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NOTES

Phototaxis and Impaired Motility in Adenylyl Cyclase and Cyclase Receptor Protein Mutants of *Synechocystis* sp. Strain PCC 6803

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We have carefully characterized and reexamined the motility and phototactic responses of *Synechocystis* sp. adenylyl cyclase (Cya1) and catabolite activator protein (SYCRP1) mutants to different light regimens, glucose, 3-(3,4-dichlorophenyl)-1,1-dimethylurea, and cyclic AMP. We find that contrary to earlier reports, *cya1* and *sycrp1* mutants are motile and phototactic but are impaired in one particular phase of phototaxis in comparison with wild-type *Synechocystis* sp.

In a screen designed to identify transposon-tagged motility mutants in *Synechocystis* sp. strain PCC 6803 (hereafter *Synechocystis* sp.), we identified a transposon insertion in the gene encoding adenylyl cyclase (*cya1* or slr1991) which caused an apparent nonmotile phenotype (5). Adenylate cyclase (AC) synthesizes 3′,5′-cyclic AMP (cAMP) from ATP. cAMP is a ubiquitous second messenger that participates in a wide variety of signal transduction systems in bacteria and in eukaryotes (1, 2). cAMP binds to a dimer of the catabolite receptor protein (CRP; also known as catabolite activator protein) which requires the allosteric effector cAMP in order to bind efficiently to DNA. In *Escherichia coli* CRP activates transcription at more than 100 promoters, by binding to a well-conserved palindromic binding motif (TGTGAN$_6$TCACA). In *Synechocystis* sp. inactivation of CRP (*sycrp1*, sll1371) resulted in an apparently nonmotile phenotype (27, 28).

Six classes of adenylyl cyclases (I to VI) with structurally unique catalytic domains are found in prokaryotes (1, 8, 18). Of these only the class III universal class of ACs is found among both prokaryotes and eukaryotes (21). All cyanobacterial ACs share homology with the catalytic domain of eukaryotic adenylate cyclases but often have other functional domains fused at either the C or the N terminus of the protein and are therefore likely to be multifunctional (17, 18). Cya1 (encoded by *cya1*) is the major class III AC in *Synechocystis* sp.; *cya3* (sll1161) encodes an AC-like product but lacks several critical residues, suggesting that it may not be functional; *cya2* (sll0646) encodes a guanylyl cyclase (15, 16). *sycrp1* (sll1371) and *sycrp2* (sll1924) encode cAMP receptor-like proteins in *Synechocystis* which are homologous to each other, although Sycrp2 lacks several residues required for cAMP binding (26).

Anabaena cylindrica and *Synechocystis* sp. exhibit rapid changes in cAMP levels triggered by different light qualities, including UV-A and UV-B (7, 12, 13, 18). When *Synechocystis* sp. is exposed to white light (100 μmol photon m^{-2} s^{-1}) after incubation in the dark for 40 min, it shows a rapid increase in intracellular cAMP content. cAMP levels increased in response to blue light (450 nm) and UV-A light (380 nm), but no other wavelengths (520, 575, 670, or 720 nm) induced this response. Furthermore, 63% of the cells were motile under blue light versus 24% in red light, suggesting that cAMP might mediate blue light signals, and it has been suggested that a BLUF domain-containing protein encoded by slr1694 may be involved in cAMP-mediated blue light signal transduction (23). Inactivation of this protein abolished positive phototaxis (19), but cAMP levels in the mutants were the same as in the wild type (WT) under specific light regimens (13).

To characterize phototaxis and motility in *Synechocystis* sp., WT cells were spotted on motility plates and grown in directional white light (5). Based on the motility behavior of individual cells, we identified three phases of motility. In phase 1, we observed single cells or cells in small groups on the agarose surface which exhibit limited motility and phototaxis (Fig. 1A and D). In the next observable phase (phase 2), which usually occurred between 16 and 48 h after the cells were spotted, cells had aggregated into larger groups which clearly exhibited phototaxis (Fig. 1B and E). Cells nearest the light source had moved and accumulated at the front edge of the spot, forming a crescent shape. Cells at the back or the center of the spot also exhibited phototaxis. In phase 3, fingerlike projections or a moving front of cells extended from the front edge of the spot toward the light source, and within these projections cells moved rapidly (Fig. 1C). Within 2 to 4 days after spotting, the characteristic fingerlike projections extended ~4 to 10 mm, depending on light intensities and the degree of surface wetness of the plate. The rates of movement of cells and the gross morphology of the moving front of cells appeared to be a function of cell density and cell doubling times, surface wetness, and light quality.

