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► **To cite this version:**

Julien Herrou, Dorothée Murat, Tam Mignot. Gear up! An overview of the molecular equipment used by Myxococcus to move, kill, and divide in prey colonies. *Current Opinion in Microbiology*, 2024, 80, pp.102492. 10.1016/j.mib.2024.102492 . hal-04778453

HAL Id: hal-04778453

<https://hal.science/hal-04778453v1>

Submitted on 12 Nov 2024

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Review

Gear up! An overview of the molecular equipment used by *Myxococcus* to move, kill, and divide in prey colonies

Julien Herrou, Dorothee Murat and Tâm Mignot



Myxococcus relies on motility to efficiently invade and predate a prey colony. Upon contact with prey, *Myxococcus* temporarily halts its motility and initiates prey cell lysis, which involves two contact-dependent predatory machineries, the Kil system and the needleless T3SS*. Predatory cells grow as they invade and feed on prey cells. When dividing, *Myxococcus* cells systematically pause their movements before division. This highlights a high level of co-ordination between motility and contact-dependent killing but also with cell division. In this review, we give an overview of the different nanomachines used by *Myxococcus* to move on surfaces, kill by contact, and divide, and we discuss the potential regulatory mechanisms at play during these different processes.

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Current Opinion in Microbiology 2024, 80:102492

This review comes from a themed issue on **Special section on Bacterial Nanomachines**

Edited by **Martin Thanbichler** and **Susan Schlimpert**

For complete overview of the section, please refer to the article collection, "[Special section on Bacterial Nanomachines 2024](#)"

Available online 5 June 2024

<https://doi.org/10.1016/j.mib.2024.102492>

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Introduction

In the wild, micro-organisms are constantly battling for survival in inhospitable conditions. They have thus developed various strategies to persist and thrive in diverse ecosystems, particularly in those that are scarce in nutrients and rich in competitors, such as soils. In this context, many micro-organisms have evolved the capacity to find nutrients by predated on their close neighbors [1,2].

While eukaryotic micro-predators have long been thought to account for most of bacterial predation in soil

ecosystems, in recent years, it has become clear that bacterial predators are highly prevalent in these ecosystems too [3]. Predatory bacteria are distributed in a wide variety of phyla and environments [4], and bacterial predation plays a key function in shaping the soil microbial foodweb [5].

Myxobacteria are the best studied group of predatory bacteria and have recently been reported as possibly being the dominating bacterial soil predators [3,6]. *Myxococcus xanthus* (referred to as *Myxococcus* in the text) is the model bacterium representative of this group. It is genetically tractable, and most of its life cycle has been characterized, at least partially, at the molecular level [7–10]. As a predator, *Myxococcus* can prey on many different micro-organisms [11–14] and is known to attack its prey in a so-called wolf-pack manner, using surface motility to invade the prey colonies. Motility involves the A- and S-motility complexes, two distinct systems with complementary functions, allowing predatory cells to co-ordinate during prey invasion (see below) [8,14–17].

As a powerful secondary-metabolite producer (biosynthetic gene clusters represent 10% of its genome), *Myxococcus* predation capacity was initially mostly attributed to the combined action of secreted toxic factors (outer membrane vesicles, antibiotics, toxic proteins etc.) in its environment [11,16,18–21]. While the contribution of a toxic secreted cocktail is demonstrated, it was recently shown that predation also depends on the direct contact of *Myxococcus* with its prey and involves two membrane-bound machineries, namely, the Kil system and a needleless-T3SS machinery (T3SS* in the text), required for contact-dependent prey intoxication and lysis [14,22,23]. This way, predatory cells trigger prey content release and use the prey macromolecules as nutrients to fuel growth of the collective [14,23,24].

