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MINIREVIEW

Light-Responsive Gene Expression in Cyanobacteria

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The cyanobacteria constitute a kingdom of the domain *Bacteria* (44) whose members manifest a rich diversity of habitats and metabolisms. Species differ with respect to possession of a panoply of specializations, a sampling of which includes diazotrophy, filamentous growth form, production of buoyancy organelles, and differentiation of specialized cell types (5, 47). However, the group can be defined by the unifying property of chlorophyll *a*-based, oxygenic photosynthesis (57). This uniquely cyanobacterial photoautotrophism is the source of the Earth's oxidizing atmosphere (51) and extends to plants, whose chloroplasts trace their ancestry to this kingdom (44, 57).

Given the central role of photosynthesis in the metabolism of cyanobacteria, it is not surprising that the light environment directly regulates expression of genes that encode key components of the photosynthetic apparatus. The two structures whose light-responsive regulation has been best studied are the photosystem II (PSII) complex itself and its closely associated light-harvesting antenna, the phycobilisome. Genes that encode components of both complexes are likely to be under the control of wavelength-specific photoreceptors that initiate light-responsive signal transduction pathways.

Regulation of genes encoding the PSII reaction center proteins. At the heart of the PSII reaction center is a dimer of two structurally related proteins designated D1 and D2, each of which contributes ligands to the cofactors that mediate primary photochemistry (1). The genes that encode these two proteins, *psbA* and *psbD*, are conserved in plants, where they reside as single genes in the chloroplast genome. In contrast, cyanobacteria have one (25) to four *psbA* genes and two *psbD* genes (21). The regulation of the *psbA* and *psbD* genes is best understood for *Synechococcus* sp. strain PCC 7942, a unicellular cyanobacterium which has three *psbA* genes that encode two distinct forms of the D1 protein. The three genes are regulated differentially in response to changes in light intensity. In the laboratory, this is demonstrated by a low- to high-light shift equivalent to the increase in light intensity that a cell in shallow water might experience when a cloud moves away from blocking the sun. At low light, greater than 80% of the *psbA* transcripts are from *psbAI*, and the only D1 protein detectable in the thylakoid membrane is form I, the product of this gene (13, 32). Immediately upon a shift to high light, the *psbAII* and *psbAIII* genes are induced, and the *psbAI* message is actively degraded (34). After 30 min of exposure, the *psbAII* message makes up 80% of the *psbA* transcript population, and the identical products of this message and that of *psbAIII*, collectively known as form II, are substituted for form I in the thylakoid (13, 32). Several lines of evidence suggest that the two forms of D1 are functionally distinct and that form II provides a selective advantage at high light (12, 13, 32, 33).

The D2 polypeptide is encoded by a two-member *psbD* family, one gene of which is subject to regulation by light (6). The dicistronic *psbDI-psbC* operon encodes D2 and CP43, a chlorophyll *a*-binding protein of PSII. It is expressed with little variation at all light intensities. The monocistronic *psbDII* gene is expressed at very low levels when cells are at low light intensity but is rapidly induced when the light intensity increases. Because the two *psbD* genes encode identical polypeptides, light-responsive *psbD* expression causes quantitative changes in D2 production rather than a qualitative interchange like that of the two forms of D1. The regulatory events seem to be important for physiology: growth of a mutant which lacks an active *psbDII* gene is impaired at high light, and its thylakoids sampled at high light show a deficiency in D2 (6).

Intensity (photon flux density) and spectral quality are interdependent characteristics of light which are not completely separable. Although light-responsive expression of the *psbA* and *psbD* genes has been studied during shifts in intensity of white light, recent experiments revealed that the characteristic low- to high-light shift responses are induced by low-fluence blue light (56). Like the phenomena associated with complementary chromatic adaptation (CCA) described below, this appears to be a chromatically reversible response, with red light canceling an inductive blue signal. Therefore, subtle shifts in the blue/red ratio may provide information about changes in the light environment that are linked to light intensity.

