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MINIREVIEW

Control of Photosystem Formation in *Rhodobacter sphaeroides*

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INTRODUCTION

Rhodobacter sphaeroides has the capacity to grow by aerobic and anaerobic respiration and photosynthetically in the light under anaerobic conditions, as well as fermentatively. It can fix atmospheric nitrogen and carbon dioxide. It resembles other gram-negative members of the class *Proteobacteria* when growing aerobically, but a reduction in oxygen tension induces an intracellular differentiation of the inner membrane, leading to the formation of the intracytoplasmic membrane system (ICM). The ICM houses the integral membrane pigment-protein complexes constituting the photosystem (PS), comprised of the reaction center (RC) and two light-harvesting (LH) complexes. For an early review of ICM biosynthesis, see reference 44. The LH complexes are designated B800-850 (LHII) and B875 (LHI), based on their respective absorption maxima. The ratio of LHI to RC is fixed at approximately 15:1, whereas the ratio of LHII to the LHI-RC unit is variable, changing in a manner inverse to the incident light intensity. These three pigment-protein complexes are the spectral complexes (SC) of the *R. sphaeroides* PS. Detailed structural information about these complexes in several species of *Rhodobacter* is emerging (72).

The LH complexes capture light energy and direct that energy to the RC, where conversion of the excitation energy takes place and is intrinsically coupled with a cyclic flow of electrons, ultimately to the periplasmically localized cytochrome c_2, which serves to rereduce the RC to allow a new cycle of electron flow (for further details, see references 42, 44, and 72).

Bacteriochlorophyll (Bchl) absorbs most of the light energy within the SCs and is critical to the assembly and final structure of the SCs (44, 84, 87). The carotenoids (Crt) have a minor role in absorbing light energy (15), but they function to protect the complexes against photo-oxidative damage, dissipate excess radiant energy, and help to maintain the structure and relative abundance of each SC (35, 50, 53).

A reduction in oxygen tension is both necessary and sufficient to induce synthesis of the ICM (reviewed in references 42 and 44), which is gratuitously produced under anaerobic dark growth conditions, in the presence of an alternate electron acceptor such as dimethyl sulfoxide (DMSO). Oxygen tension is the major environmental stimulus controlling PS induction, with variations in light intensity determining the cellular level of the ICM and the abundance of the different SCs. PS formation is tightly regulated, with checkpoints at all levels of information flow, from transcriptional through posttranslational. In the following sections, we will describe what we currently know about the regulatory processes controlling the formation and abundance of SCs in *R. sphaeroides*. We will present a working model for the regulation of PS formation in *R. sphaeroides* 2.4.1 which is based on the critical role of cellular redox carriers. For clarity, we will, throughout this review, define aerobic growth as that which occurs under highly oxygenic conditions, under which there are no detectable SCs present in wild-type membranes.

TRANSCRIPTIONAL REGULATION OF PS GENE EXPRESSION

Transcriptional regulation of PS genes, from an initial low background level, involves the coordinate action of several signal transduction pathways which result in the induction of PS gene expression following oxygen removal. Some of these pathways are specific to PS genes and others are more global, while still others are commonly recognized to be involved in cellular adaptation to anaerobiosis. Different PS genes are not all controlled by the same regulatory pathways, although these may overlap. We first describe those factors which appear to play a major role in sensing, signal generation, and transduction, as well as DNA binding. Thereafter, we describe other factors which have less pronounced effects and/or less obvious roles in PS gene transcription; these are believed to participate in fine tuning of transcriptional control. Finally, we described posttranscriptional processes essential to SC formation and abundance.

Sensors, signal generators, and transducers. (i) The Cco/Rdx redox signal-generating system. The identification of CcoNOQP cytochrome *c* oxidase and RdxBH (redox) as components (Fig. 1) of a signal-generating pathway stemmed from the observation that inactivation of the *ccoNOQP* operon resulted in PS gene expression (93, 94) under aerobic conditions. The *ccoNOQP* operon encodes the cbb_3-type cytochrome *c* oxidase of *R. sphaeroides* 2.4.1. Of the three respiratory oxidases present in *R. sphaeroides*, the cbb_3 type is believed to have the highest affinity for oxygen and is the primary oxidase present under microaerobic conditions (26, 27, 37). Immediately downstream of the *ccoNOQP* operon is the *rdxBHIS* operon (70, 94). These operons are located at **443 kbp** on chromosome I (94). (The locations of genes that map outside the PS gene cluster and are pertinent to this discussion are in boldface throughout the text.) The *rdxB* gene is a homolog of the *rdxA* gene, which maps to chromosome II (69). Both RdxB and RdxA are predicted to coordinate two [4Fe-4S] clusters (69), and these, together with other clusters of cysteine residues, may be in-