At the cellular level, the predation process needs a high level of co-ordination between motility and contact-dependent killing but also with cell division. First, during killing, the cells systematically put their motility on hold upon contact with prey cells until lysis, typically 2–3 min after contact [14,22,23]. Second, while *Myxococcus* cells feed off of the prey remnants and grow, they also pause motility in the very late stage of the cell cycle [14,25]. In this process, the daughter cells resume their movement

shortly after cytokinesis. At the molecular scale, these regulations involve the co-ordination of the two A- and S-motility complexes, the Kil- and T3SS*-complexes and the divisome. In this review, we describe these nanomachines and discuss how these systems might be uniquely regulated during predation.

Prey invasion requires the concerted action of two motility machineries

To move on surfaces, *Myxococcus* uses two distinct types of motilities (Figure 1a). The first strategy, referred to as Social (S) motility (*twitching*), relies on a Type-IVa pilus (T4aP) machinery assembled at the leading pole and enabling cells to move collectively in a co-ordinated manner. Motion is powered by the extension and retraction of pilin fibers that pull on surfaces such as grappling hooks [26,27]. The second strategy, known as Adventurous (A) motility, involves a transmembrane machinery called the Agl-Glt complex. This complex assembles at the leading pole and moves toward the lagging cell pole, propelling cells as it becomes anchored to the external surface, forming so-called bacterial Focal Adhesion (bFA) complexes [26–28].

The two motility complexes provide a strong predatory advantage to *Myxococcus* cells. Both A- and S-motile *Myxococcus* are able to kill prey cells, but prey colony invasion is facilitated by A-motile cells that detach from the predation front to kill prey cells by contact and open large breaches in which larger groups of cells can infiltrate [14,15]. Remarkably, S-motile groups tend to follow paths opened by A-motile cells, and thus, there appears to be an important cross-talk between A- and S-motile cells during prey invasion [15]. Importantly, contact-dependent predation can also occur in liquid media. In this condition, T4aPs alone allow *Myxococcus* to trap the prey cells into a tight biofilm, then digesting it by contact [14,29]. In this review, considering the importance of A-motility during predation, we will essentially focus on the Agl-Glt complex.

The ‘Agl-Glt’ A-motility complex

The Agl-Glt complex is a predicted cell envelope apparatus dynamically coupling two membrane-associated machineries to propel the cells as they interact with surfaces. While genetic approaches identified up to 17 proteins forming this complex (Figure 1a) [30], there is currently very limited data regarding the exact protein interactions that take place within this apparatus. Nevertheless, several critical components of the motility complex have been characterized, which we present here.