The physiology of PSII suggests that intensity may be the important parameter, even if the photoreception system is set up to work through light quality. The rate of photosynthetic electron transport is proportional to light intensity up to a point, but plants and cyanobacteria enter a state known as photoinhibition under very high light intensity or under other conditions in which light intensity exceeds the capacity of the cell to transport electrons (such as limitations in CO_2 or specific ions) (46, 59). The prevailing model for the source of photoinhibition is that the D1 polypeptide is specifically damaged during photochemistry and that it must be cleaved, removed from the PSII complex, and replaced to maintain photosynthetic electron transport. When the rate of damage exceeds that of synthesis of new D1 subunits, photoinhibition results (42, 43, 52, 58). The changes in *psbA* and *psbD* expression and in PSII protein synthesis and turnover match well with predictions based on a need for increased synthesis of the reaction center subunits at high light (6, 31, 32). It is not yet clear whether the blue/red and low/high-light responses of the *psbA* family in *Synechococcus* sp. result from the same, independent, or overlapping sensory pathways (56).

Unusual *cis* elements in the untranslated leader regions of the *psbA* genes. A series of facts suggested that transcriptional regulation accounts for the rapid increases in *Synechococcus* sp. *psbAII* and *psbAIII* messages at high light (7). For example,

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β-galactosidase activities expressed from translational gene fusions, whose transcripts do not have the stabilities of the native messages, increase in high light (48). The half-lives of the native messages do not increase in high light (34); in fact, the *psbAIII* message, like the *psbAI* message, is actually destabilized under high light, and the increase in steady-state message level provides an underestimate of the gene's induction (34).

More direct evidence for transcriptional control was obtained by a systematic effort to map the regulatory elements of the *psbAII* and *psbAIII* genes (36). The goal was to determine what constitutes a *psbA* promoter and where the information for light-responsive expression lies. PCR was used to generate small fragments with defined endpoints which were fused transcriptionally to *lacZ* and placed in the *Synechococcus* sp. chromosome. Light-shift experiments revealed that the upstream regions of both *psbAII* and *psbAIII* are composed of three separable elements: basal promoters, comparable in position and sequence to σ^{70}-type promoters of *Escherichia coli*; negative elements immediately upstream of the basal promoters; and light-responsive elements downstream of the transcription start sites, corresponding to the untranslated leader regions of the transcripts. The light-responsive elements have the properties of enhancers, as demonstrated by their ability to increase expression (regardless of light intensity) from a heterologous promoter in a position- and orientation-independent manner. The additional feature of conferring light-responsive expression is observed with either upstream or downstream placement but requires the native orientation. Unlike the enhancers described for *E. coli* and *Caulobacter crescentus* (20, 22, 40), these elements appear to work with σ^{70}-type promoters that do not require activation. Similar characterization of the negative elements with a heterologous promoter has not been performed.

One or more proteins binds upstream of *psbAII*, *psbAIII*, and *psbDII*, the high-light-induced PSII genes (5a, 17, 35, 39). Moreover, the regulatory regions of these three genes compete with each other for protein binding, although the binding sites share no obvious sequence motif (17, 35). DNA mobility shift assays indicated *psbAII* binding activity in both low- and high-light extracts (39) and narrowed the binding region to the enhancer element (35). However, in vivo footprinting showed protection of nucleotides from methylation only in high light, suggesting that binding is a regulated event in the intact cell. Deletion of 12 bp from within the enhancer element altered the DNA mobility shift of the fragment in vitro and greatly reduced the response to high light in a reporter gene fusion (35). These data implicate the enhancer-binding factor(s) as mediators of the light-responsive transcriptional regulation.

Posttranscriptional regulation. A significant component of the light response of the *Synechococcus* sp. *psbA* genes is posttranscriptional. Both the *psbAI* and *psbAIII* transcripts turn over more rapidly at high light than at low light, and this accelerated degradation requires de novo transcription and translation after exposure to high light (34). The stability of the *psbAII* transcript is unaffected by light intensity. After prolonged exposure to high light, the *psbAI* transcript returns to its low-light turnover rate, and the *psbAIII* message continues to be degraded at an accelerated pace (32). This information alone implicates the upstream untranslated leader region as a determinant of message stability, since this is the only portion of the message which is unique for each gene; *psbAII* and *psbAIII* messages are very similar in the open reading frame and nearly identical in the 3′ untranslated region. Construction of chimeric genes suggests that the information for *psbAI* light-responsive transcript stability resides in the first 110 bases of the message (33). If the untranslated leader region is shown to be a message stability determinant for *psbAIII*, then a stretch of 50 nucleotides will appear to serve two functions in transcriptional control, as enhancer and light-responsive element, and a major role in posttranscriptional control as well!