Spectinomycin-resistant strains with targeted mutations of *cya1* and *sycrp1* were created using a standard PCR-based inactivation strategy. To inactivate *cya1* (slr1991) and *sycrp1*

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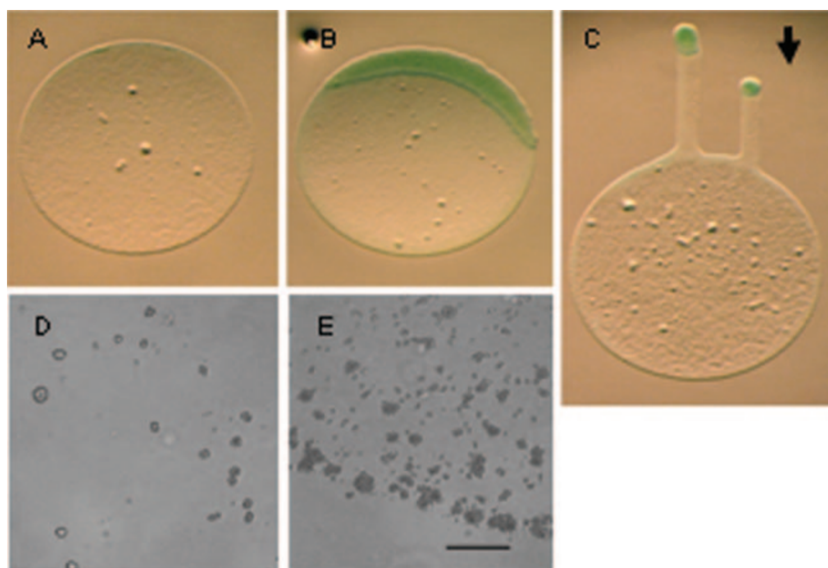

FIG. 1. Phases of phototaxis in *Synechocystis* sp. cells. Cells were spotted on motility plates, placed in unidirectional white light, and photographed. After overnight growth, cells are either single or in small groups; the faint green edge visible at the front of the spot is caused by cells that have moved and accumulated at the front edge (A and D); after 2 days (B and E), many more cells are in groups and have migrated to the front of the spot, typical of phase 2; after 4 days (C), fingerlike projections of motile cells are seen, typical of phase 3. Lower panels: single cells (D) and cell groups (E) from spots in panels A and B are shown at higher magnification. Motility plates contained 0.4% agarose in BG-11 and 10 mM glucose. The arrow shows the direction of the light. Spots are ~3 to 4 mm in diameter. Bar, 10 μm.

(sll1371), genes were amplified from genomic DNA using primers (forward primer 5′ ATGGGCACTAGTCCCCAA 3′ and reverse primer 5′ TCAAGGAAATTAGATCTT 3′ for sll1371 and forward primer 5′ GTGGATAAGCCTGCCCTA 3′ and reverse primer 5′ TTAGGGCCCTTCCGAGGC 3′ for slr1991). PCR products were purified and ligated into pGEM T Easy vector (Promega). The spectinomycin cassette (digested with SmaI) from plasmid HPΩ45 was ligated into the unique SmaI site in sll1371. The resulting plasmid was used for the transformation of *Synechocystis* sp. The plasmid containing the 1-kb slr1991 insert was digested with SalI and blunted, and the spectinomycin cassette (digested with SmaI) was ligated into the site. Colony PCR was used to assess whether complete segregation had been achieved, and these transformants were checked for motility (4).