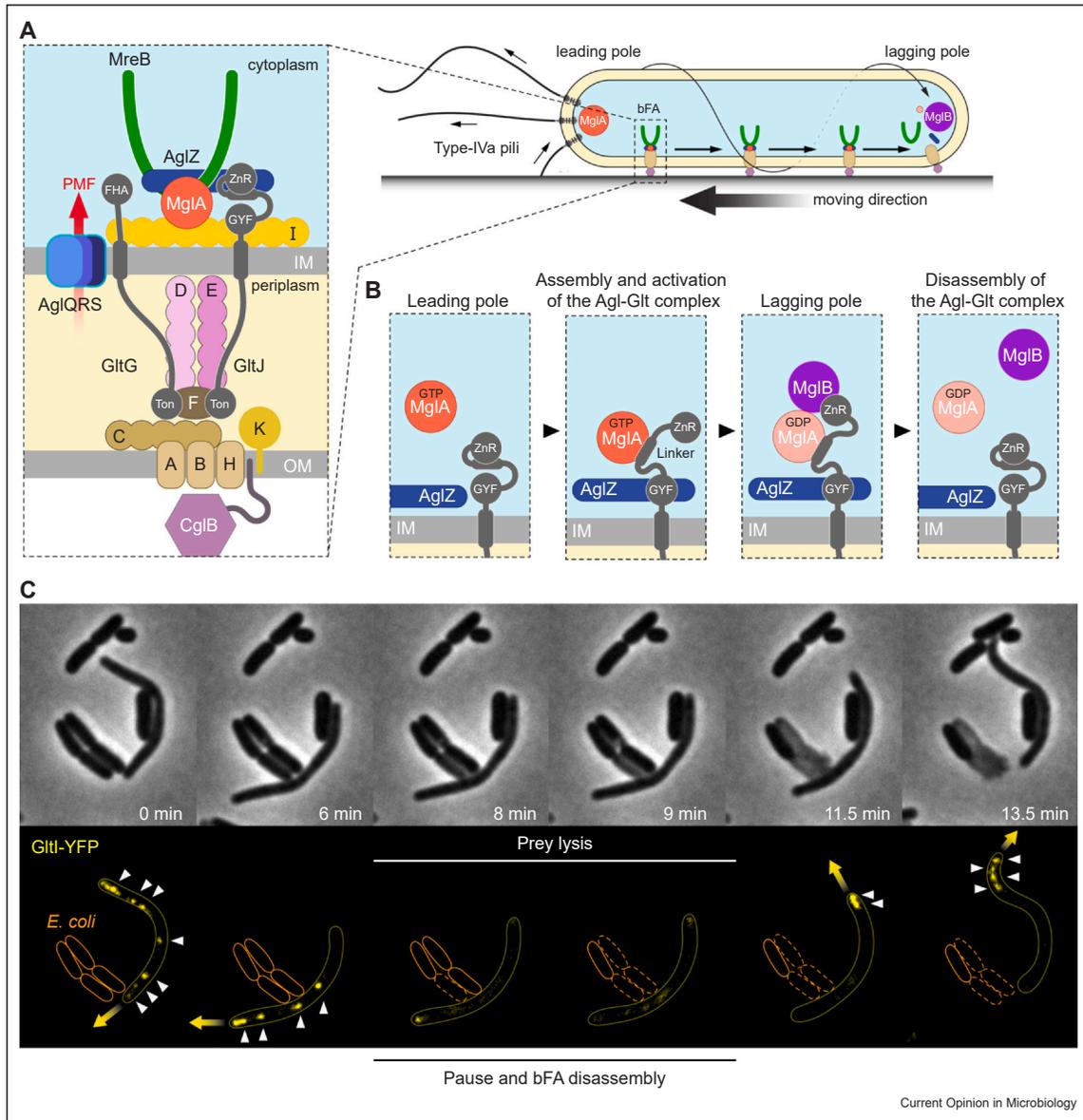
The inner membrane (IM) part of the motility complex contains the motility motor, a TolQR-like proton (H⁺)-gated channel that is required to move the motility complex in the cell envelope and propel the cell [31–34].

The IM complex is predicted to contain the proton channel components, AglQ, AglR, and AglS, as well as two integral IM proteins GltG and GltJ (Figure 1a). At the cell pole, GltJ connects the IM complex to a cytosolic platform composed of a suite of proteins, including GltI, AglZ, the GTPase MglA, and the MreB cytoskeleton (Figure 1a) [30,35–37]. Some of the molecular motifs required for this recruitment have been recently characterized and revealed that the N-terminal cytosolic domain of GltJ contains a three-protein binding region, defined by a Zinc Finger domain (ZnR) and a proline-rich binding domain (GYF), separated by a disordered Linker region (Figure 1b) [30,37]. At the cell leading pole, a proline-rich motif in AglZ interacts with the GYF domain, while MglA-GTP binds to the Linker motif (Figure 1b). Remarkably, these interactions switch the ZnR domain to an ‘ON’ conformation, which becomes important later on as the motility complex reaches the lagging cell pole (see below). Following this activation, the IM motor complex becomes active and traffics helically in the direction of the lagging cell pole, allowing the cell to move forward [37].

This trafficking requires an extensive periplasmic complex, which remains largely undefined. The IM motor is expected to interact with this complex via predicted GltG and GltJ α -helical extensions into the periplasmic space. Three periplasmic proteins GltD, GltE, and GltF are essential for motor movements (Figure 1a), presumably because they transduce its mechanical action to the external components of the complex in the outer membrane (OM; see below) [38,39]. For this reason, along with the periplasmic segments of GltG and GltJ, this supposed periplasmic complex has been coined the ‘transducer complex’, but currently, there is very little information on its structure and its protein interaction network.

