The switch complex at the base of the bacterial flagellum is essential for flagellar assembly, rotation, and switching. In Escherichia coli and Salmonella, the complex contains about 26 copies of FliG, 34 copies of FliM, and more than 100 copies of FliN, together forming the basal body C ring. FliG is involved most directly in motor rotation and is located in the upper (membrane-proximal) part of the C ring. A crystal structure of the middle and C-terminal parts of FliG shows two globular domains connected by an α-helix and a short extended segment. The middle domain of FliG has a conserved surface patch formed by the residues EHPQR_{125-128} and R_{160} (the EHPQR motif), and the C-terminal domain has a conserved surface hydrophobic patch. To examine the functional importance of these and other surface features of FliG, we made mutations in residues distributed over the protein surface and measured the effects on flagellar assembly and function. Mutations preventing flagellar assembly occurred mainly in the vicinity of the EHPQR motif and the hydrophobic patch. Mutations causing aberrant clockwise or counterclockwise motor bias occurred in these same regions and in the waist between the upper and lower parts of the C-terminal domain. Pull-down assays with glutathione S-transferase–FliM showed that FliG interacts with FliM through both the EHPQR motif and the hydrophobic patch. We propose a model for the organization of FliG and FliM subunits that accounts for the FliG-FliM interactions identified here and for the different copy numbers of FliG and FliM in the flagellum.

Bacterial flagella are built from about 25 proteins, most of which serve structural roles in forming the basal body, hook, and filament and only a few of which function in rotation (2, 23, 32). The stator is formed from the membrane proteins MotA and MotB, which form complexes with the composition MotA MotB_2 (9, 11, 25, 42), in the membrane surrounding the basal body (21). Each motor contains several MotA-MotB complexes, which can function independently to produce torque (4, 5, 41). The rotor proteins involved in rotation are FliG, FliM, and FliN. These form a large (ca. 4-MDa) assemblage termed the switch complex that is essential for flagellar assembly, rotation, and clockwise/counterclockwise (CW/CCW) switching (12, 57, 59). The switch complex is attached to the cytoplasmic face of the basal body MS ring, a large membrane-embedded structure formed from about 26 copies of the protein FliF (19, 53) (Fig. 1). FliF has two membrane-traversing segments and includes sizable domains in both the periplasm and cytoplasm. A conserved segment near the C terminus of FliF, located in the cytoplasm, forms the site of attachment for FliG (16).

Although each of the switch complex proteins performs multiple functions, mutational studies indicate that each is specialized to some extent. FliM is closely involved in direction switching and contains a segment near its N terminus that binds to the chemotaxis-signaling molecule phospho-CheY (6, 43). FliN is also involved in switching and makes a particularly important contribution to flagellar assembly, probably by binding to the flagellar export protein FliH (8, 15, 36a, 40, 55). FliG is involved most directly in rotation (20, 29). The C-terminal domain of FliG (FliG_C) in particular functions specifically in rotation; deletion of most of this domain prevents rotation but allows flagellar assembly (29). Conserved charged residues in FliG_C have been found to be important for flagellar rotation in a number of species (28, 56) and for control of speed modulation (chemokinesis) in the unidirectional rotary motor of Sinorhizobium meliloti (1). These charged residues of FliG were shown to interact with conserved charged residues in the cytoplasmic domain of the stator protein MotA (56, 60, 61). FliG also interacts with FliM (22, 34, 36, 47) and with the nucleoid-associated DNA-binding protein H-NS (35).

While the mechanism of flagellar rotation is not yet understood at a detailed level, some key features of the mechanism have been established. Energy for rotation comes from the transmembrane gradient of protons (14, 27, 33) or sodium ions (18). Recent studies support a mechanism in which the energizing ions flow through the stator complexes and drive conformational changes as they bind to and dissociate from a critical aspartate residue in MotB (3, 24, 62). We hypothesize that these conformational changes provide the power stroke that drives rotation, applying force to the rotor via the MotA-FliG interface identified in mutational studies (28, 56, 60, 61).

Although the stator appears to be the instigator of movement, the rotor has the major role in controlling the direction of rotation. Mutational replacements that affect the CW/CCW bias of the motor are fairly common in the switch complex proteins but not in MotA or MotB (13, 20, 43). The molecular events that underlie switching have not been defined but must
presumably involve some movement of the C-terminal domain of FliG, to alter the way in which the rotor engages the stator.

