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An abundant cell-surface polypeptide is required for swimming by the nonflagellated marine cyanobacterium *Synechococcus*

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ABSTRACT **Certain marine unicellular cyanobacteria of the genus *Synechococcus* exhibit a unique and mysterious form of motility characterized by the ability to swim in liquid in the absence of flagella. An abundant cell-surface-associated polypeptide that is required for swimming motility by *Synechococcus* sp. strain WH8102 has been identified, and the gene encoding it, *swmA*, has been cloned and sequenced. The predicted SwmA protein contains a number of Ca^{2+}-binding motifs as well as several potential N-glycosylation sites. Insertional inactivation of *swmA* in *Synechococcus* sp. strain WH8102 results in a loss of the ability to translocate, although the mutant strain, Swm-1, generates torque. This suggests that SwmA functions in the generation of thrust.**

Marine unicellular cyanobacteria of the genus *Synechococcus* are among the most abundant photosynthetic organisms in the open ocean and their contribution to primary production is substantial (1, 2). In 1985, Waterbury *et al.* (3) reported that several isolates of marine *Synechococcus* were capable of swimming. This discovery was remarkable for two reasons. First, unlike other motile cyanobacteria that move by gliding on surfaces, motile *Synechococcus* sp. swam in liquids. Second, although their swimming behavior resembled that of flagellated bacteria, marine motile *Synechococcus* had neither flagella nor any other visible organelle of locomotion (3). This represents a novel and still mysterious type of prokaryotic motility, and at issue are such fundamental questions as how organisms convert chemical energy into motion.

Other prokaryotes that are able to swim in liquids do so by the means of flagella, which can be thought of as helical propellers driven by a biological rotary motor (4). A flagellum consists of a semirigid helical filament attached to the cell by the hook–basal–body complex (5). The flagella, which can be external or internal to the cells, as in the periplasmic flagella of spirochetes, rotate (6, 7). Flagellar rotation generates torque and thrust and thus the cell translocates. Certain features of swimming in *Synechococcus* resemble swimming by flagellated bacteria. Swimming cells of *Synechococcus* rotate about their longitudinal axis as they translocate (3, 8) and hence generate both torque and thrust. And like flagellated bacteria, *Synechococcus* cells are slowed down and ultimately immobilized by increasing medium viscosity (8).

Efforts at visualizing the organelle or structure responsible for swimming have included transmission electron microscopy of whole and thin-sectioned cells, as well as of freeze-fractured and etched preparations (3, 8). These studies did not reveal the presence of any identifiable structure that might be associated with the ability to swim. And an examination of actively swimming cells by high-intensity darkfield microscopy, a technique that makes possible the visualization of single flagella, did not reveal any flagella (8). And, shearing experiments, which are extremely effective at eliminating motility in flagellated bacteria, did not at all affect swimming in *Synechococcus* (3, 8). Recently, Pitta and Berg (9) have ruled out self-electrophoresis as a possible mechanism.

How then does *Synechococcus* swim in liquids? The observation that cells that become attached to a microscope slide or coverslip rotate about their point of attachment (3) suggests either that the cell surface rotates or that something on the surface does. Identification of such components should provide insights into the mechanism of this type of motility. In this paper, I describe the identification of an abundant cell-surface protein that is required for swimming by *Synechococcus* sp. strain WH8102.

MATERIALS AND METHODS

Bacterial Strains and Growth Conditions. *Escherichia coli* strains MC1061 and DH5α, which were used as recipients in transformations and as donors in conjugations with *Synechococcus*, were grown in LB medium (10). Ampicillin (100 μg/ml), kanamycin (50 μg/ml), and chloramphenicol (10 μg/ml) were used where appropriate for the selection and maintenance of plasmids. *Synechococcus* WH8102 and WH8103 were grown in SN medium (11) prepared with local seawater. Cultures (50 ml) in 125-ml flasks were incubated without shaking at 25°C and with constant illumination (10 $\mu E \cdot m^{-2} \cdot sec^{-1}$). When required, kanamycin was present at a concentration of 25 μg/ml.