The transcriptional and posttranscriptional regulation of *psbA* genes in *Synechococcus* sp. is probably not unique to this strain. The *psbA* family in *Synechocystis* sp. strain PCC 6803 also has three members which theoretically encode two forms of the D1 protein; however, the *psbA1* gene, whose product would be different from that of the other two, is not expressed under typical laboratory conditions (38a). A shift from low to high light results in an increase in overall *psbA* transcript in PCC 6803. As in PCC 7942 (56), induction by high light is insensitive to inhibition of photosynthetic electron transport (38b). Marked changes in *psbA* message stability are observed between cells incubated in the light or in the dark (38b), suggesting posttranscriptional regulation. *Synechocystis* sp. strain PCC 6714 shows evidence for regulation of *psbA* genes through light-dependent shifts in herbicide resistance when one gene in the family carries a herbicide resistance allele and the other carries a wild-type allele (3).

The complex regulation of the *psbA* genes in *Synechococcus* sp. does not appear to be an economical control system for responding to light. Surely a more parsimonious strategy could protect and regulate the photosynthetic apparatus without simultaneously increasing synthesis and degradation of the same components and without using multiple genes to encode equivalent products. The utility of the network may become more obvious as other sensory input pathways are discovered. The *psbA* genes of *Synechococcus* sp. respond to light specifically but are also under the more general control of the daily cycle in nature of light and darkness, by way of a circadian clock (29). Clear evidence for the existence in cyanobacteria of daily activity cycles that have the properties of circadian rhythms has been demonstrated in recent years (10, 26, 29, 38, 50). The robust circadian regulation of *psbAI* has allowed automated monitoring of a bioluminescent *psbAI-luxAB* reporter strain which resulted in identification of circadian clock mutants (30). It is likely that additional signals from the environment also influence expression of the *psbA* genes, since the products of these genes are so crucial for growth and survival. The complex web of numerous levels of control may offer more flexibility and opportunity for adjustment than would be readily afforded without gene families.

The phycobilisome and CCA. The molecular basis of another light-responsive regulatory process is emerging in *Fremyella diplosiphon* (*Calothrix* sp. strain PCC 7601). In the process of CCA, the cyanobacterium tailors the composition of the light-harvesting phycobilisome to display the phycobiliprotein which maximally absorbs the prevailing wavelength (for recent reviews, see references 23, 24, and 55). When this species is grown in red light, the phycobilisome rods are composed of phycocyanin, a red-absorbing pigment protein. In green light, the rods contain fewer discs of phycocyanin and are tipped with discs of phycoerythrin, a green-absorbing pigment. One phycocyanin operon, designated *cpcB1A1*, is expressed constitutively. A second operon, *cpcB2A2*, is expressed only when cells are exposed to red light. Green light stimulates the *cpeBA* operon, which encodes phycoerythrin apoproteins. The photoreceptor responsible for this regulation has not been identified, but its key properties are well-known: one or two photoreversible pigments, having an action spectrum that peaks in the green (540-nm-wavelength) and red (640-nm-wavelength) regions of the spectrum, read the light environment and set in motion the signal transduction pathways that control CCA (24).

Regulatory elements of CCA. A variety of *F. diplosiphon* mutants defective specifically in CCA have been characterized (4). Among them is FdR1, described as red light indifferent because it always displays the characteristics of green-light-grown cells: high levels of expression from *cpeBA* and none from *cpcB2A2*. Recent developments in genetic manipulation of this species (14) allowed complementation of the mutant with a library of wild-type DNA, resulting in restoration of response to red light (11). The complementing open reading frame, *rcaC*, encodes a member of the response regulator family of signal transduction proteins (45) that has characteristic receiver domains at both the amino and carboxy termini (11). Mutagenesis of the putative phosphorylation site in the amino-terminal receiver domain has confirmed that this residue affects the ability of cells to regulate CCA (28). The role of the carboxy-terminal receiver domain and the nature of interaction of RcaC with the phycobiliprotein operons are not known.