Recent studies have begun to provide detailed structural information on the components of the switch complex and their overall organization. The basal body has been imaged in several electron microscopic studies (12, 48, 50, 58). Although FliG is known to be in the upper part of the C ring (16, 22, 26, 37, 61) and some features of its organization have been deduced from cross-linking (31), its exact location is not yet certain. Accordingly, two possible locations for FliG are indicated: OM, outer membrane; PG, peptidoglycan; IM, inner membrane. (B) Structure of residues 115 to 327 of T. maritima FliG (FliGMC) (7), highlighting conserved surface features.

FIG. 1. (A) Locations of proteins involved in flagellar rotation. The location of FlhN is deduced from targeted cross-linking studies (39) and electron microscopic reconstructions (12, 48, 50, 58). Although FliG is known to be in the upper part of the C ring (16, 22, 26, 37, 61) and some features of its organization have been deduced from cross-linking (31), its exact location is not yet certain. Accordingly, two possible locations for FliG are indicated: OM, outer membrane; PG, peptidoglycan; IM, inner membrane. (B) Structure of residues 115 to 327 of T. maritima FliG (FliGMC) (7), highlighting conserved surface features.

Materials and Methods

Strains and mutagenesis. Escherichia coli RP437 (wild type for motility and chemotaxis) and RP3098 (ΔflhDC) were gifts from J. S. Parkinson (University of Utah). FlhD and FlhC are master regulators of flagellar gene expression, and the deletion of these in strain RP3098 prevents the expression of all chromosomal flagellar genes. The flhG-null strain DBF225 contains an in-frame deletion of most of flhG (29). Site-directed mutagenesis used the Altered Sites procedure (Promega) on the flhG gene cloned in plasmid pSL27 (29), a derivative of pAlter-1 (Promega). The pSL27 derivatives encoding mutant variants of FlhG confer ampicillin resistance. Plasmid pDBF86 is a pACYC184 derivative that expresses FliM and FlhN from the tac promoter and confers chloramphenicol resistance (29). pDBF66 is a pACYC184 derivative that expresses CheY from the ara promoter and confers chloramphenicol resistance (40). pHV7 encodes a glutathione S-transferase (GST)–FliG fusion protein and confers kanamycin resistance, and pHIT100 is the corresponding GST-only control (47).

Function of the mutant proteins. To assay the effects of Trp replacement mutations, cells of DBF225 were transformed with pSL27 derivatives encoding the Trp mutant proteins, and fresh transformants were cultured with shaking at 32°C in TB-Ap (10 μg/ml tetracycline, 5 μg/ml NaCl, 100 μg/ml ampicillin) to mid-exponential phase. One microliter of each culture was spotted onto swarm plates (TB solidified with 0.28% Bacto-agar) and incubated at 32°C. Swarms were measured at regular intervals, and plots of diameter versus time were used to determine swarming rates. Swarming rates of the mutants are relative to wild-type controls. Plasmid pSL27 carrying wild-type flhG restored full swarming ability to the flhG-null strain DBF225 (29), indicating that the level of FlhG expression from this plasmid is sufficient for normal flagellar assembly and function.

For the analysis of swimming behavior, cells were picked from plates and grown to saturation at 32°C in TB-Ap (10 μg/ml tetracycline, 5 μg/ml NaCl, 100 μg/ml ampicillin) to mid-exponential phase. One microliter of each culture was spotted onto swarm plates (TB solidified with 0.28% Bacto-agar) and incubated at 32°C. Swarms were measured at regular intervals, and plots of diameter versus time were used to determine swarming rates. Swarming rates of the mutants are relative to wild-type controls. Plasmid pSL27 carrying wild-type flhG restored full swarming ability to the flhG-null strain DBF225 (29), indicating that the level of FlhG expression from this plasmid is sufficient for normal flagellar assembly and function.
Motility was scored visually and compared with that of wild-type cells prepared in the same way.

Mutants that failed to swarm or swim were stained by using the wet-mount procedure of Heimbrook et al. (17) to determine whether flagella were present. Wild-type controls were included, and staining experiments were done at least twice.

**Dominance and overexpression effects.** To assay the dominance of the fliG mutations, pSL27 variants expressing the mutant FlIG proteins were transformed into the wild-type strain RP437 and swarming rates were measured as described above. The control was RP437 transformed with wild-type pSL27.

