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# MINIREVIEW

# Are There Lateral as Well as Polar Engines for A-Motile Gliding in Myxobacteria?[▽]

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Jonathan Hodgkin investigated many motility mutants derived from the same genetically characterized strain of *Myxococcus xanthus*. Comparisons between mutants revealed two different swarm patterns, indicative of two different gliding engines called engine A and engine S (10, 11). The swarm patterns are known as A motility and S motility. Recent comparisons of whole myxobacterial genome sequences (8, 29, 30) suggest that many, if not all, of the 40 known species have both gliding engines. Although there is general agreement that the S engines, which emerge from the leading end of the cell, are polar type IV pili, the nature of the A-motility engine has become a matter of controversy. Because A motility is used for swarming (15) and to build fruiting bodies (10) and is regulated by the same reversal switch as type IV pili (16), understanding the molecular basis of those processes requires a clarification of the controversy.

Evidence associating A motility with slime secretion will first be laid out because it is the older model. Observing trails of slime that had been deposited on agar by the gliding cells, Jahn suggested that myxobacteria might be propelled by slime secretion (13). As mentioned, Hodgkin isolated mutants specifically deficient in engine A and established that A motility involved a set of genes different from those involved in type IV pilus-dependent S motility. Slime trails are elongating at the same rate at which the cells are moving, as indicated in Fig. 1. The figure shows two gliding cells ($A^+$ $S^+$) with trails behind them; the trail begins at the lagging end of each cell. Tracking the movie shows that the trail lengthens at the same rate as the leading end of the cell advances. The way slime secretion might push a cell forward was clarified by the discovery of slime nozzles in *M. xanthus* that resemble those found in *Phormidium* spp., filamentous gliding cyanobacteria. The measured rate of slime filament extrusion in *Phormidium* was similar to the speed of cell gliding (12). In *M. xanthus*, high-resolution transmission electron microscopy (EM) also revealed long, narrow, amorphous filaments, thought to be extruded slime, emerging from one end of the cell (39). No filaments were seen to emerge from the cell sides or from the leading end of the cell. Moreover, by phase-contrast visible-light microscopy, filaments emerged only from one end of each cell (43). EM also revealed more than 500 thick-walled rings, 80% of which are distributed between the two ends of the cell. Slime filaments emerge in the vicinity of the polar rings. The rings are uniform in size, having an outer diameter of 13 nm and an inner diameter of 6.5 nm. Their size distinguishes them from the PilQ secretin, which has an outer diameter of 16.5 nm (3). Moreover, the rings are observed in Δ*pilQ* mutants (39). Rings are interpreted to be secretory nozzles, viewed end-on, and they resemble the open ends of the amphora-shaped junctional pore complexes observed in *Phormidium* (12). (In *Phormidium*, two sets of pore complexes are seen, one above and another below each junction [12].)

The narrow filaments of slime observed in the EM (39) were observed to fuse laterally with each other to form the single broad ribbon that is visible with phase-contrast illumination (43). At any particular moment, both EM and light microscopy clearly show that the filaments are present only at one of the two cell poles (39, 43). The unipolarity of the extruded filaments of slime matches the instantaneous unidirectional movement of the cells. The phase-bright trail that is left behind when wild-type ($A^+$ $S^+$) cells move (Fig. 1) is extruded slime left behind on the agar. Charles Wolgemuth was able to estimate the magnitude of the secretion force by mechanical analysis of the flailing motion of a cell that became stuck at one end while the other end was free. He found a force between 50 to 150 pN (38), which, being similar to the retraction force of a type IV pilus (20), should be adequate to propel cells.

As mentioned, both EM and light microscopy showed that a single (united) filament emerges from one end of a cell. The opposite end has no filament even though it has more than a hundred nozzles (39). Therefore, many of the 400 polar nozzles are not secreting propulsive slime at any instant that a cell is moving. Two kinds of nonsecreting nozzles can be distinguished: approximately 40% of the total nozzles cannot secrete because they are situated at the leading end of the cell, the wrong end for pushing with slime. The next time the cell reverses its gliding direction, these polar nozzles secrete slime. A second kind of nonpropulsive nozzle is found on the sides of each cell and will be discussed shortly.

