

# Bacteria that Glide with Helical Tracks

# Minireview

Beiyuan Nan<sup>1</sup>, Mark J. McBride<sup>2</sup>, Jing Chen<sup>3</sup>,  
David R. Zusman<sup>1</sup>, and George Oster<sup>1,\*</sup>

Many bacteria glide smoothly on surfaces, despite having no discernable propulsive organelles on their surface. Recent experiments with *Myxococcus xanthus* and *Flavobacterium johnsoniae* show that both of these distantly related bacterial species glide using proteins that move in helical tracks, albeit with significantly different motility mechanisms. Both species utilize proton-motive force for movement. Although the motors that power gliding in *M. xanthus* have been identified, the *F. johnsoniae* motors remain to be discovered.

## Introduction

*“Physics can tell us what cannot happen, and it can tell us what could happen. But only experiments tell us what does happen.”—A. Katchalsky*

The most common propulsive machine for bacteria is the flagellum, propelling cells by rotary motors powered by transmembrane ion motive potential [1]. Since bacteria are very small, they live in a ‘low Reynolds number’ world where inertial forces are negligible [2]. Thus, they cannot swim using ‘reciprocal’ motions; that is, motions that when filmed look the same when the film is run backwards. To solve this problem, bacteria like *Escherichia coli* swim using stiff rotating helical flagella [1,3]. Most spirochetes have helically shaped internal flagella that are located in the periplasmic space between the inner membrane and the cell wall. In these bacteria, the rotation of the periplasmic flagella allows them to swim by generating backward-moving waves [4].

Flagellar motility is effective for bacteria that swim in water, and even allows some to ‘swarm’ over very moist surfaces, but what about microorganisms that move on surfaces that are covered with only a thin aqueous film? For these bacteria, two radically different modes of locomotion have evolved: ‘twitching motility’, which involves intermittent, ‘jerky’ cell movements; and ‘gliding motility,’ where the cell motion is smooth. Of course, these terms are strictly descriptive and give no clue as to the underlying physical mechanisms. Twitching motility is driven by the extension, adhesion, and retraction of fibrous cellular protrusions called type IV pili [5–7]. In *Myxococcus xanthus*, this movement is called social or S-motility, since the extended pili stick not only to the substrate but also to other cells, and so are important for coordinated group movements of the bacteria.

Gliding motility, by contrast, is not well understood. In the myxobacteria it is called adventurous or A-motility because it can drive the movement of isolated bacteria, even when pili are not present. These A-motile cells glide slowly at about one body length (~5 μm) per minute, and reverse direction

periodically every 8–14 minutes, suggesting that there is some internal ‘clock’ regulating reversals [8]. A-motility appears to require the secretion of slime; in myxobacteria this includes a viscous polysaccharide gel [9]. An early model for myxobacterial gliding suggested that the movement of the cell was driven by the hydration and extrusion of slime from protein ‘nozzles’ that cluster mostly at the cell poles [9]. However, recent experimental data have led to the suggestion that the motion of internal proteins, rather than the extrusion of polysaccharides, drives cell movement [10–13].

In this review we describe recent progress in understanding the different ways that bacteria, such as *M. xanthus* and *Flavobacterium johnsoniae*, employ helical tracks to glide over surfaces.

## Helical Tracks and Protein Motors

Using high-resolution fluorescence microscopy of moving *M. xanthus* cells, Nan *et al.* [12] demonstrated that a fluorescently tagged form of AgmU, a critical A-motility protein, decorated a helical ribbon that spanned the length of the cells in a closed loop (Figure 1). Astoundingly, these helices appeared to rotate within the cell cytoplasm as the cells moved forward, and when the cells reversed their gliding direction the helices rotated in the opposite direction. These results recalled previously published images that showed that *M. xanthus* cell bodies were helically twisted, as though the cell membrane had been ‘shrink-wrapped’ around a helical cytoskeletal structure [14,15]. Based on these findings, a model for gliding motility was proposed in which helical waves sweep over the cell surface as the helical rotor inside the cell rotates. Could this be the elusive A-motor, ‘pushing’ on the substrate to move the cell forward? Such a mechanism would be similar to that used by snails [16]. The surface waves in snails, however, arise from the neuromusculature of the snail’s mantle, while the waves in gliding bacteria appear to arise from the rotation of an internal helix.