To measure effects of overexpressed FlIM and FlIN on the nonflagellate fliG mutants, cells of strain DFB225 were transformed with pSL27 plasmids carrying the fliG variants that gave nonflagellate phenotype, with pDBF96 present to express FlIM and FlIN. Controls were transformed with the pSL27 variants and pACYC184. Swarming was examined in plates containing amplicillin, chloramphenicol, and 100 μM IPTG (isopropyl-β-d-thiogalactopyranoside) to induce expression of FlIM and FlIN.

To examine effects of CheY overexpression on the smooth-swimming fliG mutants, DFB225 was transformed with the fliG plasmids that conferred aberrantly smooth swimming and with pDBF66 to express CheY. Controls were transformed with the pSL27 variants and pACYC184, the parent plasmid of pDBF66. Swarming was examined in plates containing amplicillin, chloramphenicol, and 1.0 mM arabinose to induce expression of FlIM and FlIN.

**Binding assays.** Binding of FlIG to FlIM was measured using a GST pull-down assay, essentially as described by Tang et al. (47) and Mathews et al. (36) with minor modification. Levels of some of the mutant FlIG proteins were found to be decreased by coexpression of the GST-FlIM fusion protein in the same cells, and so the experiments used two strains, one expressing GST-FlIM from plasmid pHt86 (47) and another expressing FlIG or its variant mutants from plasmid pSL27 (29). Control experiments used GST only, expressed from plasmid pHt100 (47).

The strain was BL21(DE3) (44). Cells were cultured overnight at 32°C in 40 ml of TB containing the appropriate antibiotics and 400 μM IPTG. Cells were harvested and resuspended in lysozyme containing buffer as described previously (29). Control experiments used GST only, expressed from plasmid pHt100 (47). The strain was BL21(DE3) (44). Cells were cultured overnight at 32°C in 40 ml of TB containing the appropriate antibiotics and 400 μM IPTG. Cells were harvested and resuspended in lysozyme containing buffer as described previously (29).

Debris was pelleted (16,000 g, 30 min, 4°C), and 50 μl of the supernatant was saved for use in estimating the amount of FlIM present before addition of affinity beads. The rest (~1 ml) was transferred to a clean tube, mixed with 150 μl of a 50% slurry of glutathione-Sepharose 4B (Pharmacia) prepared according to the manufacturer’s directions, and incubated for 75 min at room temperature with gentle rotation to release the GST-FliM and associated proteins. Beads were then pelleted, and the supernatant was collected for analysis by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and immunoblotting using anti-FlIG antibody (29).

**RESULTS**

Trp replacements were made at 33 positions distributed over the surface of FlIGAC. Plasmids expressing the mutant variants of FlIG were transformed into the fliG-null strain DFB225, and function was assayed by measuring swimming in soft agar and motility in liquid. Immotile mutants were stained to see if flagella were assembled. The results are summarized in Table 1, and swarming phenotypes are mapped onto the FlIG structure in Fig. 2. The structure is that of Thermotoga maritima FlIG but the numbering used is for residues in the *E. coli* protein.

**Regions important for flagellar assembly.** The wild type had an average of about five flagella per cell. Flagellar assembly appeared to be completely disrupted by Trp replacements at positions 128 and 158 in the middle domain and at positions 202 and 225 in the C-terminal domain (Table 1). Cells of these mutants were immotile in liquid media and were nonflagellate. On soft-agar plates, the 128W, 158W, and 202W mutants failed to swarm, while the 225W mutant gave rise to small numbers of satellite microcolonies after prolonged incubation (not shown), indicating the rare occurrence of motile cells. Residue 128 is Q of the EHPOQR motif, and residues 202 and 225 are in the hydrophobic patch on the C-terminal domain. Met158 is in a shallow hydrophobic cleft in the middle domain, on a face distant from the EHPQR motif (Fig. 2).

Flagellation was decreased but not eliminated by Trp replacements in residues 147, 163, and 165. Most cells of these mutants lacked flagella, but a few had one or two. Consistent with the poor flagellation, these mutants appeared to be either immotile (position 147) or weakly motile (positions 163 and 165) in liquid medium. Residue 163 is adjacent to the Arg residue of the EHPOQR motif. Residues 147 and 165 are more distant from the EHPOQR motif, on the same face as residue 158 (Fig. 2).