Purification of the 130-kDa Polypeptide. The outer membrane of *Synechococcus* WH8102 and WH8103 was prepared by the EDTA method of Resch and Gibson (12) with the following modifications. Exponentially growing *Synechococcus* WH 8102 (50 ml) containing 1–4 $\times$ 10^8 cells per ml were centrifuged at 6277 $\times$ *g* for 10 min at 20°C. The cells were washed once in sterile SN medium and resuspended in ice-cold stripping buffer (50 mM Tris·HCl/50 mM Na_2EDTA/15% sucrose, pH 8.0). Following a 30 min incubation on ice, the suspension was centrifuged at 6277 $\times$ *g* at 4°C for 10 min to remove the cells. The supernatant was collected and centrifuged at 100,446 $\times$ *g* at 4°C for 90 min. The orange high-speed pellet was resuspended in TE (10 mM Tris·HCl/1 mM Na_2EDTA, pH 8.0). The high-speed supernatant contained the 130-kDa polypeptide.

The sequence of the N-terminal 29 aa of the *Synechococcus* sp. strain WH8102 130-kDa polypeptide was determined as described (13) at the University of California (San Diego) Protein Sequencing Facility.

SDS/PAGE was carried out by the procedure of Laemmli (14) or as described (15) for some mini gels. The resolving gels contained 7.5 or 10% acrylamide. To detect glycoproteins, periodic acid-Schiff staining of SDS gels was carried out as described (16, 17) using ovalbumin as a positive control.

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Data deposition: The sequence reported in this paper has been deposited in the GenBank data base (accession no. U48223).

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Isolation of the Gene Encoding the 130-kDa Polypeptide. An oligonucleotide probe based on the sequence of the N-terminal 22nd to 27th amino acids of the 130-kDa polypeptide (5′-AARGAYTGGTTYCTBGAYGC-3′, coding strand) was used to probe restriction digests of total *Synechococcus* sp. strain WH8102 DNA. A single *Pst*I fragment of 4 kb hybridized to the probe. To clone this fragment, a size-directed library consisting of *Pst*I fragments of *Synechococcus* sp. strain WH8102 DNA was constructed and screened by colony hybridization (10) as follows. Total *Synechococcus* sp. strain WH8102 DNA was digested to completion with *Pst*I and separated by electrophoresis. The fragments migrating to the area between the 3.7 and 4.3 kb λ *Bst*EII markers were excised and ligated into the *Pst*I site of pUC19 and the ligation mixture was used to transform *E. coli* DH5α. Ampicillin-resistant colonies (600) were screened by colony hybridization, using end-labeled oligonucleotide as the probe. Of these, 14 hybridizing colonies were chosen for further analysis. The plasmids from these 14 colonies all contained a 4-kb *Pst*I fragment that hybridized with the oligonucleotide probe. One of these plasmids, pMot3–3, was chosen for all further work.

Sequence analysis of the hybridizing region in pMot3–3 indicated that it encoded only the N-terminal end of the gene for the 130-kDa polypeptide, *swmA*. To clone the rest of the gene, a size-directed *Sph*I library of *Synechococcus* DNA consisting of fragments in the range of 6.4 to 7.2 kb ligated in the *Sph*I site of pUC19 was screened by colony hybridization using as probe a 2-kb *Sal*I fragment from pMot3–3 encoding the 5′ end of *swmA*. Two hybridizing colonies were obtained and the plasmids from these both contained a 7-kb *Sph*I fragment that hybridized with the probe. One of these plasmids, pSphI, was chosen for all further work.

Standard procedures were used for general molecular biological techniques (10). When the oligonucleotide was used as a probe in Southern hybridizations, the hybridization and washing temperature was 50°C. All other Southern hybridizations and washes were performed at 65°C. Double-stranded DNA probes were labeled by random priming with digoxigenin-11-dUTP using the Genius system (Boehringer Mannheim), whereas oligonucleotides were end-labeled using [γ-^{32}P]ATP and standard conditions (10).

DNA Sequence Analysis. Both strands of *swmA* and flanking regions were sequenced using either nested deletions generated by partial exonuclease III digestion with an Erase-a-Base kit from Promega or with oligonucleotide primers and pSph1 as template. Nucleotide sequences were determined by automated sequencing of plasmid templates using a PRISM Ready Reaction DyeDeoxy Terminator cycle sequencing kit and a model 373 DNA sequencer (Applied Biosystems).