The *cya1* and *sycrp1* mutants behaved like WT cells in phase 1 and phase 2 of motility. However, neither the *cya1* nor the *sycrp1* mutants made the typical fingerlike projections characteristic of phase 3 (Fig. 2A). Thus, in the *cya1* and *sycrp1* mutants the cells moved and accumulated at the front of the spot, making a crescent shape, but movement was retarded beyond this step. The *cya1* mutant cells were evenly distributed at day 2 (Fig. 2C) but had migrated to the front of the spot by day 4 (Fig. 2D). Even after several days, the *cya1* and *sycrp1* mutant cells did not form fingers, suggesting that they were phototactic but defective in phase 3 motility. As cells continued to grow and divide, they became confluent, giving the appearance of a nonmotile colony (i.e., they do not make characteristic fingers associated with WT cells), which explains why these mutants were initially characterized as being "nonmotile" (5, 22).

In the presence of exogenously added 0.1 mM cAMP, *cya1* mutant cells proceeded to phase 3 of motility and regained wild-type levels of motility and phototaxis; however, the *sycrp1* mutant did not regain phase 3 motility (Fig. 2B). Even higher concentrations of cAMP did not rescue the *sycrp1* mutant (data not shown), suggesting that in the absence of the receptor protein, the exogenous addition of cAMP has no effect on

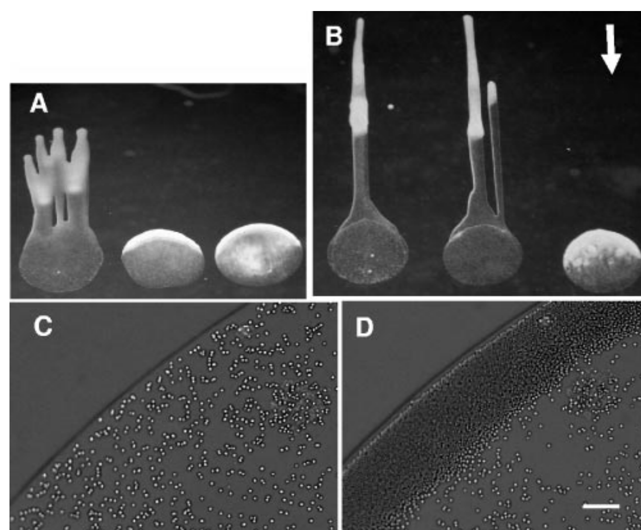

FIG. 2. Effect of cAMP on phototaxis of *Synechocystis* sp. WT and *cya1* and *sycrp1* mutant cells. Upper panels: WT (left), *cya1* mutant (middle), and *sycrp1* mutant (right) cells were spotted on 0.4% agarose BG-11 motility (no glucose added) plates in the absence (A) or presence (B) of 0.1 mM cAMP and photographed after 4 days. Lower panels: the front edge of the spot containing *cya1* mutant cells is shown after day 2 (C) and day 4 (D). Note that cells are evenly distributed at day 2, but after day 4, cells have clearly accumulated at the front. In the presence of 0.1 mM cAMP (B), *cya1* mutant cells shows the typical fingerlike projections seen in WT cells; *sycrp1* mutant cells do not respond to cAMP. The arrow shows the direction of the light. Spots are ~3 to 4 mm in diameter. Bar, 10 μm.

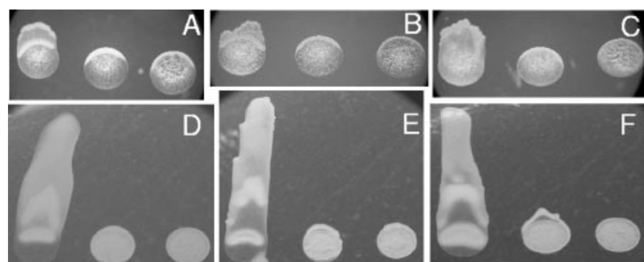

FIG. 3. Glucose effect on motility of *Synechocystis* sp. WT and *cya1* and *sycrp1* mutant cells. WT (left), *cya1* mutant (middle), and *sycrp1* mutant (right) cells were spotted on 0.4% agarose motility plates containing no glucose (A) or 0.1 mM (B), 0.2 mM (C), 1 mM (D), 5 mM (E), or 10 mM (F) glucose. Plates were placed in directional white light and photographed after 4 days. Note that *cya1* mutant cells show slight formation of fingerlike projections in 10 mM glucose, but *sycrp1* mutant cells do not.