At least seven putative biopolymer transport genes with similarity to the TolB, TolQ, and TolR genes have been found to be required for A motility (36, 42), which is suggestive of intracellular transport complexes (9). The idea that nozzles are pores that can be switched on or off arose from Rosa Yu's identification of two classes of genes required for A motility. Yu isolated 33 *Himar* transposon insertions within genes es-

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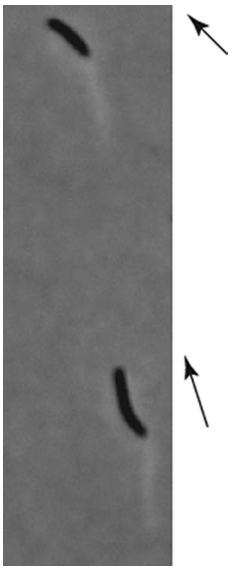

FIG. 1. Two $A^+$ $S^+$ cells gliding on an agarose gel. A single frame selected from a time-lapse movie taken by Lars Jelsbak using a Nikon 40× phase-contrast objective is shown. Cells are phase dark; slime trails are phase bright. Tracking the movie shows that the trails are laid down from the lagging pole and elongate progressively as the cells move over the agarose surface. Because cells reverse their gliding direction every 7 to 9 min, they are often found to be following and elongating their own trails. Arrows indicate the current direction of cell movement which has laid the trail down.

sential for A motility (43). Half ($n = 15$) of the mutants were nonmotile, and all these mutants were found to be secreting slime filaments from both ends simultaneously and not from their sides. Several inferences were made. First, polar nozzles are conditionally active: they can be switched from a secreting to a nonsecreting state and back to a secreting state. Second, in order for cells to glide, slime must be secreted only from one pole at a time. Third, the 20% of nozzles that are found on the sides of cells (nonpolar nozzles) do not secrete filaments of slime. That failure is correlated with a very low surface density, roughly 1/150 the density of nozzles found at the cell ends. If the nonpolar nozzles are uniformly distributed over the cell's surface and if they were to secrete constantly, the resulting secretion could cover the entire surface of the cell. It would form the capsule of *M. xanthus*, protecting it from the also-secreted extracellular digestive enzymes. This, after all, is the vital function of the capsule in all gram-negative bacteria (37), and it could explain why no $A^-$ mutant that fails to produce slime has ever been found. Such a mutation would be lethal (17).

A second half (18/33) of Yu's A-motility mutants were motile, and they all produced slime from one end only (in line with the second inference above). Although these mutants were motile, and had normal reversal frequencies, individual cells moved at a consistently lower speed than that of the $A^+$ cells. For that reason, they were called *pgl* for *p*artial *gl*iding motility. Both the rate of swarm expansion and the coefficient of elasticotaxis were lowered in the *pgl* mutants. The two measures relate to different physical qualities; the fact that they stood in direct proportion to each other suggested that they arose from quantitative defects in the same pathway. The elasticotaxis coefficient measures a cell reorientation response to elastic stress in agar gels (7, 32). The swarm expansion rate is proportional to the average cell speed (40). The DNA sequences of 13 of the 18 (*pgl*) motility genes could be obtained and their functions inferred from the NCBI database. Nine were annotated as sugar transferase or polysaccharide polymerase genes (43). The following three were annotated as regulatory protein genes: one was *mglB*, which is part of the MglAB reversal switch and long known to be partially motile (33); one was $tRNA^{Asp}$; and one was an alanine racemase gene (43). Finally, 1 of the 13 had no significant match to a gene in the NCBI database (43). Multiple sugar transferases and polymerases point to polysaccharide biosynthesis and suggested differences in sugar sequences between Pgl mutants, whereas the regulatory proteins suggested its control. None of the *pgl* genes (except *mglB*) had previously been identified. It is proposed that slime propulsion involves the secretion of large amounts of capsular slime through the nozzles at the lagging cell pole.