Nucleotide sequence data were analyzed with the DNALYSIS program written by W. J. Buikema for the Apple Macintosh and the SEQUENCHER 3.0 program (Gene Codes, Ann Arbor, MI). Amino acid sequence homology searches were carried out at National Center for Biotechnology Information using the BLAST algorithm (18) and the nonredundant data base. The PROSITE (19) data base (release 12) was searched with the PROSITESCAN program accessed through the ISREC server (Swiss Institute for Experimental Cancer Research). The theoretical molecular mass and the pI of SwmA were calculated with the Compute PI/MW program accessed through the ExPASy server (University of Geneva). A hydropathy profile of SwmA was obtained with the DNA STRIDER program (C. Marck, Centre d'Etudes Nucleaires de Saclay, France) using the Kyte–Doolittle scale (20) and a window of nine amino acids.

Insertional Mutation of *swmA*. *Synechococcus* sp. strain Swm-1, in which the genomic *swmA* gene is interrupted by a duplication and insertion of a nonreplicating plasmid was constructed as described below. The 973-bp *Bam*HI/*Nru*I fragment of pSph1 (Fig. 1), which is completely internal to *swmA*, was cloned into the *Eco*RV and *Bam*HI sites of pMUT100 (30), a pBR322 derivative that can be mobilized into *Synechococcus* sp. strainWH8102 but that cannot replicate in this host. The resulting plasmid, pMUT5, was mobilized into *Synechococcus* sp. strain WH8102 by conjugation using methods described elsewhere (30). Exconjugants were selected on SN pour plates containing kanamycin (25 μg/ml) and were screened by Southern blot hybridization to confirm the expected restriction pattern that was indicative of integration of pMUT5 at the site of homology. Prolonged exposure of the Southern blot further confirmed that no wild-type (uninterrupted) copies of *swmA* were present in the transconjugants (data not shown).

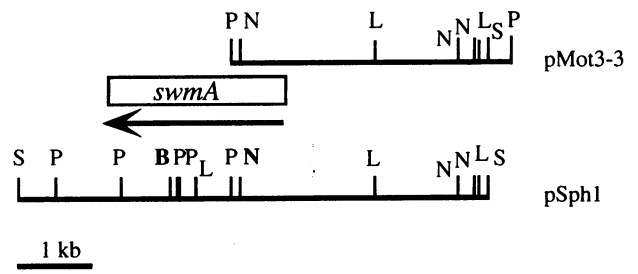

FIG. 1. Restriction map of the region containing the *Synechococcus* sp. strain WH8102 *swmA* gene. The fragments contained in pMot3–3 and pSph1 are aligned with respect to their overlapping regions. The open box represents the *swmA* coding sequence and the arrow below it indicates the direction of transcription. The *Bam*HI and *Nru*I sites in boldface delineate the fragment used to inactivate *swmA*. B, *Bam*HI; L, *Sal*I; N, *Nru*I; P, *Pst*I; S, *Sph*I.

Microscopy. Microscopic examination of *Synechococcus* cultures was carried out with a Zeiss Axioskop microscope equipped with epifluorescence, phase contrast, and differential-interference-contrast optics.

RESULTS

Identification of the 130-kDa Polypeptide. The observation that cells of motile *Synechococcus* that become attached to a microscope slide at one cell pole rotate about their point of attachment (3, 8) suggests that there may be cell surface components that rotate. To identify such components, preliminary experiments were carried out in which actively swimming cells of *Synechococcus* WH8103 were briefly treated with the protease proteinase K. Motility stopped within 5 min, and when whole cell extracts of proteinase K-treated cells were compared with those of untreated cells by SDS/PAGE, an abundant band of 130,000 Da was greatly diminished in intensity (data not shown). To determine whether the presence of this polypeptide was associated with motility, whole cell extracts of a number of motile as well as nonmotile marine *Synechococcus* strains were compared by SDS/PAGE (Fig. 2). An abundant polypeptide of 130,000 Da is present in all of the motile strains tested but in none of the nonmotile ones.