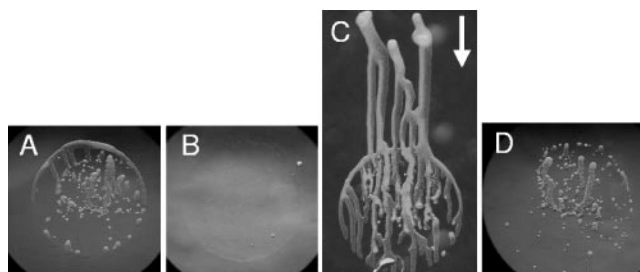

FIG. 4. Motility in the presence of glucose and DCMU. *Synechocystis* sp. WT cells were spotted on 0.4% agarose BG-11 plates (A) or plates containing 10 μM DCMU (B), 10 mM glucose (C), or 10 mM glucose and 10 μM DCMU (D). Spots were photographed after 4 days. After 4 days the cells growing in DCMU have not divided and cannot be easily seen at this magnification. Spot diameter is ~4 mm.

motility. If the action of cAMP is due solely to its role as an allosteric effector working by binding to Sycrp1, this result is to be expected. cAMP levels in *Synechocystis* cells have been measured between 600 and 1,000 pmol cAMP mg Chl^{-1} when cells are transferred to blue light from darkness (13, 23). Cellular cAMP content in the *cya1* mutant cells was less than 5% of that in wild-type cells (22).

We checked the effect of glucose on phototaxis, since glucose has a marked effect on cAMP levels in *E. coli* and it is known that glucose can support photoheterotrophic growth in *Synechocystis* sp. (6, 29, 30). The addition of increasing concentrations of glucose to the solid medium strongly affected motility (Fig. 3). In the absence of glucose, WT cells moved as a front, while *cya1* and *sycrp1* mutant cells accumulated at the front of the spot as described above. With increasing concentrations of glucose (ranging from 0.1 mM to 1 mM) there were progressively longer projections of motile cells representative of phototaxis. This rapid phototaxis is likely to be caused by a combination of factors including an increased cell density (because of a faster cell doubling time in the presence of glucose) and perhaps a specific effect, such as the enhanced production of extracellular material, which assists cell motility.

The *cya 1* and *sycrp1* mutants appeared to have an enhanced cell density in the presence of increasing glucose concentrations, and at 5 and 10 mM glucose there is a modest increase in forward motility but the *cya1* mutant cells still do not make the fingerlike projections typically observed in WT cells. Thus, addition of glucose alone could not compensate for the absence of cAMP under these conditions. To separate the possible effects of glucose on growth from a specific effect on phototaxis, we checked the effect of 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) on phototaxis (24). DCMU is a specific inhibitor of photosynthetic activity, and cell growth is strongly inhibited in the presence of 10 μM DCMU. We found that in the presence of DCMU, cells did not divide and remained as single cells on the plate (Fig. 4A); however, in the presence of 10 mM glucose and 10 μM DCMU (Fig. 4D) some of the typical fingerlike projections indicative of phototaxis did appear, indicating that glycolysis can provide some of the energy (ATP) required for cell division and phototaxis. We observed maximal phototaxis in the presence of 10 mM glucose and white light (Fig. 4C); in the absence of glucose, slower cell growth and possibly the lack of other factors, such as polysaccharide production (10), that enhance motility may reduce the extent to which cells move (Fig. 4B). Maximal rates of motility were observed when there were both active photosynthesis and energy provided from the respiration of exogenously added glucose. Glucose is phosphorylated by a glucose kinase (sll0593) and enters the oxidative pentose phosphate pathway, glycolysis, and the tricarboxylic acid cycle to produce ATP, NADPH, and carbon skeletons for growth. Doubling times of *Synechocystis* in the presence of 10 mM glucose and light (i.e., photomixotrophic conditions) were 8.2 h compared to 22.2 h under photautotrophic conditions (9, 20).