## NOZZLE PROTEIN CANDIDATES

The annotated *M. xanthus* genome includes 550 genes for the synthesis of the cell envelope—a substantial fraction of the 7,000 total genes (8). Polysaccharides isolated from *M. xanthus* contain a variety of sugars (35). Many of the envelope polysaccharides are repeat-unit polysaccharides (RUP), like its O-antigen lipopolysaccharide (19) and its fibril polysaccharides (1), also called exopolysaccharides (18). *Escherichia coli* capsular RUPs are synthesized in large multisubunit complexes of glycosyl transferases associated with a specific secretory pore that has been visualized (27). Since each RUP includes its own set of glycosyl transferases and since each transferase is sugar specific, the *M. xanthus* genome would be expected to include many glycosyl transferase genes. This was confirmed by a scan of a contiguous one-quarter of the genome (by the author) that turned up 11 glycosyl transferase genes and 9 other genes plausibly involved in polysaccharide biosynthesis. Because glycosyl transferases are numerous, the absence of the correct transferase in a *pgl* knockout mutant might exhibit partial A motility when a glycosyl transferase from a different RUP became associated with the secretory pore. One example is PglB, whose amino acid sequence belongs to the large group 1 family of glycosyl transferases (Pfam00534). When the PglB protein is missing, its place could be taken by another group 1 transferase normally involved in the biosynthesis of a different RUP that happens to fit (albeit imperfectly) into the slime RUP synthesizing/secreting enzyme complex. Because the incorporation of a mitigating transferase into the complex for slime biosynthesis is likely to be imperfect and unstable and will possibly introduce the wrong sugar, such incorporation would lower the synthetic rate. Slow synthesis might be expected to decrease the swarm expansion rate and decrease the elasticotaxis coefficient in direct proportion to each other, because they would have arisen from the same chemical change (43).

Established RUP biosynthesis includes within a single multiprotein complex enzymes that export as well as enzymes that polymerize the polysaccharide chains. Best studied are the Wzx, Wzy, and Wba proteins of group 1 capsular polysaccharides in *E. coli* (5, 6). Their biochemical specificities are dia-

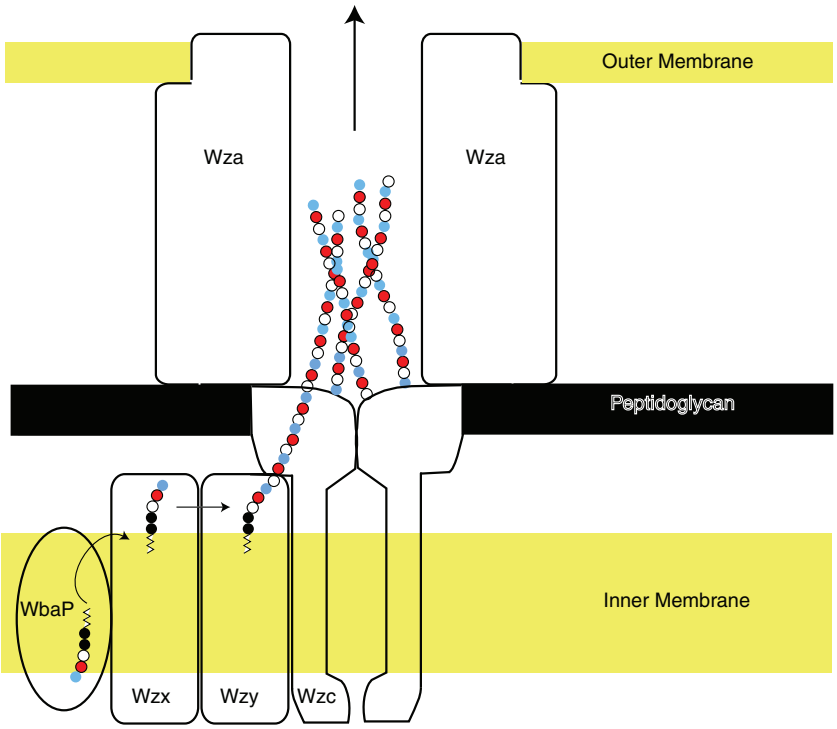


FIG. 2. A proposed structure of the polar slime secretion nozzles, based on *pgl* mutants that decrease slime synthesis, and on the X-ray structure of the Wza translocon. The translocon forms a large chamber within the outer membrane, open to the outside. The amino acid sequence of each protein, identified by transposon insertion to be required for A motility, was compared with the public protein databases to infer its biochemical function. The *pglN*, *pglB*, and *pglD* genes resembled constituents of the WbaP complex most closely. This complex, which is localized in the inner membrane, synthesizes an oligosaccharide repeat unit. Once synthesized on the cytoplasmic face of the inner membrane, the repeat unit is then "flipped" from the cytoplasmic to the periplasmic side of the inner membrane by the integral inner membrane protein Wzx (most similar to PglF). A polymerase Wzy (possibly PglJ) completes the slime polymer by the serial addition of repeat units to the base of the growing chain. Polysaccharide chains are transported by Wzc into the water-filled Wza chamber, located in the outer membrane. Although the *E. coli* chamber has eightfold symmetry, the slightly larger chamber in *M. xanthus* is diagrammed with four chains so that its Wza would produce a gel ribbon instead of the single *E. coli* polysaccharide chain. Interactions between the polysaccharide chains and their binding of water would ensure the formation of the hydrogel. Reprinted from reference 17 with permission of the publisher.