### Table 1. Effects of Trp replacements in fliG

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<tr>
<th>Mutation</th>
<th>Swarming rate</th>
<th>Swimming rate</th>
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<th>Dominance</th>
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* Motility and flagellation were measured in cells of the fliG-null strain DFB225 transformed with mutant variants of pSL27.

* Residue numbering is for *E. coli* FlIG; numbers for *T. maritima* FlIG are greater by one (for positions 117 to 181) or by two (for positions 191 and higher).

* Rates are relative to that of a wild-type control strain included on the plates. R/T, normal pattern of runs and tumbles; R, smooth swimming; T, tumble; r/T, tumble biased but short runs noted; w, motility weak; Imm, immotile.

* Nonflagellate; +/+, one or two flagella per cell; n, the majority of cells are nonflagellate but a rare cell has one flagellum; NM, not measured.

* Swarming rate of wild-type (RP437) cells transformed with the mutant FlIG plasmids, relative to controls expressing wild-type FlIG from the plasmid.
Defects in switching. Several of the Trp mutants were flagellate and motile but swarmed poorly owing to an aberrantly CCW or CW motor bias (Table 1). Smooth swimming, which is characteristic of exclusively CCW motor rotation, was seen for the Trp replacements at positions 117, 125, and 214. Residue 125 is E of the EHPRQ motif, and residue 214 is a V in the hydrophobic patch. Residue 117 is in the middle domain but somewhat (about 10 Å) apart from the EHPQR motif (Fig. 2). Milder swarming defects associated with a less severe CCW bias occurred for the mutations at position 152, which is near the margin of the EHPQR motif, and position 191, on the interdomain helix near the Gly-Gly linker. Tumbly motility indicative of CW motor bias was seen for mutations at position 196 in the hydrophobic patch and position 235 in the relatively narrow “waist” between the upper and lower parts of the C-terminal domain (Fig. 2).

Regions that tolerate Trp replacements. Most (19 out of 33) Trp replacement mutants swarmed at 70% of the wild-type rate or better, indicating nearly normal function of the mutant FliG proteins in assembly, rotation, and switching. Positions where Trp was tolerated occur in the interdomain helix at the end attaching to the middle domain, on and around the charge-bearing ridge, and in several positions near the bottom of FliGc, but outside the hydrophobic patch. The functionally important region at the bottom of FliGc is delineated by five positions that tolerated Trp replacement (205, 207, 217, 218, and 228) surrounding four positions that did not (196, 202, 214, and 225) (Fig. 2). In the middle domain, Trp was fully tolerated at only one position, residue 140, on the face of the domain opposite the EHPQR motif.

Dominance and overexpression effects. To examine dominance of the Trp replacement mutants, plasmids encoding the FliG mutant variants were introduced into wild-type cells and swarming rates were measured. All of the strong switch bias mutations (at positions 117, 125, 214, and 235) exhibited strong dominance in this assay, indicating that the mutant proteins can be incorporated into flagellar motors and impose an aberrant switch bias (Table 1). Most of the Fla+ mutations also were dominant, reducing swarming rates of the wild type to less than 5% of normal. One exception was the mutation at position 225, which as noted above also allowed infrequent flagellar assembly (as evidenced by satellite colonies in swarm plates).

FliG mutations might affect flagellar assembly or function by impeding the installation of FliM. To determine whether flagellar assembly in the mutants could be improved by increasing the amount of the other switch complex proteins, cells of the poorly flagellated and nonflagellated mutants were transformed with a plasmid that allowed IPTG-induced overexpression of FliM and FliN. (The two proteins were coexpressed because overexpression of FliM alone causes severe motility impairment owing to an imbalance between FliM and FliN levels [10, 46].) Additional FliM and FliN markedly improved the swarming of the 202W and 225W mutants (Fig. 3). The other assembly-defective mutants (128W, 147W, and 158W) were not helped by additional FliM and FliN. Several CCW-biased mutants were also tested, but none showed any improvement in swarming upon overexpression of FliM and FliN (data not shown; the mutants tested were 117W, 125W, 191W, and 214W).