We have previously shown that red light is effective even at a fluence of about 1 μmol photon m^{-2} s^{-1} for phototaxis, although rates of phototaxis were enhanced up to 40 μmol photon m^{-2} s^{-1} (14). For the photomotility assays, light intensity was set between 6 and 10 μmol photons m^{-2} s^{-1} using light-emitting diode light sources (Roithner Lasertechnik, Austria, or SuperbrightLEDs.com). Light-emitting diodes were white (bimodal with a sharp peak at 465 nm and a broader peak between 559 and 587 nm), red (peak at 636 nm), green (528 nm), or blue (464 nm).

The motilities of *cya1* and *sycrp1* mutants and WT cells were

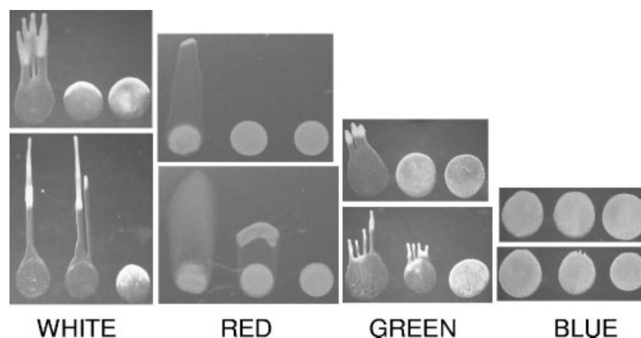

FIG. 5. Motility of *Synechocystis* sp. WT and *cya1* and *sycrp1* mutant cells in different light qualities. WT (left), *cya1* mutant (middle), and *sycrp1* mutant (right) cells were spotted on 0.4% agarose BG-11–10 mM glucose motility plates in the absence (top panels) or the presence (bottom panels) of 0.1 mM cAMP. Plates were placed in different light sources (white, red, green, or blue) and photographed after 4 days. Spot diameter is ~4 mm.

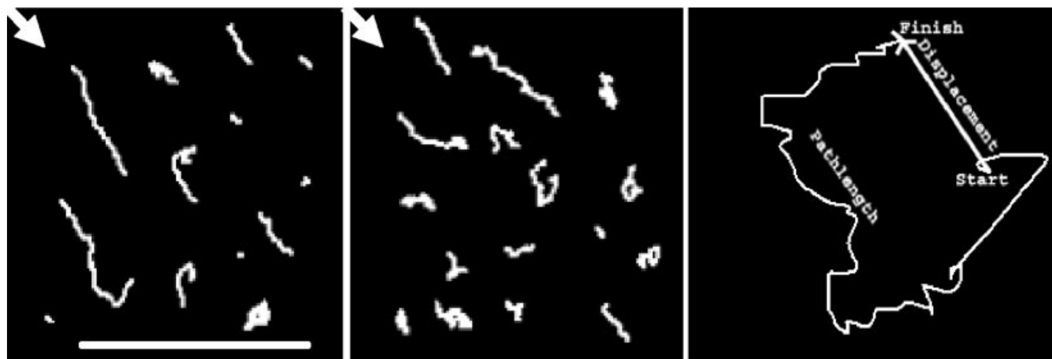

FIG. 6. Representative examples of movement tracks of WT cells and *cya1* mutants in white light. WT cells (left) and *cya1* mutant cells (middle) were spotted on 0.4% agarose BG-11–10 mM glucose motility plates under directional white light for 24 h. We identified single cells (since groups of cells are not possible to track), tracked cell movements for 30 min by time-lapse video microscopy, and quantified them with Metamorph tracking software. A composite made up of randomly selected individual movement tracks was copied and combined into a single figure. The direction of the white light source is shown by an arrow. Bar, 100 μm. The panel on the right shows the path length for a particular track from start to finish and the displacement which is calculated from the start to the finish points.