Purification of the 130-kDa Polypeptide. Because of its protease sensitivity, it was suspected that the 130-kDa polypeptide (or a portion of it) would be located in or associated with the outer membrane. Accordingly, cells of *Synechococcus* sp. strain WH8102 were treated with EDTA, a procedure that has been shown to strip the outer membrane from cells of *Synechococcus* PCC7942 (12). The material released from cells by EDTA treatment was further subjected to ultracentrifugation and the soluble and insoluble fractions were examined by SDS/PAGE (Fig. 3). The insoluble fraction, which consisted of a transparent orange pellet, contained two major polypeptides of 70 kDa and 45 kDa and five minor polypeptides. The soluble fraction contained a major polypeptide of 130 kDa and two minor polypeptides. The 130-kDa polypeptide migrated to

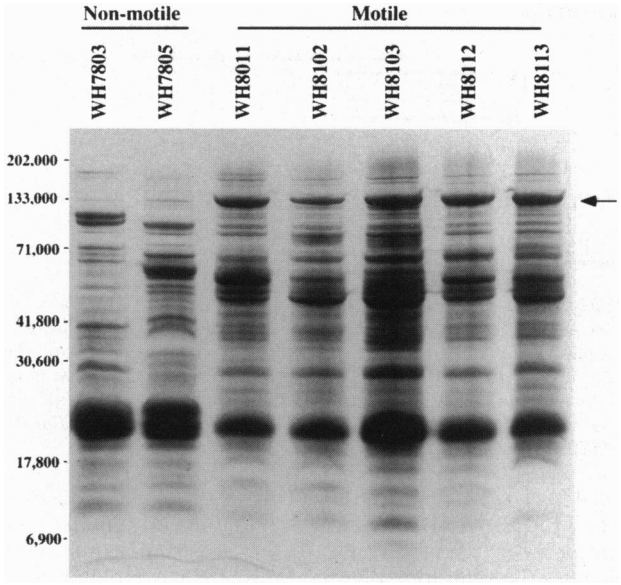

FIG. 2. Coomassie blue-stained 10% SDS polyacrylamide gel of whole cell extracts of nonmotile (WH7803 and WH7805) and motile (WH8011, WH8102, WH8103, WH8112, and WH8113) strains of marine *Synechococcus*. The migration of molecular mass standards is indicated on the left in daltons. The arrow points to a 130,000-Da polypeptide present in all of the motile strains but not the nonmotile ones.

the same position as the major polypeptide of 130 kDa seen in whole cell extracts of *Synechococcus* sp. strain WH8102 and other motile strains (data not shown). Periodic acid-Schiff staining indicated that both the 130-kDa and the 70-kDa polypeptides are likely to be glycosylated (data not shown).

The sequence of the first 29 aa of the 130-kDa polypeptide purified from *Synechococcus* sp. strain WH8102 (Fig. 4) and of the first 15 aa of the polypeptide purified from *Synechococcus* sp. strain WH8103 were determined. The first 15 aa were identical in both strains. In both cases, the fifth residue was unidentifiable, giving rise to a peak not matching that of any known amino acid phenylthiohydantoin derivative, suggesting that it was modified in some way. On the basis of the DNA sequence of the gene encoding the 130-kDa polypeptide (see below) from *Synechococcus* sp. strain WH8102, this residue is predicted to be a threonine. The nature of the modification, which did not resemble that seen when threonine is either glycosylated or phosphorylated (M. Williamson, personal communication), is unknown.

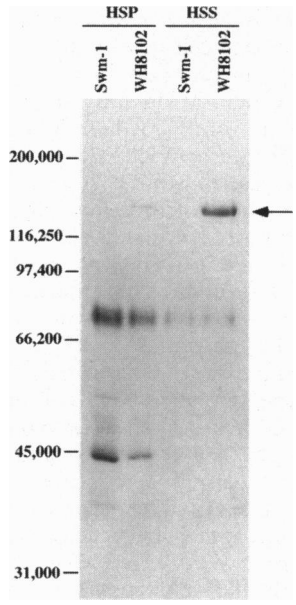

FIG. 3. Coomassie blue-stained 7% SDS polyacrylamide gel of material released by EDTA treatment of either wild-type (WH8102) *Synechococcus* sp. strain WH8102 or a mutant strain (Swm-1) in which *swmA* has been insertionally inactivated. High speed pellet (HSP) denotes the insoluble fraction, whereas high speed supernatant (HSS) denotes the soluble fraction. The migration of molecular mass standards is indicated on the left in daltons. The arrow points to the 130-kDa polypeptide.