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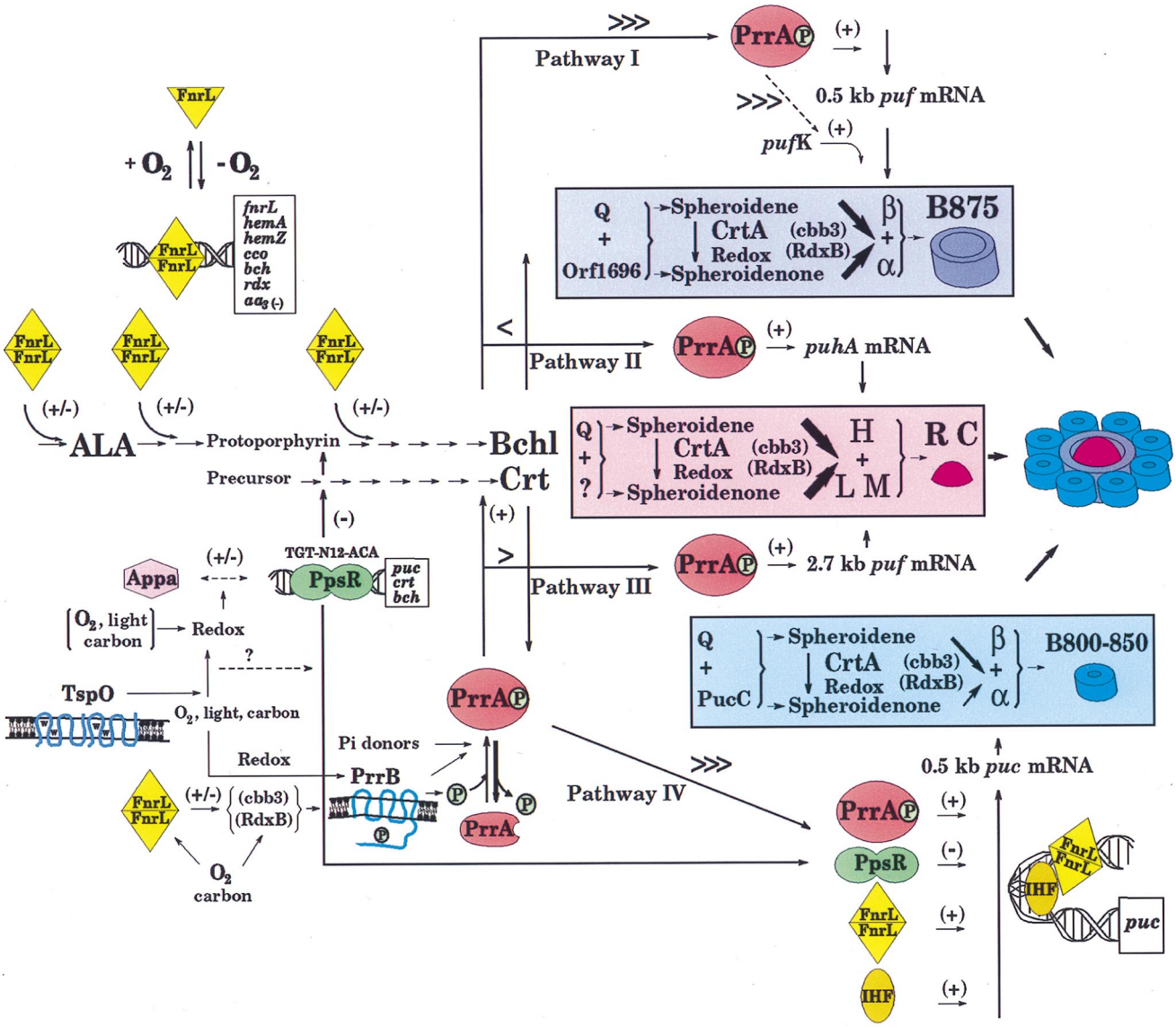

FIG. 1. Most of the PS genes are arranged in a cluster on chromosome I of *R. sphaeroides* 2.4.1 (83). This region of approximately 60 kb begins with the *pufKBALMX* operon and extends, in a clockwise direction, to the *pucBAC* operon, which is 18 kb downstream of *puhA*. The *pufB* and *-A* genes encode structural polypeptides of the LHI SC, the *pufL* and *-M* genes encode two of the three structural proteins of the RC, and the two structural polypeptides of the LHII SC are encoded by *pucB* and *-A*, respectively (reviewed in reference 44). The *Q* gene, located upstream of the *puf* operon, together with the *pufK* and *pucC* genes, are not part of the PS, nor is *orf1696*. The *pufX* gene product is required for PS competence (4); it facilitates light-driven cyclic electron transfer (23). Between *pufKBALMX* and *puhA*, the latter of which codes for the largest structural polypeptide of the RC, are genes encoding enzymes catalyzing the biosynthesis of Crt and Bchl (1–3, 8, 9, 17, 52) and some genes that have a regulatory role in PS gene expression. Between *puhA* and the *pucBAC* operon is the *cycA* gene (19, 97), encoding the obligatory cytochrome c_2 protein. In the related bacterium *R. capsulatus*, a similar arrangement of a portion of these genes extending from *puf* to *puh* (25), called the photosynthesis gene cluster, is present, and the DNA sequence of the entire *R. capsulatus* photosynthesis gene cluster has been determined (14). The PS gene cluster is defined as the region from 0 to 3,000 kbp on chromosome I of *R. sphaeroides* 2.4.1. In the text are presented the locations, in boldface, of those genes that map outside this cluster and are pertinent to the discussion presented here. For a detailed description of the above model of PS gene regulation, see the text. ALA, 5-Aminolevulinic acid.

volved in redox processes. They are both likely to be membrane bound, as has been shown for RdxA (69).