The rapid increases in *cpeBA* and *cpcB2A2* messages in green and red light, respectively, occur without a change in messenger half-life, suggesting control by transcriptional regulation (41). Not surprisingly, proteins bind to DNA upstream of the *cpeBA* and *cpcB2A2* operons. Sobczyk et al. (54) identified two proteinaceous *cpeBA*-binding activities, designated RcaA and RcaB, in extracts from cells grown in green light, but not in those from cells grown in red light. Phosphatase treatment prevented binding of RcaA to DNA, suggesting a phosphorylation-dephosphorylation transition. Schmidt-Goff and Federspiel independently reported that a protein binds to the same site as RcaA upstream of *cpeBA* and designated the binding activity as PepB (49). However, this group observed the DNA-binding activity in both red- and green-light-grown cells. Both groups propose that posttranslational modification of the protein is responsible for the light-responsive change in *cpeBA* expression, and both speculate that an alternate sigma factor may be involved. Sobczyk et al. suggest that RcaB, which interacts with the regulatory region of *cpeBA* and modifies the footprint of RcaA and RNA polymerase, could be such a sigma factor (54).

Two groups also identified protein-binding sites upstream of *cpcB2A2*. Sobczyk et al. defined two sites, box 1 and box 2, which bind a factor designated RcaD (53). They detected the DNA-binding activity only in cells grown in red light. They propose that RcaD is a positive effector of *cpcB2A2* expression whose activity is modified by the CCA signal transduction pathway. Casey and Grossman (9) also found that a factor binds upstream of *cpcB2A2* at a sequence equivalent to box 1. However, they found this activity in both red- and green-light-grown cells and concluded that it is not related to CCA regulation, because transcriptional fusions between *cpcB2A2* promoter fragments and the β-glucuronidase gene do not require this binding site for red-light-responsive expression (9). Both groups detected a second binding activity near the promoter which may be involved in regulating CCA.

Despite discrepancies among the findings of different research groups, the components of the CCA signal transduction pathway are nearing elucidation. The combination of in vitro and in vivo methods for analyzing protein-DNA interactions at the regulated loci, as well as the ability to identify additional loci through complementation of CCA mutants, should result in a unified picture of the regulatory circuit. Hopefully, the elusive photoreceptor, too, will yield to the power of genetics.

Other light-regulated genes: pursuit and serendipity. Effects of light intensity or light quality on expression of known genes and new genes are being investigated in many cyanobacterial species. Phycobiliprotein genes, regulated by spectrum in some species, are also responsive to light intensity, and this regulation may occur at both transcriptional and posttranscriptional levels (2, 16, 27). Marraccini et al. (37) devised a selection for light-regulated promoters in *Synechocystis* sp. strain PCC 6803. They attached random fragments from the PCC 6803 genome to a promoterless *cat* gene and selected for chloramphenicol-resistant cyanobacterial colonies that formed in bright light. Among these clones, they identified several that did not grow well on the antibiotic in dim light, suggesting differential expression of the reporter gene in the two light regimens. Comparison of a number of the inserts identified a consensus sequence which may be involved in regulation, but the physical relationship of the aligned consensus with respect to transcription start sites has not yet been determined.

A new light-regulated gene in *Synechococcus* sp. was identified recently by analysis of DNA in proximity to a known lipopolysaccharide biosynthesis gene (18, 19). The open reading frame of the new gene, *hliA*, predicts a protein that shows strong similarity to the chlorophyll *a*-binding proteins and early light-induced proteins of plants. Subsequent analysis of *hliA* revealed that its expression is induced by white light of high intensity or by blue wavelengths (19).

The recognition of specific wavelengths is clearly an important feature of the machinery that mediates light-regulated gene expression in cyanobacteria. The differentiation of hormogonia and heterocysts is regulated by red and green light in *Calothrix* spp. strains PCC 7601 and 7504 (8, 15). Despite the spectral similarity with CCA, the differentiation signal seems to work through the photosynthetic apparatus itself, and electron transport, and is independent of CCA (8).

Concluding remarks. Undoubtedly the signal transduction pathways for light-responsive gene expression in cyanobacteria will have much in common with known bacterial pathways. However, their integration with the photoreception event promises to present some novelty. Several twists on the common themes, such as the odd enhancers of the *psbA* genes, are likely to remind us that the cyanobacteria have had billions of years to develop tricks for adapting to the environment and that they are not, after all, just blue-green *E. coli.*

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