grammed in Fig. 2, and potentially homologous functions are paired in the figure legend. PglF is predicted to have two glycosyl transferase domains: one in the N-terminal half and one in the C-terminal half (43). Typically each biosynthetic step in RUP synthesis requires a distinct membrane-localized glycosyl transferase that hands its products off to another glycosyl transferase for the next step (27). The structure of PglF, which includes two different glycosyl transferase domains, strongly suggests such a handing-off from one transferase domain to the other. In particular, the C-terminal glycosyl transferase domain of PglF resembles those that transfer the sugar from a sugar nucleotide to a range of acceptors that include dolichol phosphate. Growing polysaccharide chains are generally anchored to the inner membrane by undecaprenylphosphate (27), and PglF may catalyze the formation of an undecaprenylphosphate-oligosaccharide.

Figure 2 shows a cylindrical protein vessel that opens through the outer membrane representing Wza. The *E. coli* Wza structure has been determined by a combination of protein crystallography and electron cryomicroscopy with negative staining (2, 4). The inner diameter of the Wza vessel is about 60 Å, just a bit smaller than the 65-Å inner diameter of the slime nozzles as determined by negative staining. Crystallographic and EM studies of Wza show it to be an octamer (2, 4). Although sequence homologs of Wza and Wzc have yet to be identified in *M. xanthus*, the size of the nozzle rings in *M. xanthus* imply that, as suggested in Fig. 2, a slightly larger *M. xanthus* Wza vessel should be able to hold several polysaccharide chains within the long cylindrical chamber, where those chains would become entangled as they bound water. The *E. coli* Wza vessel is thought to hold a single polysaccharide chain. It is thus plausible that a long, topologically entangled, ribbon-shaped hydrogel like those observed in Hoiczyk's electron micrographs (39) would be secreted from an enlarged vessel in *M. xanthus*. The Wza-Wzc complex employed in *E. coli* capsule formation is a long cylinder that extends from the cytoplasm to an orifice in the outer membrane (Fig. 2). After a monomer repeat-unit oligosaccharide is assembled on an undecaprenyl diphosphate lipid carrier on the cytoplasmic face of the inner membrane, as shown for the WbaP complex in the figure, that unit is then transported (flipped) from the cytoplasmic side of the inner membrane across the inner membrane to that membrane's periplasmic side by a Wzx flippase protein. A homolog of Wzx has been shown to flip isoprenyl-PP-GlcNAc in vesicles (28). Once across the inner membrane, the lipid-linked oligosaccharide becomes the monomeric substrate for a character-

istic Wzy-dependent polymerization (6). When viewed from outside the cell, a Wza-Wzc protein complex should appear like the thick-walled rings that Wolgemuth et al. have associated with polar slime secretion in *M. xanthus* (39).

Polysaccharide secreted through Wza in *E. coli* forms a gel layer that covers the whole cell. According to Whitfield (37), the primary function of a capsule is to bind water from the environment, which maintains the cell in a hydrated state. Most likely, the sugar sequence of the capsule helps the polysaccharide chains cover the entire cell surface and absorb water for the cell. The ability of capsular slime to bind environmental water supplies the precedent for the slime propulsive force in *M. xanthus*. Force arises from the hydration of the secreted polymer (39). Water that each sugar residue had picked up in the cell's cytoplasm will have been partially stripped off as each group of residues is flipped by Wzx through the (hydrophobic) inner membrane into a dry region of the periplasm (possibly a crevice within a globular protein component of the Wza-Wzc complex). Accordingly, the sugar sequence of the slime polymer is expected to have maximized the amount of water bound by slime in soils where *M. xanthus* is found. The same sequence would also be expected to maximize the amount of gel expansion per sugar residue within the chamber formed of Wza protein that opens to the environment. Mutations of Wza in *E. coli* do not result in the accumulation of nonsecreted polymer, and this is taken to indicate a direct coupling between export and polymerization that is necessary to keep the sugar dry before the polymer chains are secreted (24). The coupling of export to polymerization in *M. xanthus* would account for the pauses that are observed in the secretion of slime by the *pgl* mutants that lack a particular glycosyl transferase involved in polymerization. To test this mechanism of propulsion, the nozzle proteins should be identified and the polysaccharide sequence should be obtained.