checked under four different light conditions, either in the presence or in the absence of cAMP (Fig. 5). In white, red, and green light, WT cells exhibited rapid phototaxis, both in the absence and in the presence of cAMP. In white, red, and green light, in the presence of exogenously added cAMP, the motility of the *cya1* mutant was restored (lower panels) In blue light, cell division and growth were lower than in other light conditions, but if cells were spotted at a high density (or allowed to grow for long periods on plates), one observed small fingerlike projections. In blue light, exogenous cAMP did appear to rescue the *cya1* mutant but the results were not as obvious as in the other light regimens since the fingerlike projections were very small. In contrast to the *cya1* mutant, the *sycrp1* mutant cells were not rescued by the addition of cAMP. It has been postulated that the blue light signal for phototaxis is mediated by cAMP, although the regulatory mechanisms have not yet been identified (23). It has also been proposed that the phytochrome Cph2 is a component of a signal transduction event inhibiting the movement of *Synechocystis* sp. strain PCC 6803 cells towards blue light (24). Recently, a flavin adenine dinucleotide-binding BLUF domain protein (the slr1694 gene product) has also been suggested to play a role in cAMP-mediated blue light signaling for positive phototaxis (11, 19, 23).

We used a combination of time-lapse video microscopy and tracking software to analyze the movement of individual WT and mutant cells. WT and *cya1* mutant cells were spotted at a low density on motility plates and incubated for 24 h with directional white light. By this time most cells were in phase 2. We identified and tracked single cells since tracking groups of cells is challenging. Movements of wild-type and *cya1* mutant single cells in response to unidirectional white light (95 μmol photon m^{-2} s^{-1}) were recorded using a Coolsnap Pro Monochrome (Media Cybernetics, Silver Spring, MD) camera attached to a Nikon TE300 inverted microscope. Movies were acquired in 120 frames for a total of 1,800 s for each movie, with the time between frames set at 15 s. The movement of ~20 to 30 individual, well-separated cells was tracked with Metamorph software (Universal Imaging Corporation, Downingtown, PA) using a combination of automated and manual tracking. Representative tracks of individual cells (WT cells and *cya1* mutants) over a 30-min period are shown in Fig. 6. Two motion parameters were measured, the shortest linear distance or displacement (D) from the start point of a time-lapse recording to the finish and the total distance or path length (T) traversed by a cell. The ratio of D/T provided a measure of directed motility or phototaxis. The average total path length (T) for WT and *cya1* mutant cells was computed to be 30.2 ± 10.6 μm and 41.3 ± 9.2 μm, respectively, over the 30-min period. The total displacement (D) was 15.5 ± 8.1 μm and 17.0 ± 4.6 μm, respectively, for WT and *cya1* mutant cells. Thus, the average of the D/T ratios of individual cells was found to be 0.44 ± 0.08 and 0.44 ± 0.13 for WT and the *cya1* mutant, respectively. The mean velocities were found to be 1.00 ± 0.35 μm/min and 1.38 ± 0.31 μm/min for WT and *cya1* mutant cells, respectively. Based on these parameters (i.e., directed motility and velocity) individual *cya1* mutant cells are indistinguishable from WT cells in the early phases of motility. Once the cells have moved to the front and are packed together, it is no longer possible to track the movement of single cells and so we cannot estimate motility rates beyond phase 2. These results confirm that the *cya1* mutant cells are motile and phototactic but cannot make the fingerlike projections that are characteristic of the WT cells. *sycrp1* mutants also show the same behavior (data not shown).

In summary, we carried out a detailed characterization of the phototactic response of *Synechocystis* sp. and *cya1* mutants to different light regimens, glucose, and cAMP. The results of our study provide evidence that *cya1* and *sycrp1* mutants are motile and phototactic at the individual cell level and the characteristics of movement (in phases 1 and 2) are very similar to those of WT cells. The strong phototaxis that is characteristic of phase 3 (in which WT cells move rapidly forward in the fingerlike projections) is impaired in *cya1* and *sycrp1* mutants. The fact that phase 3 motility can be restored to WT levels, simply by the addition of cAMP to the *cya1* mutants, in all light conditions tested indicates that cAMP levels are critical for phase 3 phototaxis. A combined effect of glucose, cAMP, and light is probably involved in the final output that controls the complex phototactic pathway (3, 25).

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