Once the IM motor reaches the part of the cell envelope in contact with the underlying surface, it makes contact with the OM part of the complex, which creates local adhesions and leads to the formation of bFA complexes (Figure 1c) [39]. This OM part of the A-motility apparatus is formed by a complex of six proteins, coined the ‘OM platform’, which regulates the surface exposure of the major motility complex adhesin, a von Willebrand A domain-containing protein called CglB (Figure 1a) [40]. When the motility complex is active, CglB is exposed at the cell surface via its interaction with GltA, GltB, GltH, and GltK (Figure 1a) [40]. GltA, GltB, and GltH are porin-like proteins integrated into the OM [30]. GltA and GltB interact with each other and also with GltC, an OM-associated periplasmic protein featuring multiple TPR domains (Tetratricopeptide Repeat domain), which presumably promotes contact with the periplasmic transducer proteins [31,39]. The OM lipoprotein GltK facilitates the proper insertion of GltA and GltB

Figure 1



The motility strategies of *Myxococcus*. **(a) Right:** *Myxococcus* can activate two motility machineries at its leading pole. The S-motility machinery (twitching motility) allows the movement of groups of cells and involves retractile Type-IVa pili (T4aP) that pull cells forward like grappling hooks. The A-motility machinery (gliding motility) requires the Agl-Glt complex that assembles at the leading pole and transits directionally toward the lagging pole. Propulsion is produced when moving complexes adhere to the underlying surface via CglB. **Left:** cartoon representation of the predicted domain architecture of the Agl-Glt machinery. The AglQRS proton channel energizes the Glt complex by generating a proton motive force. The Glt complex interacts with the MreB cytoskeleton via a protein platform constituted by GltI, AglZ, MglA, and the cytoplasmic domains of GltG and GltJ. CglB tethers the Glt complex to the underlying surface. **(b)** Proposed activation/inactivation mechanism of bFAs. When the Agl-Glt complex is inactive and not interacting with AglZ and MglA, the switch is 'OFF', and the ZnR/Linker regions are in a closed conformation. At the leading pole, interactions of the GYF domain and the Linker motif with AglZ and MglA-GTP allow activation of the Agl-Glt complex. The ZnR domain switches to its 'ON' conformation. The complex can thus move toward the lagging cell pole, forming bFA complexes adhering to the underlying substratum. At the lagging pole, MglB is recruited by the ZnR domain and converts MglA to its GDP-bound state, which dissociates from the Linker region. This regulation leads to the release of the cytoplasmic platform, and the full process can start over. **(c)** Prey detection leads to bFA disassembly. In a *Myxococcus* strain expressing GltI fused to the Yellow Fluorescent Protein (YFP), the Agl-Glt complexes can be observed assembling on the substratum, forming bFAs (white arrowheads). Upon contact with prey (*Escherichia coli*), the bFAs are disassembled, and *Myxococcus* pauses to lyse its prey. After prey death, motility resumes by reassembling the bFAs on the underlying surface. Yellow arrows show the direction of movement. *Myxococcus* is outlined in yellow, and the prey is outlined in orange.

into the OM [31]. GltK is also important for the function of CglB, but it is currently unclear if these two proteins make a direct interaction [40]. Once it is adhered, the motility complex retains a fixed position relative to the substratum until it becomes disassembled at the lagging cell pole, which translates into the release of CglB adhesion [40]. The exact ligand of CglB remains unidentified.