## ANOTHER A-MOTILITY ENGINE?

Recently, a new model of A-motility force generation was proposed. It grew from the discovery by Mignot et al. of focal adhesion complexes containing AglZ protein that should play a role in A motility (23). Strikingly, the focal adhesions remained fixed in position with respect to the agar substrate as the cell body moved forward. AglZ is a filament-forming coiled-coil protein that is specific for A motility and plays no role in S motility (41). Moreover, the adhesions are transient; they form at the leading cell pole, become distributed along the cell length as the cell moves forward, and disappear from the trailing half of the cell (23). Focal adhesions would provide rigid, but transient, connections between AglZ filaments within the cell and the agar substrate. Mignot et al. elaborated on the discovery by suggesting that each focus was associated with an as-yet-uncharacterized motor that propelled the cell forward by pulling on an unidentified element of the cytoskeleton (23).

Another investigation of the movement patterns of filaments generated by treating cells with cephalexin (cefalexin) led to the suggestion made by Sliusarenko et al. that the A-motility engine is not localized to the lagging pole but is distributed along the length of the cell, as in the model of Mignot et al. (31). The suggestion of a distributed engine was based on the assumption that cephalexin would have blocked the synthesis of slime nozzles in the vicinity of the sites where septa would have formed in the absence of the antibiotic. Cephalexin, acting like penicillin, is thought to bind the peptide cross-links between cell wall glycan chains. Sliusarenko et al. did not verify the correctness of that assumption, despite the fact that the work establishing the inhibition of septation by cephalexin had shown that one to four preseptal FtsZ rings formed in treated *E. coli* cells (25). If multiple FtsZ rings formed in the presence of cephalexin, why shouldn't slime secretion nozzles have formed as they do in *Phormidium* (12), at the sites where septa should have formed? Moreover, Sun et al. reported that long cephalexin-induced filaments were able to separate into individual, normal-size cells when the antibiotic was removed from non-growth medium (34). Since nutrients were not required for completing cell division and cell separation, it would seem that the filamentous cells had already synthesized protein structures normally associated with the septum, such as the slime-secreting nozzles.

In addition to a lack of experimental verification, the distributed lateral-engine model has several unresolved difficulties. Since the focal adhesion sites appear to lie on a helix (23), which would account for their periodicity, the cell would need to rotate about its long axis as it progressed over the substratum. But that needed rotation has not been demonstrated, and a rotation mechanism would be nontrivial for several reasons. First, many of the images from the work of Mignot et al. show that the cells are often bent in several places. The rotation of a bent cell about its long axis would require the cell either to straighten out by sliding to the side or to break one or more of the focal adhesions, neither of which has been observed. Second, *M. xanthus* cells extend several type IV pili forward. If two pili from the same cell were to attach to cells ahead, cell rotation would wind them around each other, which ought to oppose pilus retraction. Thus, the distributed motor model of A motility predicts that A and S motilities should compete with each other. Instead, it has been found that the two engines work together and enhance each other (14). Third, the distributed motor model overlooks the question as to how the adhesion motor complexes might encode the direction that the cell should be moving, which would have to be the same for all the adhesions on a cell. The directional indicator would specify which of the cell's poles should be leading and which trailing. Although the filaments of AglZ protein that assemble at each adhesion site can produce striated arrays (41), striations indicate only that the filaments are aligned in register. There is no sign of a polarity in that alignment that could specify the proper direction for gliding. Finally, the recent discovery that *frzCD aglZ* double deletion mutants retain their A motility shows that AglZ is not essential for A motility but regulates it instead (21, 22). The findings of Mauriello et al. eliminate an important motivation for assigning motor activity to AglZ.

Considering these observations, is there some reason to favor the hypothesis of Mignot et al. that motors are associated with the adhesion complexes? If not, there should be some other reason behind the formation of focal adhesions. Might focal adhesions play a role in propulsion by polar slime secretion? Consider a cell that is gliding on its layer of capsular slime, which is the interface between the cell's lipopolysaccharide and the agar substrate, as illustrated in Fig. 3A. As that cell is pushed forward by slime secretion from its lagging end,