CheY is the signaling protein that, when phosphorylated, promotes CW rotation of the motor and tumbling of cells. To determine whether additional CheY could improve the function of the CCW-biased, smooth-swimming mutants, the mutants were transformed with a plasmid allowing arabinose-regulated overexpression of CheY. The 117W, 125W, and 214W mutants, which exhibited the strongest swarming defects, were not helped by extra CheY. The 152W and 191W
mutants, which exhibited milder swarming defects, were much improved by the extra CheY, swarming at about one-third of the wild-type rate (Fig. 3).

**Binding to FliM.** A GST pull-down assay was used to determine whether the EHPQR motif and hydrophobic patch function to bind FliM. The experiments employed a fusion of glutathione S-transferase to the amino terminus of FliM and procedures used previously in a study of FliG-FliM interaction (47). Wild-type FliG was reproducibly coisolated with GST-FliM in this assay. Three mutations in regions of the C-terminal domain apart from the hydrophobic patch (residues 243, 284, and 310) did not measurably weaken the FliG-FliM interaction, as evidenced by a similar yield of coisolated FliG. Binding was greatly reduced (to 10% or less of the wild-type level) by the Trp replacements at positions 128, 181, 202, and 225 and was partially reduced (to about half of the wild-type level) by the replacements at positions 170 and 202 (Fig. 4).

**DISCUSSION**

**Regions that tolerate mutation.** Trp replacements were tolerated in many positions in the C-terminal domain of FliG, including the charge-bearing ridge that has been implicated in electrostatic interactions with the stator (56, 61). Previous mutational studies showed that while the charged residues of the ridge are collectively important, no single residue is indispensable for rotation (28, 60, 61). The ability of the ridge to tolerate

![Diagram](http://jb.asm.org/)
Trp replacements indicates that the detailed topography of the ridge also is not critical. Several Trp replacements on the sides of the domain also had no measurable effect on flagellar assembly or function, as evidenced by normal rates of swarming. We conclude that these side surfaces of the C-terminal domain do not participate in functionally important binding interactions. This finding is consistent with recent cross-linking results, which support a model in which adjacent FligC domains are spaced somewhat apart from each other but with no other protein in between (31).

**Importance of the EHPQR motif and hydrophobic patch.** As expected from their high degree of conservation, both the EHPQR motif and the hydrophobic patch were found to be important for flagellar assembly and function. Tryptophan replacements in these regions either disrupted flagellar assembly or altered the CW/CCW bias of the motor. These findings are in agreement with previous studies of spontaneous fliG mutants, initially isolated in Salmonella (20) and subsequently characterized further in E. coli (29, 62). Most spontaneous fla mutations in E. coli fliG encoded replacements in or near the EHPQR motif (at positions 125, 128, 129, and 132) or in the hydrophobic patch (at positions 201, 202, and 219) (29, 62). Defects in flagellar assembly were also seen for some Trp replacements in the middle domain on the face opposite the EHPQR motif (positions 147, 158, and 165) (Fig. 2). These fall along a shallow cleft lined by several residues with conserved hydrophobic character (Leu 146, Met 158, Ile 161, and Phe 164 in E. coli, corresponding to Leu 147, Leu 159, Ile 162, and Leu 165 in T. maritima). This surface of the middle domain might also participate in functionally important contacts. However, because a substantial portion of this domain (>100 residues) is missing from the crystal structure, this surface of the domain could be buried in the intact protein and the replacements in this region might affect function by altering the overall domain structure rather than by disrupting an important interaction. By contrast, the EHPQR motif is polar and is almost certain to be on the protein surface, where it could engage in functionally important interactions.

A previous study using the two-hybrid system in yeast indicated that both the middle and C-terminal domains of Flig participate in binding to Flim (34). The present mutational results identify the EHPQR motif and hydrophobic patch as likely candidates for interaction with Flim. Trp replacements in these regions gave strong phenotypes and caused a substantial reduction in binding to Flim as assayed by GST pull-down experiments (Fig. 4). The overexpression experiment also points to an interaction through the hydrophobic patch; two hydrophobic-patch mutants that showed strong motility impairment when Flim levels were wild type swarmed well when Flim and Flin were overexpressed (Fig. 3). The Flim-binding site may also include parts of the interdomain helix in Flig, because a mutation in residue 181 also weakened the interaction. Interactions in this region of Flig appear less critical for flagellar assembly, however, because the 181W mutation did not impair swarming (Table 1).