Cloning and Sequence Analysis of *swmA*. The amino acid sequence determined from the 130-kDa polypeptide purified from *Synechococcus* sp. strain WH8102 was used to derive a mixed oligonucleotide probe with which a plasmid, pMot3–3, was identified (Fig. 1). DNA sequence analysis of the region in pMot3–3 hybridizing to the oligonucleotide revealed that this plasmid encoded only the N-terminal portion of the 130-kDa polypeptide. To clone the rest of the gene encoding it, a probe made from pMot3–3 was used to identify a larger plasmid, pSph1, containing a 7-kb *Sph*I fragment of *Synechococcus* WH8102 DNA that includes all of *swmA*, as well as flanking sequences (Fig. 1).

Sequence analysis of 3492 base pairs revealed an ORF that encoded the 29 aa determined by sequencing the purified protein (Fig. 4). Two potential translational initiation codons were found (Fig. 4). The TTG codon at nucleotide 503 is the more likely start codon because it is preceded, by 11 bp, by a putative ribosome binding site. This ORF, which encodes a polypeptide of 835 aa, is designated the *swmA* gene (for *sw*imming *m*otility), and the protein it encodes as SwmA. The N-terminal 20 aa of the predicted protein are absent in mature SwmA, indicating that they are cleaved, possibly during secretion. The predicted molecular mass of the polypeptide encoded by the *swmA* ORF is 83,568 Da, and that of the mature polypeptide, lacking the N-terminal 20 aa, is 81,342 Da, which is 38% lower than the molecular mass determined by SDS/PAGE. The mature deduced polypeptide has a calculated net charge of −83 and a predicted pI of 3.85.

Following the stop codon of *swmA* is a region characteristic of rho-independent terminators (21), containing a set of GC-rich inverted repeats followed by a string of thymidines. Inspection of the sequenced region upstream of *swmA* (342 bp) revealed a small ORF of 51 aa with a termination codon 3 bp upstream of the *swmA* ATG. No significant similarities were found in comparisons of its predicted protein sequence to the nonredundant protein data base. Inspection of the sequenced region downstream of *swmA* (484 bp) did not reveal any ORFs. Southern blot analyses indicate that *Synechococcus* sp. strain WH8102 has a single copy of *swmA* (data not shown).

The predicted amino acid sequence of SwmA was used to search the PROSITE library of protein patterns and motifs (19). Two calcium-binding motifs were found: an EF-hand loop calcium-binding domain (PROSITE pattern PS00018) and a hemolysin-type calcium-binding region signature (PROSITE pattern PS00330) (Fig. 4). In addition, 13 potential N-glycosylation sites were found, eight of which are in the C-terminal fourth of the protein (Fig. 4)

The predicted amino acid sequence of SwmA was also used to search the nonredundant peptide sequence database using the BLAST algorithm. Significant matches were found to a diverse group of proteins including RsaA, a paracrystalline surface layer protein from *Caulobacter crescentus* (22); AlgE4, a mannuronan C-5′-epimerase from *Azotobacter vinelandii* (23); NodO from *Rhizobium leguminosarum* (24); a number of hemolysin and hemolysin-like proteins including CyaA from *Bordetella pertussis* (25); and to a partial ORF, HlyA, from the cyanobacterium *Anabaena* sp. strain 7120 (GenBank accession no. U13767). Inspection of the alignments of SwmA to these proteins revealed that the regions of homology were largely