Mutations in either *ccoNOQP* or the *rdxB* gene generate mutants which, under aerobic growth conditions, produce SCs, as well as high levels of expression of *pucBAC* and *puf KBALMX*. Uncoupling of PS gene expression from the presence of oxygen in Rdx and Cco mutant strains involves the activation of the two-component PrrBA regulatory system (71). Thus, a pathway involving cbb_3 through its interaction with oxygen serves as an oxygen "sensor" to generate an "inhibitory signal" through RdxB to, presumably, PrrB (see below), which results in a lack of PrrA activation. This inhibitory signal may be through the membrane portion of PrrB. However, no physical interaction between cbb_3 and PrrB has been demonstrated. This is analogous to the Arc system of *Escherichia coli* (40). The fact that the levels of SCs and PS gene expression in cbb_3 and RdxB mutant strains are also substantially increased relative to those of the wild type, even under anaerobic conditions, strongly affirms the likely functioning of these redox carriers under such conditions. Under these same conditions, the activation of PS gene expression in these mutant strains is dependent upon the Prr system. FnrL (see be-

low) is indirectly involved in modulating the activity of PrrA by regulating the level of *ccoN* gene expression in response to oxygen levels (~10-fold increase following a high-to-low O_2 transition; 66) and, presumably, *rdxB* as well.

(ii) The redox-responsive two-component Prr system. Prr (photosynthetic response regulator) is a signal transduction system (Fig. 1) involved in the activation of PS gene expression (20–22). The *prrA* gene product functions as a response regulator, and the *prrB* gene product functions as a sensor histidine kinase/phosphatase. A third gene, *prrC*, located upstream of *prrA*, encodes a membrane-associated protein. This genetic region is located outside of the region of PS genes, **1149 kbp**, on chromosome I of *R. sphaeroides* 2.4.1. The *prrCA* genes form a transcriptional unit, although *prrA* has its own promoter and *prrB* is divergently transcribed from *prrCA*. Prr is similar to the *R. capsulatus regBA*-and-*senC* system, which is also involved in regulation of PS gene expression (64, 79), as well as the *Rhizobium meliloti actRS* system, which is involved in acid tolerance (86). PrrBA regulates transcription of *puhA*, *cycA*, and the *puc* and *puf* operons, as well as some of the genes involved in Bchl and Crt synthesis and/or accumulation. The expression of genes involved in CO_2 and N_2 fixation is also affected by the Prr system (41). Inactivation of *prrA* causes a photosynthesis-negative phenotype, as the result of a nearly total shutdown of PS gene expression. Mutants defective in *prrB* are photosynthesis negative when grown at low-to-medium light intensities but grow photosynthetically at high light intensities, albeit with severely reduced levels of SCs. PrrC inactivation also leads to a small reduction in SC formation, but cells remain photosynthetically competent.

PrrA is thought to be cytoplasmically located. The carboxy-terminal end of PrrA lacks similarities with other DNA-binding proteins. Nonetheless, PrrA acts either by binding DNA directly (45) or by interacting with another protein(s) that binds DNA. When *prrA* is present in multicopy in aerobically growing wild-type or $PrrB^-$ cells, such cells aberrantly produce spectral complexes and have increased *puc* expression despite the inhibitory signal emanating from cbb_3 (22). This further suggests that other phosphodonors are active in activating PrrA (20, 30, 71) and that PrrB may possess an intrinsic phosphatase activity (20). Thus, the cellular levels of PrrA must be carefully regulated (22). Additionally, there is some evidence to suggest that different PS genes require different levels of active PrrA for their activation (20).

In *R. capsulatus*, a truncated form of the PrrB homolog RegB lacking the membrane-associated domain phosphorylates the PrrA homolog RegA in vitro (39). A point mutation involving a Leu-to-Pro change at position 78 of the PrrB protein (PRRB78) confers an oxygen-insensitive phenotype on the cell (21); i.e., PS gene expression is "full on" under both aerobic and anaerobic conditions. This region of the protein is believed to be located in a nonmembranous cytoplasmic loop far removed from the active histidine.

The role of PrrC, SenC in *R. capsulatus* (11), in PS gene expression is much more subtle than that of PrrA or PrrB. PrrC shares amino acid sequence similarity with the yeast ScoI and ScoII proteins (10) believed to be involved in the assembly of mitochondrial cytochrome *c* oxidase subunits. PrrC appears to be membrane localized, based on *prrC′-′phoA* fusion analyses (21), with the carboxy-terminal region of the protein located in the periplasmic space.

(iii) AppA. The *appA* (activation of photopigment and *puc* expression) gene resides at approximately **1285 kbp** clockwise on chromosome I; i.e., approximately 1.2 Mb from the PS gene cluster (28). Deletion of *appA* results in greatly diminished SC formation, which correlates with decreased expression of the corresponding PS genes (28) (Fig. 1). Low levels of SC result in a prolonged lag in the growth of an AppA null mutant when it is shifted from aerobic to anaerobic light conditions, and this is followed by reduced photosynthetic growth rates (31). However, anaerobic dark growth is normal (31).