The mechanism of myxobacterial gliding has other similarities to snail movement. The rearward-propagating waves in a crawling snail do not push directly on the substrate, but on the slime that the snail secretes. This slime sticks more tightly to the substrate than to the snail’s foot, and so the propulsive force of the mantle waves that push on the slime is transmitted to the substrate. The same seems to be true for *M. xanthus*, since the slime that the bacteria secrete appears to be necessary for cell locomotion and is present in all the gliding myxobacteria. Moreover, the slime does indeed adhere more strongly to the surface than to the cell [17], allowing the helical waves to transmit the propulsive force to the substrate via the slime. But what makes the internal helix rotate to generate the surface waves?

A careful examination of single *M. xanthus* motor proteins labeled with a photo-activatable version of the mCherry fluorescent tag revealed that they move around a helical track [11,12]. Motor movement is powered by the proton gradient across the cytoplasmic membrane, also referred to as the proton motive force [11]. The motors are composed of the proteins AglR, AglQ and AglS. AglR is related to the well-studied bacterial flagellar motor protein MotA, and AglQ and AglS are similar to MotA’s partner, MotB. In *E. coli*, MotA and MotB form a complex that harvests the proton motive force and drives rotation of the flagellar

<sup>1</sup>Department of Molecular and Cell Biology, University of California, Berkeley, CA 94720, USA. <sup>2</sup>Department of Biological Sciences, University of Wisconsin-Milwaukee, WI 53201, USA. <sup>3</sup>National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, MD 20892, USA.

\*E-mail: [goster@berkeley.edu](mailto:goster@berkeley.edu)



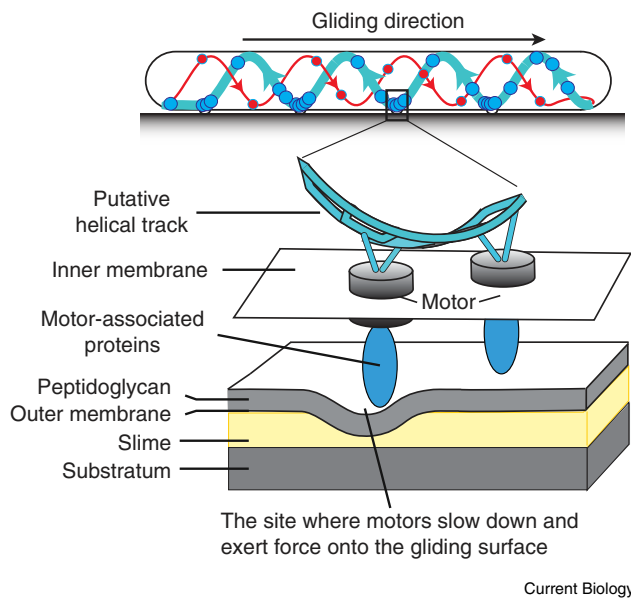


Figure 1. The helical rotor mechanism in *M. xanthus*.

A schematic of the endless helical protein track on which the motors (large circles) walk. The thick lines show the leftward motion of the motors that drive the rightward direction of gliding. The thin red lines show the reduced number of rightward-moving motors on the opposite strand. The model shows that the motors slow down in the ventral traffic jam where they encounter the high drag region. The higher resolution inset shows the motors walking on the cytoplasmic track carrying large cargos of motor-associated proteins that deform the cell wall. The deformation pushes on the external slime, providing the thrust that drives the cell gliding.

filament [18,19]. The MotAB proteins of the bacterial flagellar motor are anchored to the peptidoglycan cell wall, and function as so-called ‘stators’ since they ‘walk in place’ to drive rotation of the flagellum [20]. The myxobacterial motors, however, are not anchored to the cell wall, but are free to move within the cytoplasmic membrane, walking along an apparently helical cytoskeletal track [11]. Thus, the gliding motion of *M. xanthus* operates differently from *E. coli* flagellar swimming.