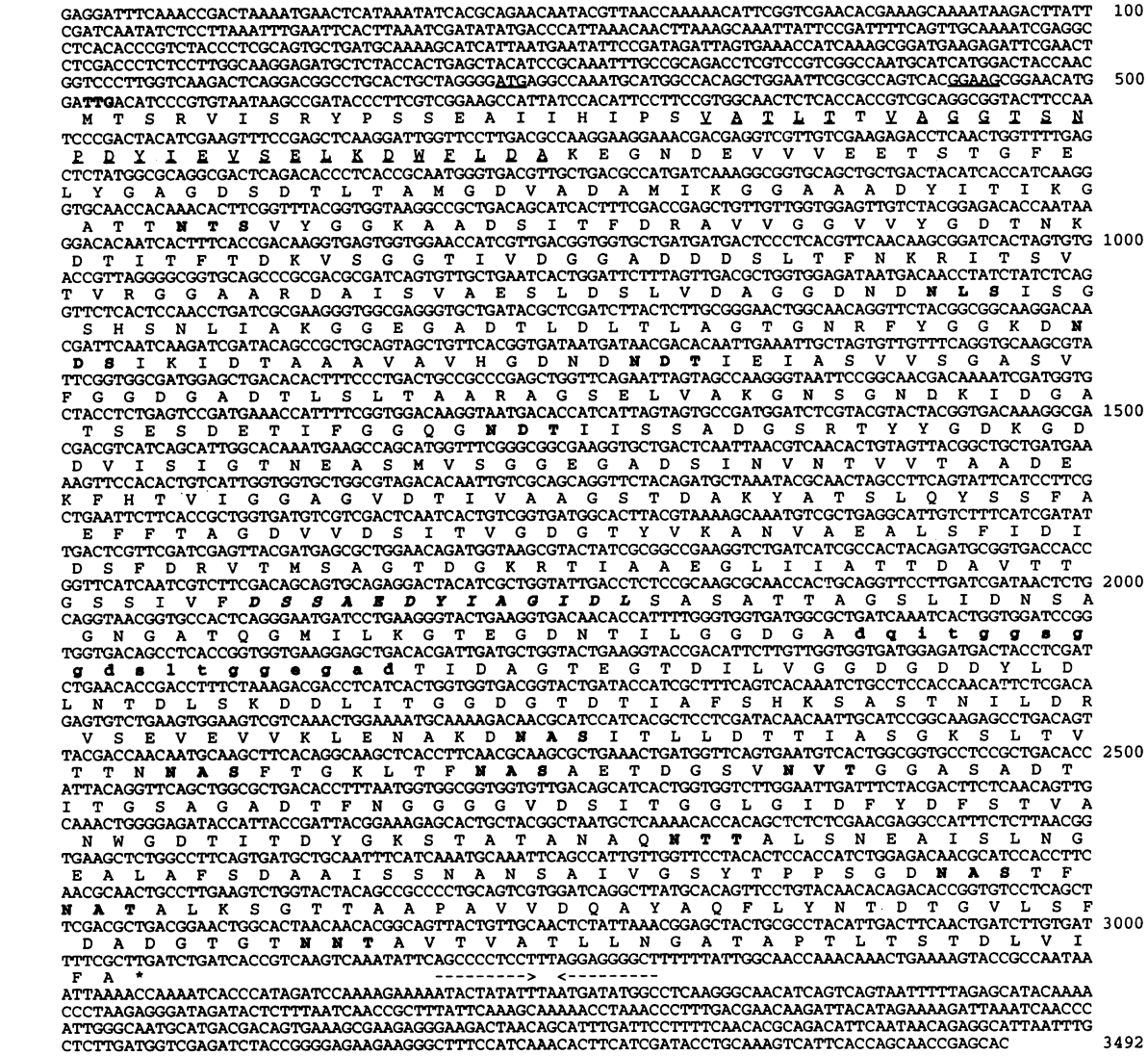

FIG. 4. Nucleotide sequence of the *Synechococcus* sp. strain WH8102 *swmA* gene and 5′- and 3′-flanking regions. The TTG translational start codon is indicated in boldface type and the alternative ATG start codon is underlined. A putative ribosome binding site (bp 487–491) is underlined. The arrows indicate GC-rich inverted repeats. The predicted amino acid sequence of *swmA* is shown in single-letter code. The 29 N-terminal residues determined by protein sequencing are underlined. The unidentifiable amino acid (see text) is not underlined. Potential N-glycosylation sites (NXS or NXT, where X is any amino acid) are in uppercase boldface type. The EF hand Ca^{2+}-binding domain (residues 473–485) is indicated by italicized boldface uppercase letters and the hemolysin-type Ca^{2+}-binding region signature (residues 526–544) is indicated in boldface lowercase letters.