AppA consists of 450 amino acids and shows no homology with proteins of known function. Following overexpression of several truncated forms of the protein, specific ligand binding has been observed. The approximately 120-residue amino-terminal domain has been shown to bind flavin. The approximately 70-residue carboxy-terminal domain contains an unusual cysteine-rich motif which is capable of binding iron or, more likely, an Fe:S center. The central region of AppA appears to bind a heme. Removal of either the amino- or carboxy-terminal portion of AppA result in only slight impairment of AppA function, as judged by complementation of an AppA null mutant (32).

The activity associated with AppA is currently unknown. However, AppA affects PS gene expression independently of both the Prr and FnrL systems (31). Existing data strongly suggest that AppA antagonizes PpsR repressor activity (31) (see below). Whether the role of AppA is limited to PpsR alone has yet to be elucidated.

DNA-binding factors. (i) Anaerobiosis activator/repressor FnrL. The *fnrL* gene encodes the *R. sphaeroides* 2.4.1 homolog of Fnr (Fig. 1), the *E. coli* anaerobic regulatory protein. It is located at approximately **443 kbp** on chromosome I, in close proximity to the *ccoNOQP* and *rdxBHIS* operons of *R. sphaeroides* 2.4.1 (94). $FnrL^-$ mutant strains are unable to grow anaerobically, either photosynthetically or in the dark using DMSO as an electron acceptor, and such mutants have no detectable SCs under conditions of ICM induction (94).

FnrL has 11 of 22 amino acid identities with *E. coli* Fnr within the region corresponding to the helix-turn-helix DNA-binding domain. Residues that are thought to form direct amino acid-base contacts (54) are identical between the two. Therefore, we suggest that the recognition sequence for binding by FnrL resembles the FNR (fumarate nitrate regulator) consensus sequence, TTGAT-N_4-ATCAA. Sequences similar or identical to this consensus sequence have been identified upstream of several genes involved in tetrapyrrole and Bch1 biosynthesis, including *hemA* (68), *hemZ* (94), *hemF* (recently renamed *hemN*) (18) and *bchE* (28, 95), as well as the *puc* operon (56). Under conditions of reduced oxygen tension, FnrL activates expression of *hemA* (94) and the *puc* operon (95). These results provide good evidence that the target sequence for regulation by FnrL is the FNR consensus sequence.

In contrast to *R. sphaeroides*, an FnrL mutant strain of *R. capsulatus* which has been shown to contain an *fnrL* homolog is unaffected in its ability to grow photosynthetically with many of the genes described above, lacking upstream FNR-binding sequences (92). However, like *R. sphaeroides* FnrL mutant strains, an FnrL mutant of *R. capsulatus* is incapable of anaerobic growth in the dark with DMSO (92). For both *R. sphaeroides* and *R. capsulatus*, the DMSO-inducible cytochrome *c* (encoded by *dorC*) and DMSO reductase, encoded by *dorA* (65), are not induced in the mutant strains (93). It appears that the role of FnrL in *dor* operon (65) expression is mediated through the FnrL-dependent regulation of *dorS*. DorS, which encodes a redox-responsive sensor histidine kinase, regulates its cognate transducer, the product of the *dorR* gene (65).

Based on the presence of upstream FNR consensus sequences (94), the *ccoNOQP* operon, the *rdxBHIS* operon, and the *ctaD* and *ctaABC* genes (24, 36) encoding subunits of the aa_3 cytochrome oxidase are likely to be regulated, in whole or part, by FnrL in *R. sphaeroides* (94). Thus, FnrL could, by

affecting the abundance of these redox carriers, control the flow of electrons through the respective terminal oxidases and the Rdx redox center. This has been shown to be true for the *ccoN* gene. In addition to directly effecting *puc* operon transcription due to the presence of an upstream FNR-binding sequence (95), FnrL could also indirectly affect *puc* operon transcription (95, 96) through its regulation of *ccoNOQP* and, thus, the signal reaching Prr (71).

(ii) PpsR. The upstream regions of many *bch* and *crt* genes and operons, as well as the *puc* operon from both *R. sphaeroides* and *R. capsulatus*, contain the same dyad symmetry motif (Fig. 1), TGT-N_{12}-ACA (52, 56, 61). The localization of this motif downstream of, or overlapping with, putative σ^{70}-type promoters of PS genes and studies of the *cis* regulatory elements of the *puc* operon (56, 59) suggest that this sequence can function as a repressor-binding site. This repressor-encoding gene, designated *ppsR* (photopigment suppression) in *R. sphaeroides* (73; Fig. 1), was identified because of its ability to suppress photopigment production in wild-type cells when provided in extra copy. Inactivation of *ppsR* results in constitutive expression of both the photopigment genes and *puc*, which triggers production of SCs, even under aerobic conditions, thus making such mutants genetically unstable under these conditions (31, 74).

ppsR is localized within the region of PS genes, at approximately **26 kbp** on chromosome I. PpsR contains a putative helix-turn-helix motif at the carboxy terminus which resembles those found in several known DNA-binding transcription factors (74). The ability of PpsR to act as a repressor has been demonstrated genetically in *E. coli* (74) and in *Paracoccus denitrificans* (29). Point mutations in the putative DNA-binding region result in decreased repression (31, 56, 59). It has been shown that the PpsR protein overexpressed in *E. coli* and *P. denitrificans* specifically retards DNA fragments containing the sequence TGT-N_{12}-ACA (32). The PpsR homolog from *R. capsulatus*, designated CrtJ, has been purified recently and shown to bind to this motif as a dimer (75). Cooperative binding of CrtJ to two binding motifs positioned in the regulatory regions of several putative CrtJ/PpsR target genes, has also been indicated, which correlates well with earlier genetic studies (29).