Here, Newton’s laws of motion come into play. As the motors walk on the helix, their propulsive force exerts an equal and opposite reaction force on the helix. This reaction force can be resolved into two components: one parallel to the cell axis, and one tangential to the cell radius. The longitudinal force pushes the helix in the direction of gliding (towards the ‘leading’ pole), while the tangential force tends to rotate the helix about the cell axis. By Newton’s third law, the tangential force that rotates the helix creates a reaction force that tends to rotate the cell in the opposite direction, but, because the myxobacterial slime is so viscous, there is only a small counter-rotation of the cell. Thus, the motors moving on the helical track rotate the helix. In principle, this could generate a wave of helical deformations on the cell surface to propel the cell, similar to a snail. It turns out, however, that things are not so simple and, before the mechanical picture is complete, we need to address how the rotary force on the helix is transmitted to the cell surface.

### Motor Complexes and Traffic Jams

The images of helically shaped cells mentioned above were prepared by dehydrating the cells, in effect shrink-wrapping

the cell body about the helical cytoskeleton. In a live myxobacterium, the helical shape is not prominent. Therefore, another mechanism must transmit the force exerted by the motors on the rotating helical track to the external surface. Currently, the nature of the helical track is not known, although it may comprise MreB or MreB-associated proteins, since both gliding motility and the movement of motors are sensitive to the MreB inhibitor A22. MreB is an actin homolog involved in many functions, such as the determination of cell shape, cell-wall biosynthesis and chromosome segregation [21–24]. In *E. coli*, MreB forms short ‘patchy’ filaments; the previously reported helical conformation of MreB may be an artifact created by a fluorescent tag fused at its amino terminus [25]. However, the *M. xanthus* MreB filaments appear helical when stained with antibody-conjugated fluorescent dyes, where the fluorescent tag inducing the helical artifact was not used [26]. This implies that the conformation and function of *M. xanthus* MreB differs from its homolog in *E. coli*.

Importantly, Nan *et al.* [11] observed that moving motors in *M. xanthus* always slowed down dramatically when passing through the ventral turn of the track closest to the slime-covered surface and formed accumulations. This is because the surface distortion created by the motor–cargo complex (Box 1) has to push its way through the ventral slime layer where the drag is highest because the slime sticks more to the substratum than to the cell [11].

Thus, motors entering into the ventral region are retarded by ‘traffic jams’ from which they eventually escape to continue on their way towards the trailing cell pole. The traffic jams were visualized using fluorescently labeled motility proteins and appear as nearly stationary clusters to an observer looking at the cell surface. These ventral accumulations were seen earlier by Mignot *et al.* [27], who viewed them as ‘focal adhesions’, analogous to similar adhesion plaques in crawling eukaryotic cells. A motor complex temporarily trapped in one of these traffic jams slows down because it is exerting propulsive thrust on the slime, which transmits the force to the substratum. Since there are about 4–8 turns in the helical track of *M. xanthus* cells (about one turn per  $\mu\text{m}$  of cell length), there are the same number of ventral traffic jams. Accordingly, the thrust developed by the motors in these aggregates act as 4–8 force generators to propel the cell forward. In time-lapse movies it appears as though the motors ‘crawl through’ each traffic jam. When a jam reaches the trailing pole it dissipates as the motors escape, and a new jam is generated at the leading cell pole as motors encounter the increased drag from ventral slime (Figure 1) [12,27]. In moving cells, force generation by traffic jams is invisible since each traffic jam remains almost stationary with respect to the substratum [27,28]. Sun *et al.* [19] visualized the propulsion of the traffic jam sites by attaching polystyrene beads to the surface of cells. In their report, instead of using moving cells, the authors immobilized cells onto a coated glass surface. In this case, beads were propelled from the leading to the lagging cell pole; significantly, the beads co-localized with the traffic jam sites.