The Agl-Glt complex is disassembled in the vicinity of the lagging cell pole. This regulation is driven by MglB, an MglA GTPase-activating protein that localizes at the lagging pole [41,42]. This mechanism is facilitated by the GltJ-ZnR domain, which binds directly to MglB and thus promotes its action on MglA, leading to the conversion of MglA-GTP to MglA-GDP. This leads to the detachment of MglA from the GltJ-Linker motif and the dispersal of the cytosolic platform (Figure 1b) [37]. This regulation ensures that the cell keeps moving forward as new active Agl-Glt complexes are formed at the leading cell pole. Disassembly depends solely upon the regulation of the nucleotide state of MglA. Remarkably, MglA mutants (MglA^{Q82A/L}) that are locked in the GTP-bound state, and insensitive to the action of MglB, are stuck in a perpetual pendulum-type movement [43]. In this context, motility complexes that are not inactivated at the lagging cell pole invert their direction of movement in the envelope until they reach the other end of the cell and reverse again, endlessly.

Unresolved aspects of the motility mechanism

While it is clear that intracellular trafficking of the Agl motor and its interaction with the OM proteins are the major mechanism of force transduction underlying propulsion [39], there are still major unresolved questions. In particular, the motor movements are highly directional as it moves along a shallow helical trajectory, which leads to cell rotation during movement [28,33,39,44]. This suggests the existence of an underlying track in the cell envelope, but the identity of such tracks remains elusive. The mechanism of force transduction itself also remains mysterious. The MreB cytoskeleton, a component of the Agl-Glt cytoplasmic platform, could provide a rigid scaffold allowing motor forces to be transduced to the external substratum [35,36,39]. Such an anchored IM motor could thus push on the OM complex by provoking conformational changes in the periplasmic transducer and driving cyclical interactions with the OM platform [39,45]. These interactions could thus direct CglB to active bFA complexes and couple traction forces and adhesion [40]. It is not currently known how the IM motor interacts dynamically with the OM platform through the peptidoglycan. But based on our knowledge of the Tol-Pal system, which shows many similarities with the Agl-Glt

system, it was proposed that helical periplasmic proteins such as GltG or GltJ could contract and extend through the peptidoglycan layer as it has been proposed for TolA when energized by TolQR [39,45]. A definitive elucidation of this mechanism awaits a structural characterization of the Agl motor and its associated periplasmic proteins.