R. sphaeroides ppsR gene expression is largely independent of growth conditions, and PpsR repressor activity does not respond directly to oxygen deprivation. Therefore, other cellular factors are required to communicate to PpsR the state of oxygen availability and changes in growth conditions (31, 32). Further, PpsR functions as a repressor not only under aerobic but also under anaerobic photosynthetic conditions, where it regulates LHII SC abundance, depending on light intensity (31). Because it is unlikely that PpsR senses both oxygen and light directly, we believe that PpsR must respond to an integral signal generated by changes in either of these environmental stimuli, e.g., either redox poise or a redox carrier. This view is complemented by the in vitro analysis of the *R. capsulatus* repressor protein, whose DNA-binding capacity has been shown to depend on redox-active agents (75).

The nature and regulation of the redox sensitivity of PpsR remain largely unknown (Fig. 1). Disruption of the *ppsR* gene in an AppA null mutant background completely overcomes the impairment(s) imposed by the *appA* null mutation. Furthermore, a large number of suppressors of the *appA* null mutation, showing improved photosynthetic growth, contain point mutations in *ppsR*, and partial suppression of the *appA* null mutation can be achieved by titration of PpsR in vivo (31, 32). AppA significantly decreases PpsR-mediated repression when these genes are simultaneously expressed in *P. denitrificans* (31). Therefore, we believe that AppA serves to regulate PpsR repressor activity by communicating a redox signal to PpsR which is ultimately derived from oxygen- and/or light-generated redox responses. The signal could affect either the redox state of PpsR or its dimerization or both. The overall role of PpsR in both oxygen and light control of SC formation, as well as its specificity toward PS genes, makes it a major player in PS gene regulation.

(iii) Spb. The *hvrA* gene of *R. capsulatus*, located immediately downstream of the response regulator *regA*, is involved in regulation of RC and LHI SC gene expression (12). The *hvrA* gene encodes a small, 104-residue DNA-binding factor. Analogous to *hvrA*, a homolog from *R. sphaeroides*, termed *spb*, is positioned immediately downstream of *prrA* (77). Both Spb (*Sphaeroides puf*-binding protein) and HvrA bind upstream of the *puhA* gene and the *puf* operon under various conditions (49, 80). The amino-terminal regions of Spb and HvrA bear similarity to the HU-like proteins from *E. coli* and bacteriophages (49, 77). These proteins often have multiple targets and therefore are involved in transcriptional regulation of a variety of processes. Conforming with this idea are recent observations that HvrA is also involved in the ammonium regulation of nitrogen fixation in *R. capsulatus* (62). Thus, these proteins may be involved in PS gene expression by facilitating the binding of other transcription factors, e.g., PrrA, RNA polymerase, etc., and their role in light regulation may be a reflection of their abundance as a function of light intensity. It was also found that the amino terminus of Spb has some homology to the leucine zipper motif of the eukaryotic transcription factor c-JUN. This motif is proposed to play a role in protein dimerization (80). Because it is difficult to describe a more specific role for these factors, they have not been included in Fig. 1.

(iv) IHF. The integration host factor (IHF) of *E. coli* is a global regulatory protein which acts by binding DNA targets and bending the DNA so that looping occurs. This brings together noncontiguous regions of the DNA, which interact through DNA-binding regulatory proteins (88). The genes for the two subunits of the *R. sphaeroides* IHF heterodimer have been cloned (78). Interestingly, the *himA* gene is located at approximately position **1760 kbp** on chromosome I, while the *himD* (*hip*) gene is located at approximately **830 kbp** on chromosome II. In vitro studies have shown that IHF of *E. coli* binds to the regulatory region of the *puc* operon (58). Additionally, mutations that alter the consensus IHF-binding site in this region abolish binding in vitro and negatively affect *puc* expression in vivo.

Modulators of PS gene transcription. The *tspO* gene (formerly *crtK*, or *orf160* in *R. capsulatus*), located within the *crt* gene cluster on chromosome I, encodes a 17-kDa protein (tryptophan-rich sensory protein) which is characterized by an unusually high content of aromatic amino acids, especially L-tryptophan. TspO (Fig. 1) has been localized to the outer membrane of *R. sphaeroides* 2.4.1 (90) and is present at severalfold higher levels in photosynthetic than in aerobically grown cells. Results of cross-linking studies suggest that TspO may form a homodimer within the bacterial membrane (91).