But, if the helical rotor is a closed loop, what about the cargo proteins that pass through the trailing pole and commence moving towards the leading pole? Won’t they generate an equal and opposite force and cancel out the propulsive thrust? This would indeed be the case if equal numbers of cargo proteins of similar size moved in opposite directions. But if there were a consistent difference in the

### Box 1

#### The peptidoglycan problem.

Myxobacteria are unusually flexible because single cells are often observed to bend sharply and turn. Although flexible, these cells require a peptidoglycan matrix under their outer membrane that enables them to maintain their cylindrical shape despite the interior osmotic pressure that would otherwise inflate the cell into a sphere, or worse yet, burst the cell. Thus, the integrity of the peptidoglycan is essential. But how then are the motile motors to transmit their propulsive force through the peptidoglycan layer without destroying it?

A plausible solution has been provided by recent experiments by Nan *et al.* (Nan, B., Bandaria, J.N., Guo, Y.K., Yildiz, A. and Zusman, D.R., unpublished data). These experiments show that MglA, a Ras family GTPase that is localized largely at the cell's leading pole, is responsible for assembling AglR motors along with associated 'cargo' proteins (usually large proteins, such as AgmU) onto the helical tracks. These assembled motor complexes can be large enough to deform the peptidoglycan without tearing it. We propose that the rearward motion of the cargo proteins towards the trailing pole will create surface deformations that will slow down when they enter the high drag environment of the ventral traffic jams. It is this viscous coupling of the surface deformation to the substratum via the slime that enables the motors to drive cells forward.

number and/or size of cargo proteins migrating in the opposite direction, then the thrust would not be cancelled out and the cell would move alternately forwards and backwards depending on the relative amounts of cargo moving in each direction at any time, as is observed. Nan *et al.* (Nan, B., Bandaria, J.N., Guo, Y.K., Yildiz, A. and Zusman, D.R., unpublished data) tagged and followed motors cycling on the two strands and found that there was a persistent difference in the number of motors moving towards the trailing and leading poles, with the majority consistently moving opposite to the direction of gliding. If this asymmetric distribution of motors persists, then that would explain the periodic directional reversals. What remains to be explained is the behavior of mutants that glide without reversing. This may occur by the repeated assembly of motor complexes at only one cell pole with recycling occurring by free diffusion. This could also explain the ability of wild-type cells to move in a directional manner in response to a stimulus.

Although the chemistry is still unclear, it appears that the reversals are slave to a biochemical oscillator at the cell poles that couple the motors and their cargo to the helical looped track. The nature of the mechanical coupling remains to be investigated. Cells may gain some control of their movements by breaking free of this rigid oscillation. A model that might explain how this could occur exploits the similarity between the chemotactic signal transduction (Che) system that controls the direction of rotation of the bacterial flagellar motor and the Frz signaling system in myxobacteria that controls cell reversal frequency (Eckhart, E., Davis, A., Oster, G., and Berleman, J.E., unpublished data.).

#### Do Other Gliding Bacteria Move in the Same Way?

Many bacteria belonging to different phyla exhibit gliding motility, and for most of these the mechanisms of movement have not been studied [29]. Some of these bacteria may utilize the helical rotor mechanism described for *M. xanthus*. However, others clearly do not, suggesting that bacterial 'gliding motility' is not a single phenomenon. For example, the wall-less bacterium *Mycoplasma mobile* glides by a mechanism that is powered by ATP hydrolysis rather than by proton motive force [30]. *Mycoplasma* gliding involves large cell-surface proteins that appear to function as 'legs' [31,32]. ATP-driven movements of these legs are thought to allow *Mycoplasma* cells to walk centipede-like over

surfaces. Filamentous cyanobacteria also glide, although the mechanism is not well understood: recent evidence has implicated type IV pili [33] and polysaccharide extrusion [9,34], either or both of which could propel these cells.