confined to parts of the protein containing calcium-binding patterns (data not shown), including the region containing a hemolysin-type calcium-binding region signature, as well as regions containing the motif GGXGXD, which has been shown to bind calcium (26) (Fig. 5). The latter motif is found 12 times in SwmA (Fig. 5).

A hydropathy profile of the predicted polypeptide sequence of SwmA using the Kyte–Doolittle scale revealed alternating hydrophilic and hydrophobic regions and the absence of extended regions of high overall hydrophobicity (not shown). A region of sufficient length and hydrophobicity to span a membrane is found at aa 14–33 and could serve as a signal sequence. This sequence overlaps with the mature N terminus, and in this respect resembles a type IV prepilin signal peptide in which the cleavage site precedes the hydrophobic α helix, which remains part of the mature protein (27).

Inactivation of *swmA*. To determine the role of *Swm*A, if any, in swimming motility in *Synechococcus* sp. strain WH8102, the *swmA* gene was inactivated *in vivo* by insertionally interrupting its coding sequence by homologous recombination between the chromosome and a completely internal fragment of *swmA* cloned in a nonreplicating vector. The resulting strain was designated *Synechococcus* sp. strain SWM-1. To confirm that this construction had indeed resulted in the inactivation of *swmA*, *Synechococcus* sp. strain Swm-1 was treated with EDTA, and the material released from the cells was examined by SDS/PAGE (Fig. 2). The insoluble material released from Swm-1 contained all of the major and minor polypeptides seen in preparations from the wild type, with the exception of the 130-kDa polypeptide (Fig. 2). Furthermore, the soluble fraction released from Swm-1, which in the wild type is greatly enriched in the 130-kDa polypeptide, completely lacked the 130-kDa polypeptide (Fig. 2).

Microscopic examination of strain Swm-1 indicated that, unlike wild-type WH8102, it could no longer translocate; the cells stayed in place, indicating that thrust was absent. Cells of strain Swm-1 that became attached to the microscope slide or coverslip, could still rotate about their point of attachment, indicating that torque was still being generated. Because no other ORFs are found downstream of *swmA*, this phenotype is due to the inactivation of *swmA* and not to a polar effect of the insertion.

A.

Sign.		DxaxxxxGxDxaxGGxxxD
SwmA	518	TILGGDGA**D**QI**T**GGS**GGD**SL**T**GG**E**GA**D**
HlyA	177	TIISGDGD**DT**LYGDS**GND**TL**T**GG**N**GN**D**
CyaA	1417	VLRGAGGA**D**VL**A**GGE**GDD**VLL**GGD**GDD
AlgE4	392	PLVGGDTD**DQ**LQGGS**GAD**RL**DGGA**GDD
NodO	101	VLYAGPGS**D**IL**V**AGD**GAD**VL**T**GG**DDG**D
RsaA	879	TIIGGAGA**DT**LVYT**GGTD**FT**GGT**GA**D**

B.

Sign.	GGxGxD
209	**GGEGAD**
268	**GGDGAD**
310	**GGQGND**
348	**GGEGAD**
373	**GGAGVD**
521	**GGDGAD**
530	**GGSGGD**
539	**GGEGAD**
528	**GGDGDD**
579	**GGDGTD**
678	**GGGGVD**
687	**GGLGID**

FIG. 5. (*A*) Amino acid alignment of the region of SwmA and other proteins containing the hemolysin-type Ca^{2+}-binding region signature. The signature is on the top line where x stands for any amino acid and a stands for either L or I. Residues conforming to this pattern are in boldface type. The GenBank accession numbers for these sequences are: U13767, *Anabaena* HlyA; Y00545, CyaA; L39096, AlgE4; U26541, NodO; and M84760, RsaA. (*B*) Alignment of the repeats found in SwmA containing the GGxGxD Ca^{2+}-binding motif. The motif is on the top line where x stands for any amino acid.