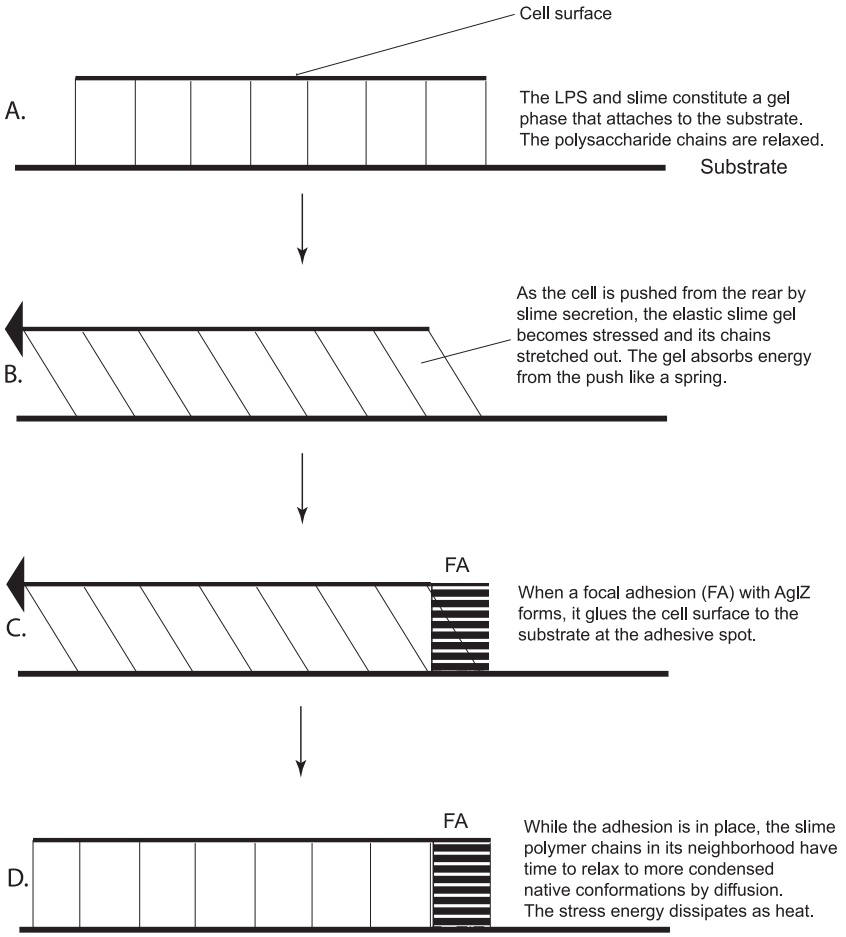


FIG. 3. Proposed role for focal adhesions. The diagram shows the cell surface and the gel layer, composed of lipopolysaccharide (LPS) and capsular slime, which attaches the cell to the substrate. (A) Relaxed conformation of the gel found before movement. (B) Stressed conformation resulting from slime secretion at the lagging pole that pushed the cell in the direction of the large arrowhead. Like a spring, the gel has absorbed energy from the push, which is represented by the slanting lines. (C) A focal adhesion forms that rigidly links the cell to the agar. It prevents the elastic strain energy in the gel from pushing the cell backward. (D) While the adhesion is in place, the slime polymer chains have an opportunity to relax to more condensed conformations by diffusion.

the slime chains would be stretched out from native, condensed conformations that are indicated by slanting the lines that represent the gel (Fig. 3B). These motions are taking place at a very low Reynolds number; the cell is slowly pushed by its own slime secretion from its lagging end. Under these conditions, viscous forces dominate, and momentum plays no role (26, 38); consequently, the stress would oppose the cell's forward motion. The formation of a focal adhesion (Fig. 3C) might allow the accumulated stress to be dissipated by the diffusive relaxation of the slime polysaccharide chains to more condensed (native) conformations (Fig. 3D). The stress energy dissipates as heat. Without the dissipation of the stress energy, forward motion due to polar slime secretion would slow and possibly come to a stop. Focal adhesions are separated from each other, are few in number, and are lost, because once the polymer chains have relaxed, focal adhesions (Fig. 3) are no longer needed and the cell is ready to form new adhesions forward of the focal adhesions. Thus, focal adhesions would serve as molecular ratchets that allow the cell to glide continuously in one direction for each cycle of reversal. According to this view, focal adhesions are sites of energy dissipation, rather than sites at which translational energy is somehow injected. In sum, the translation of *M. xanthus* across a surface can be produced by pulling with type IV pili that are strong enough to break any adhesions or by polarized secretion that pushes the cell through the adhesions. For the A-motility model proposed here, it should be emphasized that propulsive slime is chemically the same as capsular slime but is physically different because it is aligned by the dense, polar array of nozzles that secrete it.

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