for CCA—Three regulatory elements critical for CCA are RcaE, RcaF, and RcaC. Although these polypeptides have features of bacterial two-component regulators, the CCA phosphorelay (Fig. 2) is unique because it includes five potential phosphoacceptor domains among these polypeptides. RcaE, the putative photoreceptor, perceives the light signal. RL causes RcaE to undergo an

[2] D. M. Kehoe and A. R. Grossman, unpublished data.

autophosphorylation followed by transfer of the phosphoryl groups to the response regulator RcaF. In the absence of RcaE, RcaF may interact with other phosphoryl donors. RcaF then may transfer phosphoryl groups to the conserved histidine of the H block within RcaC, which can pass it to either the N- or C-terminal receiver domain. The N-terminal receiver domain of RcaC is critical for CCA, whereas the role of the C-terminal receiver domain is unclear. In GL RcaE acts as a phosphatase or blocks phosphotransfer by binding to RcaF; this inhibition causes activation of *cpeBA* and suppression of *cpcB2A2*.

Not surprisingly, other regulatory components also appear to be involved in controlling CCA. A class of mutants that only affects *cpeBA* expression has been identified and is designated turquoise (FdTq). These mutants exhibit normal regulation of *cpcB2A2* but cannot activate *cpeBA* in GL. Complementation of the FdTq strains uncovered two genes, *trqA* and *trqB*, encoding polypeptides related to protein phosphatases[3]; this finding is interesting, especially because phosphorylation of the putative regulatory protein RcaA has been implicated in the control of *cpeBA* expression (21).

Integration of Light and Nutrient Signals in PBS Biosynthesis

Specific Nutrient Limitation Responses—Responses of organisms to nutrient availability may be classified as those specific to the limiting nutrient and those that are more general, occurring during any of a number of different nutrient limitation conditions. Specific responses include metabolic changes enabling organisms to efficiently scavenge the limiting nutrient; these responses may include synthesis of high affinity transport systems (30) and production of hydrolytic enzymes that facilitate utilization of alternate forms of the limiting nutrient (31).

General Responses—General responses to nutrient-limited growth include changes in cell morphology and metabolism. *Synechococcus* cells starved for iron, nitrogen, or sulfur accumulate low levels of thylakoid membrane, PBS, and ribosomes (32, 33). Nutrient deprivation also causes a rapid loss of O_2 evolving activity, reflecting a decline in PSII function (34). A visually dramatic, general response of Cyanobacteria to nutrient-limited growth is the decrease in cellular pigmentation or bleaching (35), which includes an almost complete loss of PBS (36). Degradation of the PBS could provide amino acids or carbon skeletons for production of other cellular constituents required during nutrient deprivation and reduce absorption of excitation energy, making cells less susceptible to photodamage.

Mutants of *Synechococcus* were isolated that could not degrade their PBS during nutrient deprivation. Some of these mutants, designated *nbl* (nonbleaching), only survived in relatively low light. Complementation of one *nbl* mutant led to the isolation of *nblA* (nonbleaching), which encodes a 59-amino acid polypeptide. The *nblA* transcript only accumulates to high levels in cells starved for nitrogen or sulfur; low levels of the *nblA* mRNA are observed in cells maintained in phosphorus-free or complete medium. Under all conditions tested, *nblA* expression correlated with decreased PBS levels, even under conditions that do not normally provoke PBS degradation (37). *nblA* may be the primary (only) gene whose activity must be elevated to provoke bleaching during sulfur- or nitrogen-limited growth. Although NblA is probably not a protease itself, it may function to activate or alter the specificity of a protease or somehow tag PBS for degradation.

A second *nbl* mutant was complemented by *nblB*. NblB has homology to subunits (*e.g.* CpcE, CpeZ) of lyases that catalyze covalent attachment of phycochromobilin chromophores to apophycobiliprotein subunits (38). This finding suggests that NblB interacts directly with tetrapyrrole chromophores attached to phycobiliproteins. Because *nblB* mutants do not degrade PBS during nutrient limitation, it is reasonable to propose that NblB catalyzes removal of chromophores from holophycobiliprotein subunits and that only after the chromophore is removed can phycobiliprotein subunits be degraded.