A TspO mutant has higher levels of Crt and Bchl than do wild-type cells when strains are grown aerobically or semiaerobically. The effect of TspO on pigment production occurs at the level of transcription of those same *crt* and *bch* biosynthetic genes which constitute the PpsR regulon (90). TspO also negatively affects the expression of the *puc* operon under both aerobic and photosynthetic conditions but has no effect on the expression of the *puf* operon. The available evidence implicates TspO as a both oxygen- and light-responsive modulator of PS gene expression with the same spectrum of regulatory

targets as the PpsR repressor (29, 90). How a signal is transmitted from outer membrane-localized TspO to the cell interior is unknown. However, the quantitative effect of TspO upon PS gene expression is not as strong as that of PpsR, which suggests that there must be other elements or factors interposed between TspO and the PpsR regulon.

Useful information about the physiological activity of TspO may be drawn from studies on its mammalian homolog (91), the 18-kDa outer membrane component (pk18) of the mitochondrial peripheral benzodiazepine receptor (MBR). Although the precise biological role of MBR is not yet understood, this receptor was shown to be involved in the regulation of steroidogenesis, heme biosynthesis, mitochondrial respiratory control, and other metabolic activities (51). The rat pk18 gene, when expressed in *R. sphaeroides* in the TspO$^-$ background, functionally substituted for the bacterial homolog, negatively affecting the expression of *crtI* and *puc*. Additionally, when the pk18 gene was expressed in *R. sphaeroides*, it retained its quantitative binding properties towards a variety of MBR ligands. These results suggest that TspO is both evolutionarily and functionally related to the mammalian MBR and indicate the possibility that in both bacterial and mammalian cells, these proteins are involved in similar metabolic activities (91).

How TspO might "sense" and "transmit" an oxygen- or light-generated signal remains a mystery, although no ligand has been found to be associated with TspO. Very recent results suggest that TspO may be involved in regulating the entry of specific small molecules through the outer membrane in response to oxygen and light. Mutants with single changes of the five conserved tryptophans have either lost or retain partial TspO activity.

Other elements in PS gene expression. Several additional factors have been observed to effect PS gene expression, but their precise role is presently unknown. The *ppa* (photopigment activation) gene (also known as *ppsS* and *orf192*) is positioned immediately upstream of *ppsR* (29). A role for Ppa in controlling PpsR repressor activity has been proposed but not yet tested (74). Thus, we have not included Ppa in Fig. 1. A locus at **2110 kbp** on chromosome I, identified by virtue of its effect on expression of the *puc* operon (76), has been named *mgpS* (modulator of genes for photosynthesis) and contains an open reading frame of ~2.8 kb. The N-terminal portion of *mpgS* shares similarity with a number of RNA helicase proteins which are involved in RNA modifications (76), suggesting that *mpgS* may have an additional role in posttranscriptional regulation of PS gene expression. The *rpgS1* gene (regulator for photosystem genes; 77) a histonelike protein located approximately **500 bp** clockwise from *prrA*, encodes a protein with similarity to the functionally unidentified *orf5* gene from *R. capsulatus* (13). The *rpgS1* gene is predicted to encode a protein of 187 amino acids which contains a putative helix-turn-helix motif located at its amino-terminal end (77).

Regulation of PS gene expression at the message level. As is evident from the previous sections, transcriptional regulation of PS gene expression plays a crucial role in PS formation in *R. sphaeroides* 2.4.1. However, it has been observed that mRNAs corresponding to the structural polypeptides of the SCs are produced in excess, relative to the actual levels of the complexes present under a variety of growth conditions (34, 55, 67, 81). This provides evidence of posttranscriptional regulation. Such complexity of regulation is not surprising, given the fact that PS development requires coordination of a variety of diverse processes, involving protein and pigment biosynthetic pathways, SC assembly, and adaptation between alternative cellular steady states.

In the related organism *R. capsulatus*, PS genes are organized into superoperons (reviewed in references 5, 6, and 48). It is thought that there are multiple transcription initiations at several start sites within the superoperons which result in differences in message levels for genes and operons within the superoperons. However, degradation of larger transcripts is also considered to contribute to the disproportionate representation of gene messages in the cell (reviewed in reference 48). Some data also suggest the possibility that degradation of large transcripts is regulated by oxygen tension (46, 47).

While certain large transcripts indicative of a similar superoperon organization have been detected in *R. sphaeroides* (63), their physiological role, if any, is not known. In the case of the *puc* and *puf* operons of *R. sphaeroides*, multiple transcripts with differing half-lives are also detected. The longer *puc* and *puf* transcripts have been shown to be products of transcriptional readthrough of terminatorlike sequences within those operons (55, 57).

The first gene of the *R. sphaeroides puf* operon is the *pufK* gene, encoding a 20-amino-acid-residue polypeptide. It has been demonstrated that *pufK* is translated and that its expression is elevated severalfold under high-light PS conditions, compared to that in aerobically grown cells (33). An interesting feature of the *pufK* gene is the high occurrence of rare codons, with 9 of the 20 codons of *pufK* among those rarely used by *R. sphaeroides* 2.4.1. Studies suggest that translation through these rarely used codons serves to "gate" the entry of ribosomes to the immediate downstream *pufBA* cistrons (Fig. 1) and, as such, can affect the cellular abundance of the α and β polypeptides of the LHI complex (33).