*Flavobacterium johnsoniae* (previously called *Cytophaga johnsonae*) is a gliding bacterium for which extensive molecular analyses of motility have been performed. *F. johnsoniae*, a member of the bacterial phylum *Bacteroidetes*, is not closely related to *M. xanthus*. Many members of the phylum *Bacteroidetes* exhibit rapid gliding motility and *F. johnsoniae* is a model organism for studies of this form of movement. There are many parallels between *M. xanthus* and *F. johnsoniae* gliding, suggesting that they might use a common mechanism, but a deeper look also reveals differences. At first glance, *F. johnsoniae* appears to be a supercharged version of *M. xanthus* that glides about 25 times faster. Like *M. xanthus*, cell movement is powered by proton motive force [35,36]. Cells are long and somewhat flexible, and they move in the direction of either pole, occasionally reversing direction. Interestingly, motility of both bacteria seems to utilize a helical track [12,35].

Decades of research on *F. johnsoniae* and on *M. xanthus* have revealed many proteins required for gliding in each organism [37–39]. Surprisingly, however, there are few similarities between these components at the molecular level, suggesting that these systems may have evolved independently. For *F. johnsoniae* we know a great deal about the cell-surface and cell-envelope components, but the motors that drive movement and the links to cytoplasmic components are unclear [40–42]. As discussed above, however, the motors associated with motility in *M. xanthus* have been identified, as have components in the cytoplasm that may interact with and control these motors. By contrast, the components on the cell surface of *M. xanthus* that transmit the force to the substratum are less well understood. Further study of both systems may thus reveal common elements.

Some motility behaviors of *F. johnsoniae* and related bacteria differ from those of *M. xanthus* and are difficult to explain by the helical rotor model. First, cells floating in liquid, or attached to and gliding on a surface, bind to and rapidly propel small particles, such as latex microspheres [36,43,44]. When asymmetrical particles are examined, they often appear to be rigidly attached to the cell surface and maintain the same orientation at their point of attachment as they travel the length of the cell, around the pole, and

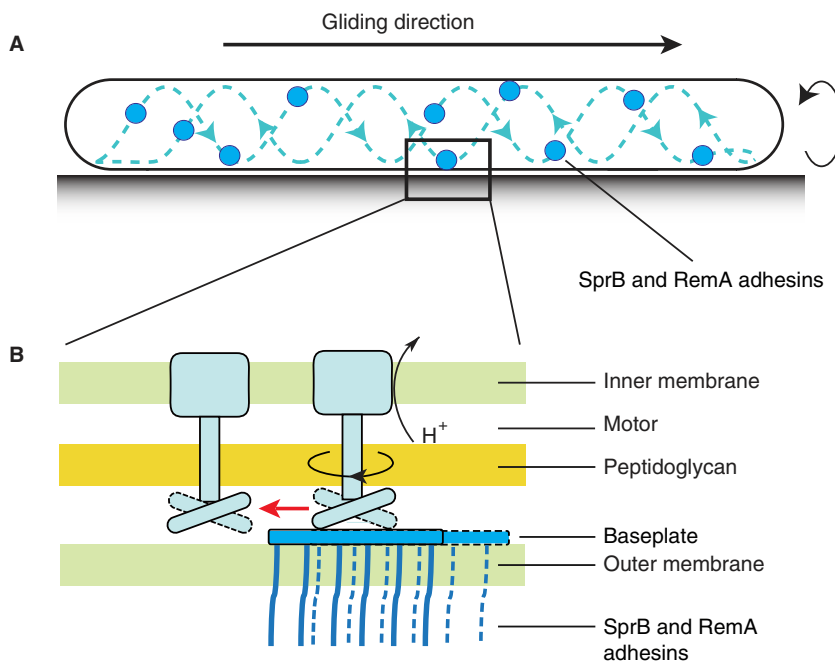


Figure 2. Speculative model for movement of *F. johnsoniae* cell-surface adhesins.