DISCUSSION

A 130-kDa polypeptide that is required for swimming motility by the marine unicellular cyanobacterium *Synechococcus* sp. strain WH8102 has been identified. SwmA is associated with the cell surface: it is accessible to proteinase K and it copurifies with the outer membrane. It does not appear to be an integral outer membrane protein because it becomes soluble rather that partitioning in the insoluble membrane fraction. SwmA, or a protein of similar molecular mass and abundance, is found in all of the motile strains we have tested so far and not in two nonmotile ones. Preliminary results using an antibody raised against SwmA purified from strain WH8102 indicate that it reacts with the abundant 130-kDa polypeptide seen in all of the motile strains but does not react with whole cell extracts of nonmotile strains.

SwmA is similar to a diverse group of proteins including RsaA (an S-layer protein from *C. crescentus*), NodO (a nodulation gene from *R. leguminosarum*), AlgE4 (a mannuronan C-5′-epimerase), and various hemolysin and hemolysin-like proteins. All of these proteins have two properties in common. (*i*) They are external to the cell, being either secreted into the growth medium as is the case for NodO, AlgE4, and hemolysins, or they are associated with the cell surface, like RsaA. (*ii*) Furthermore, they all contain glycine and aspartate-rich repeats that have been shown to function in Ca^{2+}-binding. Like RsaA, SwmA is abundant, carries a net negative charge, and dissociates from the cell surface following treatment with divalent cation chelators. It has been proposed that RsaA attaches to the outer membrane through calcium ion bridging (28). Such a mechanism may also account for SwmA's association with the outer membrane of *Synechococcus* sp. strain WH8102.

On the basis of periodic acid-Schiff staining, SwmA appears to be glycosylated. The fact that SwmA has 13 potential N-glycosylation sites supports this possibility. Glycosylation probably accounts for the discrepancy seen between SwmA's predicted molecular mass and that determined by SDS/PAGE. Such discrepancies are common, as is the case for S-layer glycoproteins whose genes have been sequenced (29). Furthermore, it is known that glycoproteins migrate anomalously in SDS/PAGE (17), resulting in overestimates of molecular mass.

The gene encoding SwmA was isolated and insertionally inactivated in *Synechococcus* sp. strain WH8102. The resulting strain, Swm-1, which no longer makes SwmA, has lost the ability to translocate, but can still rotate about a point of attachment, thus generating torque but not thrust. SwmA's abundance in motile strains and its absence in nonmotile strains of marine *Synechococcus* support its importance to motility.

How then does SwmA function in motility? SwmA is unlikely to be the motor, or torque generator, or one of its constituents because cells lacking SwmA still rotate. Instead, SwmA appears to be involved in the creation of thrust. In this respect, its role could be indirect or direct. An example of an indirect effect would be if the presence of SwmA were required for the proper insertion or positioning in the outer membrane of other components of the motility apparatus. In a direct effect, SwmA may function as a "propeller," obviously not in the sense of a flagellum because no such structure is visible, but in the sense that it causes the cell to propel itself using the torque generated by the motor. It could do this by changing the cell shape or surface characteristics, although as judged by light microscopy, not detectable differences in cell shape or size were observed between the wild-type and strain Swm-1. Pitta and Berg (9) have experimentally ruled out self-electrophoresis as a possible mechanism and have argued that jet propulsion is unlikely. Rather, they suggest that mechanical deformation or movement of the cell surface are plausible alternatives. To address these possibilities, it will be necessary to determine whether SwmA is distributed evenly on the surface, or if there are localized concentrations, at one cell pole for example. Also, by attaching antibody-coated beads to swimming cells, it should be possible to determine whether SwmA itself moves along the surface. These analyses, along with a study of SwmA's interaction with other outer membrane proteins, such as the glycosylated 70-kDa polypeptide for example, should begin to answer these questions and provide insight into the fascinating mystery of how marine *Synechococcus* spp. manage to swim without flagella. What is clear is that at least one polypeptide component of the cell surface is important for this type of motility.

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