THE ROLE OF BCHL AND CRT

Bchl and Crt are major nonprotein structural components of the SCs whose synthesis is regulated in response to oxygen availability and/or light intensity (Fig. 1). The availability of Bchl has been shown to be of critical importance and the limiting component for the formation and stability of the RC and the LH SCs (87). The *puf*-encoded RC-M and RC-L polypeptides, as well as the *puhA*-encoded RC-H protein, are detected at low levels under aerobic growth conditions but are rapidly degraded due to the absence of Bchl, both in *R. sphaeroides* and in *R. capsulatus* (16, 84, 85, 87).

Unlike Bchl, Crt are not essential components of the RC or the B875 complex, and mutants defective in Crt biosynthesis genes are photosynthetically competent. In contrast, the formation of the LHII complex requires colored Crt (intermediate products of the Crt biosynthetic pathway downstream of neurosporene), and it has been proposed that this requirement may be at the level of assembly (53). The type of Crt accumulated is also important in determining the relative stoichiometry between the two LH complexes. Spheroidenone (SO), the end product of the Crt biosynthetic pathway, is predominantly associated with the RC and LHI complexes, whereas spheroidene (SE), the penultimate product of the Crt pathway, is more abundant in the LHII complex (38, 89). The relative amounts of these two major Crt pigments are regulated in response to oxygen tension and/or light intensity, apparently through the localized cellular redox state (Fig. 1), which affects the CrtA-catalyzed conversion of SE to SO (89).

The Cco/RdxB putative oxygen-sensing, redox signal-generating pathway has been demonstrated to affect the regulation, through the Prr regulon (71), of the type of Crt accumulated under photosynthetic and diazotrophic growth conditions. Presumably, the localized redox poise generated through these pathways affects CrtA activity (70). Thus, the signal generated

by the cbb_3 oxidase-RdxB system is transferred not only to transcription factors through the Prr pathway (see the section on sensors, signal generators, and transducers) but also to another pathway(s) involved in the control of the conversion of SE to SO under photosynthetic conditions. The recently discovered gene likely to encode the first enzyme in the isoprenoid biosynthetic pathway (82) and the next downstream gene in the biosynthetic pathway, mapping to the PS gene cluster, are of major importance and are predicted to be highly regulated. These data provide further evidence that Cco and RdxB are "functional" as redox carriers under anaerobic, as well as aerobic, growth conditions, perhaps interacting with a light-generated redox intermediate.

Conditions which limit Bchl availability (such as high light illumination or high oxygen tension) are also characterized by low SE and high SO levels and correspond to limited LHII formation (89). Therefore, the net result is that available Bchl is incorporated into the RC and LHI complexes (Fig. 1), which appear to have no preference for which Crt is used in the assembly process (Fig. 1). In this way, changes in environmental conditions (to which Bchl levels and Crt accumulation are evidently sensitive) can be rapidly reflected by changes in LHII complex formation and this, in turn, may result in the de facto regulation of RC and LHI complex assembly and ultimate abundance (89).

ASSEMBLY FACTORS

Genes have been identified whose proposed role in the regulation of PS formation is at the level of SC assembly (Fig. 1). One of these genes is the monocistronic *Q* gene, located immediately upstream of the *puf* operon of *R. sphaeroides*, encoding a 187-amino-acid polypeptide (34). Single amino acid substitutions in the *Q* gene product have dramatic consequences for the formation of SCs, e.g., loss of LHI complex formation ability for one group of mutants, loss of LHII complex formation ability for another group, and altered levels of both SCs relative to those in wild-type cells for a third group, despite little or no changes in specific mRNA levels or Bchl and Crt availability. In the absence of the *Q* gene product, the cells are unable to grow under PS conditions. Based on these studies (34), the *Q* gene product is proposed to be involved in the assembly of the SCs. However, in *R. capsulatus*, the structure of the *Q* gene and the function of its product have been interpreted differently (7).

orf1696, located immediately upstream of *puhA* (14) of *R. sphaeroides* 2.4.1 (Fig. 1), has been shown to be required for LHI complex formation (81). The *pucC* gene product (Fig. 1) is required for LHII complex formation (43, 57, 85). These genes are proposed to encode complex-specific assembly factors (CSAFs). These two factors, together with the product of a third gene, which is called *orf428* in *R. capsulatus* (14) but is not known to participate in SC formation (8), are similar to each other. This similarity may indicate common interactions with other factors, as part of an overall assembly hierarchy for the SCs. One of these interactions may be with the *Q* gene product (81). Thus, the specific role of the *Q* gene product is thought to be through the insertion of Bchl into the developing SCs (Fig. 1) in a process involving interaction(s) with the CSAFs (Fig. 1) (34). The difference in the affinities of the Q polypeptide for the CSAFs would then account for the preferential assembly of the RC complex, followed by the LHI complex, and finally the LHII complex when Bchl is plentiful.

Some of the more conventional chaperone genes have also been identified in *R. sphaeroides*; e.g., there are two *groE* operons (60). However, the role (if any) of these and/or other chaperones in SC formation has not yet been defined.