**Model for subunit organization in the switch complex.** A variety of evidence indicates that Flig is located in the upper part of the C ring (16, 45, 48, 61). As discussed previously (31), previous data were consistent with either of two locations for Flig, one with the C-terminal domain at the outer edge of the C ring and the other with Flig in a more “inboard” location (Fig. 1). In the more inboard location, the middle domain of Flig would be located at the lower edge of the MS ring, quite distant from the parts of the C ring that might contain Flim. The binding of Flim to the EHPQR motif therefore argues against the inboard location and instead supports the “outboard” location shown in Fig. 5A. Figure 5 also presents a model for the overall organization of Flig, Flim, and Flin. This model accounts for the present results and appears to be
consistent with all available mutational, cross-linking, and electron microscopic data.

A major feature of the model is that FliM is positioned between FliG and FliN, to account for the finding that FliM interacts with both FliG and FliN (34, 35, 47, 51, 52, 57). The space occupied by FliM in the model has a height of about 5 nm. This is similar to the ca. 5.3-nm long dimension of the major domain of FliM, determined in a recent crystal structure of the T. maritima protein (38). The crystal structure shows that the short dimension of the domain is about 2.5 nm, which is comparable to the thickness of the C ring wall in this region. The intermediate dimension of the FliM domain, ca. 4 nm, is very close to the spacing between adjacent units seen in bottom views of the C ring, and targeted cross-linking experiments showed that adjacent FliM subunits are in contact along this intermediate dimension (38).

Given its shape and size, a single FliM subunit appears to be unable to contact both the EHPQR motif and the hydrophobic patch simultaneously. Although the two domains of FliG might be arranged somewhat differently in the flagellum than in the crystal, there does not appear to be an accessible conformation that brings the EHPQR and hydrophobic patch close together, and the structures seen by electron microscopy show the domains separated by about 5 nm (48, 49). Accordingly, we suggest that some of the FliM subunits in the C ring interact with the EHPQR motif while others interact with the hydrophobic patch. The C ring contains about 34 copies of FliM but only about 26 copies of FliG. This mismatch implies that the FliM subunits in the flagellum cannot all occur in strictly equivalent, symmetry-related environments. The proposed subunit arrangement is consistent with (and might be the reason for) this difference in FliG and FliM copy numbers. We propose that 26 copies of FliM bind to the hydrophobic patch on the C-terminal domain, while the remaining 8 are tilted inward to interact with the EHPQR motif on the middle domain, as detailed in Fig. 5B and C. This would account for the binding data and is also consistent with the electron microscopy images, which show most electron density in the C ring occurring under the C-terminal domain of FliG but some density also reaching inward toward the middle domain. We note that two molecules of FliM might act cooperatively to bind FliG, because adjacent FliM subunits are also likely to interact with each other.

Switching. Previously, we proposed that CW/CCW switching might involve FliM-regulated movements of the FliG C-terminal domain relative to the middle domain (7). The subunit arrangement proposed here is consistent with such a switching mechanism. The phenotypes of Trp replacements are also in accord with the model. Motor bias was affected by certain Trp replacements in and near the EHPQR motif (positions 117, 152, and 125), in the hydrophobic patch (positions 196 and 214), and also in the waist between the upper and lower parts of the C-terminal domain (position 235). Mutations in the EHPQR or hydrophobic patch might alter the relative positions or orientations of the domains by directly altering the FliG-FliM interface. The mutation at position 235 might alter the orientation of the upper part of the C-terminal domain relative to the lower part, moving or reorienting the charge-bearing ridge. This region of the domain is fairly narrow and does not appear to impose any strong structural constraints between the upper and lower parts. As noted above, regions on the interdomain helix might also participate in contacts with FliM. One bias-altering Trp replacement occurred on the bottom of the interdomain helix, at the end nearest the hydrophobic patch (position 191), and several spontaneous mutations affecting switch bias also encoded replacements on the interdomain helix (43). While they might be important for switching, these contacts involving the interdomain helix are evidently less important for flagellar assembly.

In summary, the mutational results, in conjunction with data from previous mutational, binding, and structural studies, lead to a specific model for subunit arrangement in the switch complex. Although the molecular movements responsible for direction switching are not yet precisely defined, the available data are consistent with a model in which the C-terminal domain of FliG moves relative to the middle domain, altering the rotor-stator interface by altering the position or orientation of the charge-bearing ridge.

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