A third mutant, *nblR*, was complemented by a gene encoding a response regulator (39). Cyanobacteria strains null for NblR (*a*) have ~150% the level of PBS as wild-type cells during nutrient-replete growth, (*b*) fail to degrade PBS during sulfur or nitrogen limitation, and (*c*) cannot properly modulate PBS levels during exposure to high light (HL). The (*a*), (*b*), and (*c*) phenotypes probably reflect the fact that the *nblR* mutant (*d*) cannot activate *nblA* during nutrient limitation. The mutant also dies rapidly when starved for either sulfur or nitrogen or when exposed to HL (39). Hence, in addition to controlling PBS degradation, NblR modulates functions critical for cell survival during nutrient-limited and HL conditions. The presence of the photosynthetic electron transport inhibitor DCMU slows the death of the *nblR* mutant during sulfur and nitrogen starvation, suggesting that death of the mutant is a consequence of its inability to properly down-regulate photosynthesis upon exposure to stress conditions. Hence, NblR appears to have a pivotal role in regulating some general stress responses and is critical for integrating various environmental signals with cellular metabolism.

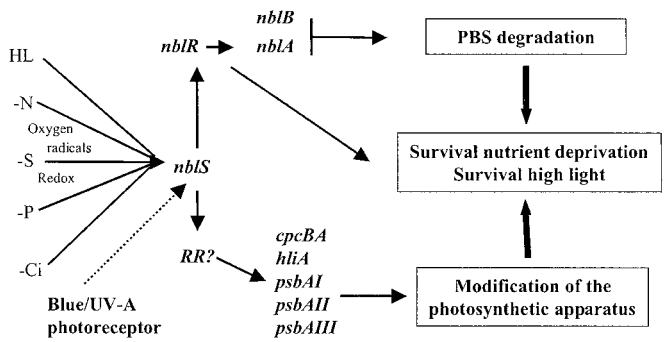

FIG. 3. **Model depicting the role of NblS in controlling both HL and nutrient stress responses.** The genes shown in the figure are discussed in the text. *HL*, *-N*, *-S*, *-P*, and *-Ci* represent conditions in which the cells are exposed to HL or are deprived of nitrogen, sulfur, phosphorus, or inorganic carbon, respectively. The model suggests that these conditions cause a change in cellular redox and/or in the level of reactive oxygen species, which is sensed by NblS. NblS may also control some process through direct absorption of blue/UV-A light.

The most recently characterized *nbl* mutant is *nblS*. Like *nblR*, the *nblS* mutant is sensitive to HL and nutrient limitation and cannot properly activate *nblA*. Furthermore, the *nblS* strain cannot activate *hliA* (gene encoding a polypeptide with similarity to chlorophyll *a*- and *b*-binding proteins of plants that helps Cyanobacteria acclimate to HL (40)), *psbAII,* and *psbAIII* during exposure of *Synechococcus* to HL or blue/UV-A light.[4] NblS is also important for the down-regulation of the *psbAI* and *cpcBA* genes in HL.

The *nblS* gene encodes a sensor histidine kinase of two-component regulatory systems, with two predicted N-terminal membrane-spanning domains followed by a region bearing a PAS domain. PAS domains often bind flavin or heme prosthetic groups and may respond to light or redox conditions (28). Preliminary work suggests that NblS binds a flavin.[5] Because NblS controls nutrient stress responses and accumulation of *nblA* mRNA (like NblR), we assume that NblS and NblR constitute a sensor-response regulator pair, although this has not been proven. Interestingly, induction of *hliA*, *psbAII,* and *psbAIII* in HL does not appear to be under the control of *nblR*, suggesting that NblS modulates regulatory pathways that are distinct from those controlled by NblR. Overall, the results suggest that NblS serves to link the light environment and cellular redox to global modulation of metabolic processes.