(A) The surface SprB and RemA adhesins move along a looped helical track. (B) A portion of the cell envelope is shown. The motors, anchored to the peptidoglycan, harvest the proton gradient across the inner membrane. A portion of the motor complex extends through the peptidoglycan layer and interacts with an outer-membrane-associated protein (baseplate) that carries the cell-surface adhesins. Repeated movements of this portion of the motor propel the baseplate and attached adhesins along the cell surface until they are engaged by the next motor.

the motor complex could engage the baseplate and propel it a short distance. The same motor could then repeat this process multiple times to move the baseplate and attached adhesins over a longer distance, at which point it could be engaged by another motor complex.

This model leaves unaddressed how the motor is controlled to result in directed movement. In *M. xanthus* a chemotactic signal transduction

system similar to those found in flagellated bacteria is thought to perform this function, but the core components of such systems (the methyl-accepting chemotaxis proteins, the histidine kinase CheA, and the adaptor protein CheW) are lacking in *F. johnsoniae* [38]. The closed helical loop track presents the same problem for *F. johnsoniae* as it does for *M. xanthus*. Since different adhesins move simultaneously toward the front and back of the cell on different regions of the helical track, how does the cell make any net progress? Are only some adhesins actively engaged in binding to the substratum and, if so, how is this controlled? Identification of the actual motor complex and its interactions with other proteins will help to reveal the mechanism of *F. johnsoniae* cell movement and directional control. It may also help to determine whether there are underlying similarities between the myxobacterial and flavobacterial solutions to movement over a surface.

### Conclusions

Bacterial gliding remains largely mysterious, although experiments are beginning to shed light on some mechanisms. Experiments on myxobacteria have provided sufficient evidence to propose a helical rotor model, in which gliding motors move actively along internal tracks. While the rapid movement of cell-surface adhesins is thought to result in *Flavobacterium* gliding, the molecular nature of the motors that propel the adhesins remains mysterious. Nevertheless, it is intriguing that both motility mechanisms are powered by transmembrane ion motive potentials and involve the ubiquitous presence of helical structures.

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back toward the opposite pole. Second, cells gliding on a glass surface often flip end-over-end [35,43]. These observations suggest that outer membrane adhesins are propelled along the cell surface and may drive *Flavobacterium* gliding.

Genetic experiments identified a candidate adhesin — the huge 669 kDa protein SprB [40]. SprB proteins decorated with fluorescently labeled antibodies are propelled rapidly (2  $\mu\text{m}$  per second) along the cell surface on what appears to be a closed helical loop [35]. SprB proteins traverse the length of the cell, loop around the pole, and migrate toward the opposite pole within 5 to 10 seconds. As a cell moves forward, some SprB proteins on the cell surface remain attached to the substratum, suggesting that the action of a motor pushing against these adhesins propels the cell. Dissipation of the proton motive force with chemical uncouplers rapidly and reversibly inhibits cell gliding and SprB movements [35]. *F. johnsoniae* has other motility adhesins — such as the carbohydrate-binding lectin RemA — that are propelled along the cell surface [41]. Secreted polysaccharides that interact with RemA have also been implicated in gliding [41]. These polysaccharides may coat the substratum, and form a ‘road’ that interacts with specific ‘tires’, such as RemA. The ability to use different adhesins, and perhaps different polysaccharides, may explain how *F. johnsoniae* cells can crawl over diverse surfaces.

The motor involved in *F. johnsoniae* gliding is not known, but we expect it to span the cytoplasmic membrane and be anchored to a large structure, such as the peptidoglycan layer. Such a motor complex could have components that reach through the peptidoglycan to propel the outer membrane adhesins. One model envisions large ‘baseplates’ adjacent to the inner face of the outer membrane to which multiple adhesins are attached (Figure 2). The large repetitive protein SprD, which supports SprB function, might form such a structure. There may be multiple positions at which

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