OVERALL MODEL FOR THE REGULATION OF SC FORMATION

In the presence of oxygen, the *R. sphaeroides* cell membrane resembles that of other gram-negative bacteria. Energy for cellular metabolism is derived from aerobic respiratory chains, terminating at either one of two cytochrome *c* oxidases (the low oxygen affinity aa_3 oxidase or the high oxygen affinity cbb_3 oxidase or a bb_3-type quinol oxidase (reviewed in reference 26). There is virtually no Bchl or Crt synthesized. There are low but detectable levels of transcription of the PS genes encoding the apoproteins of the SCs; in the absence of Bchl, these polypeptides turn over rapidly, although they are transported to the cell membrane and assume their proper topological orientation within the membrane.

When oxygen tension is reduced, transcription of PS genes is activated in response to three key regulatory systems, namely, the FnrL, Prr, and PpsR systems. We propose that these systems reflect change in the localized cellular redox poise or a critical redox intermediate(s) when oxygen tension is lowered. Yeliseev and Kaplan have demonstrated that TspO modulates this response (90). Similarly, the biosynthesis of Bchl and Crt ensues in response to these changes and SC formation can now occur.

The state of activation of the putative oxygen-labile [4Fe-4S] center of FnrL as a function of oxygen availability is thought to regulate the activity of FnrL. FnrL can act directly to activate specific PS gene transcriptions, which reflect critical points of control, in the tetrapyrrole and Bchl pathways, as well as *puc* operon expression. FnrL also affects PS gene expression through its regulation either positively or negatively of specific redox chains, e.g., cbb_3 or aa_3.

The Prr two-component regulatory system, in response to an altered signal emanating from the cbb_3/RdxB redox pathway can begin to activate transcription of all or, certainly, most of the PS genes. Presumably, PrrB is no longer inhibited by this signal, to which it responds by activating PrrA in a mechanism believed to involve PrrB autophosphorylation and subsequent phosphorylation of PrrA. While the means by which PrrA regulates transcription of the PS genes is not precisely known, it may involve direct DNA binding or the formation of transcriptional complexes involving other protein factors, the net result being an increased expression of genes encoding all of the structural components of the SCs. The data also suggest that different levels of activated PrrA may be required to activate different subsets of PS genes.

In contrast to FnrL and Prr, which have a broad spectrum of gene targets, PpsR appears to repress specifically PS genes; i.e., most of the *bch* and *crt* genes, as well as the *puc* operon, which contain the repressor-binding sequence TGT-N_{12}-ACA. Repression by PpsR, although relieved under anaerobic conditions, is still responsive to light under these conditions, and derepression is brought about, at least in part, by the action of the flavohemoprotein AppA, which acts either directly or indirectly on PpsR. The fact that PpsR is involved in PS gene control in response to both oxygen and light, perhaps through AppA, suggests that a different source of a redox-generated signal is involved in the transmission of this effect than in Prr-mediated regulation. However, this remains to be proven.

TspO modulates the rate of activation of PS gene derepression as oxygen tensions are decreased. Other modulators appear to include PrrC, Ppa, HvrA (Spb), and MgpS, as well as IHF. Overall, as the O_2 tension decreases, there develops a

cascade of both orderly and progressive events which serve to reinforce the induction of PS gene expression. This cascade must also involve subtle interactions between the system of activation and repression of PS gene expression, as oxygen tensions change and as light intensities vary under anaerobic conditions.

The net result of these transcriptional changes is that all of the components leading to SC formation are present at increased levels when oxygen tension is reduced, and the assembly process specific to each can take place. The total amounts of SC formed depend upon the cellular level of Bchl which, in turn, is determined not only by the activity of Bchl biosynthetic enzymes but also by the available pool of 5-aminolevulinic acid, and therefore, Bchl serves as the ultimate "governor" regulating cellular abundances of SCs. The Q protein, together with CSAFs, direct the flow of Bchl into the assembling SCs in a hierarchical fashion, with RC assembly first, followed by assembly of the B875 SC, and then assembly of the B800-850 SC. Usually, the mRNA levels, and the levels of apoproteins produced, are in excess of cellular needs. The abundance and composition (SE and SO) of the Crt present are also important for the ratio of LH complexes. Thus, posttranscriptional regulatory pathways serve to increase the ability of cells to respond rapidly to changes in oxygen availability and light intensity.

Although it has long been known that the amount of SC varies dramatically under anaerobic conditions, depending on the light intensity, the mechanism(s) of light control of PS formation is less well understood than that of oxygen control. We do know, however, that light regulation is achieved, at least in part, through the same transcriptional regulatory machinery which operates in response to changes in oxygen tension, e.g., PpsR/AppA (31) and HvrA (Spb) (49). It is also tempting to speculate that cbb_3 and RdxB play some role(s), as well, in photosynthetically growing cells. This lends support to the idea that changes in light intensity are first transformed into changes in the cellular redox state. However, the presence of specific light-dependent regulators cannot be ruled out at present. We currently believe that light control of PS formation occurs mostly at posttranscriptional levels and involves rapid changes in Bchl levels and the cellular levels of SO and SE. Together with the available assembly factors, these elements will rapidly determine the abundance and kinds of SCs.

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ADDENDUM IN PROOF

We have recently observed that the *ccoNOQP* operon is negatively regulated by the two-component Prr system under aerobic growth conditions.

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