Model—The integration of light and nutrient limitation responses are presented in Fig. 3. NblS is critical for integrating these responses, which include modulation of PBS levels and controlling the activities of *nblA*, *hliA*, *psbAI*, *psbAII*, *psbAIII,* and *cpcBA*. It is not yet clear if NblS is critical for regulating transcription of other genes that rapidly respond to changes in light conditions. During nutrient limitation, as cells generate reduced photosynthetic electron carriers, NblS initiates a phosphorylation cascade in which NblR is activated. NblR turns on the *nblA* gene,

[3] D. M. Kehoe, personal communication.

[4] L. van Waasbergen, N. Dolganov, and A. R. Grossman, submitted for publication.

[5] J. Christie, L. van Waasbergen, W. Briggs, and A. R. Grossman, unpublished data.

and NblA in conjunction with NblB (constitutively expressed) actively degrades PBS. NblR must also activate other processes in the cell (*e.g.* modification of the photosynthetic apparatus) that favor survival during nutrient stress and HL conditions. During HL exposure, NblS modulates the activity of many genes encoding components of the photosynthetic apparatus. Regulation of a number of these genes does not require NblR. Hence, in addition to interacting with NblR, NblS must associate with other response regulators that have not yet been identified.

A major question remaining is how may NblS be sensing the redox state of the cell. During nutrient limitation, when the anabolism of the cell is slowed down or completely arrested, $NADP^+$, the final electron acceptor of the photosynthetic electron transport chain, is not recycled as fast as under nutrient-replete conditions and the electron carriers are maintained in a relatively reduced state. Similarly, under HL conditions the electron carriers may be reduced faster than they are re-oxidized. Thus, nutrient limitation and HL result in absorption of excess light energy by photosynthetic pigments, and the overall cellular environment would become highly reduced. The redox state of photosynthetic electron carriers is known to modulate cellular transcription (41), translation (42), state transitions, and changes in the stoichiometry of the photosystems (43). The results discussed above suggest that NblS/NblR control is linked to the redox state of the cells and possibly couples to the degree of reduction of specific photosynthetic electron carriers. Furthermore, both DCMU, which prevents photosynthetic electron flow beyond Q_A, and DBMIB, which inhibits electron flow through the cytochrome b_6f complex, inhibit PBS degradation and the accumulation of *nblA* mRNA during sulfur and nitrogen stress. Like PBS degradation and *nblA* expression, expression of *hliA* is altered by both DCMU and DBMIB. Interestingly, upon treatment of *Synechocystis* PCC6803 with cyanide (which inhibits both respiration and photosynthetic electron flow at plastocyanin) *hliA* is activated in low light. Similarly, in a PSI mutant of *Synechocystis* PCC6803, there is elevated expression of *hli* genes even in moderate light.[6] These results suggest that hyper-reduction of an electron transport carrier prior to PSI may strongly affect acclimation during HL and nutrient limitation conditions. Factors in addition to the redox state of the photosynthetic apparatus may be important for tuning the activity of NblS. For example, NblS control seems to promote PBS degradation during nutrient limitation, but in HL it biases control toward activation of *hliA/psbAII/psbAIII/cpcBA*. Interactions with a blue light photoreceptor or the direct absorption of blue/UV-A light by NblS may also bias NblS action toward the *hliA/psbAII/psbAIII* regulatory pathway over the *nblA* pathway. Reactive oxygen species may also serve as regulatory signals that modulate the activity of the *nbl* system. Several aspects of the model are speculative and/or incomplete. However, it represents an attractive unifying view that predicts global metabolic effects in response to redox status of photosynthetic electron carriers and links nutrient conditions, growth potential, and light to overall regulation of cellular metabolism.

[6] W. Vermaas, personal communication.

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