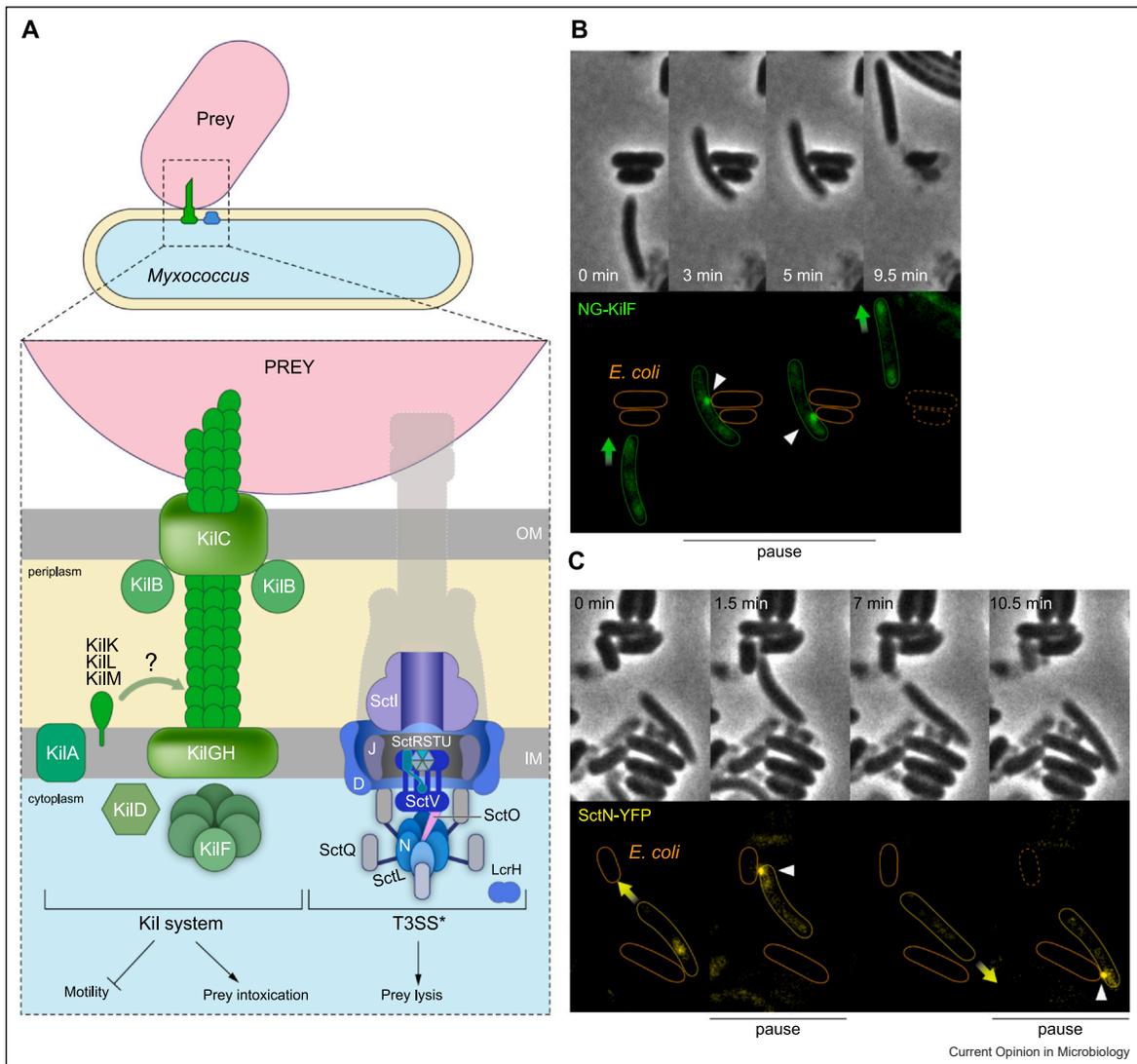
Killing with a sense of touch

Once in the prey colony, *Myxococcus* A-motile cells kill their preys by direct contact [14]. This process is remarkable because prey contact triggers two major changes in cell behavior and organization:

- (i) An immediate motility arrest, which is due to the rapid disassembly of the Agl-Glt complex once contact with the prey has been established (Figure 1c) [14,22].
- (ii) The assembly of a lethal machinery at the prey contact site, which can occur anywhere along the cell body and simultaneously at multiple positions when the *Myxococcus* cell establishes contact with several prey cells. The lethal complex was recently characterized genetically. It forms a fascinating composite apparatus, coupling predicted Tight adherence-like pilus proteins (Kil) and components of a needleless Type-3 Secretion System (T3SS*) [14,23].

The Kil system is encoded within two genetic clusters, comprising up to 21 predicted genes [14]. While many genes encode hypothetical proteins, the two *kil* clusters contain the full genetic complement of a putative member of the Tad (Tight Adherence) Type-IVc pilus family [46,47]. These specific genes are essential for prey-induced motility arrest and contact-dependent killing [14]. Briefly, the Kil system is predicted to adopt the classic architecture of a T4cP with some subtleties (Figure 2a) [47,48]. It comprises the secretin KilC, the IM platform proteins KilG/H, the pilus assembly protein KilB, and KilD, a cytoplasmic protein of unknown function. The ATPase KilF resembles the bifunctional ATPase CpaF found in the *Caulobacter* Tad pilus, which drives both the extension and retraction of the Tad pilus [14,49]. The predicted secretin (KilC) is remarkably short, missing an N0 domain typically found in secretin proteins. These features of KilC are however not unique to this protein and are comparable to that of other recently characterized Tad pilus secretins [14,50,51]. Although the formation of a Kil pilus has yet-to-be-demonstrated, the *kil* clusters encode two minor pilins (KilL and KilM) and one major pilin (KilK). A prepilin peptidase (KilA) is also present within the *kil* gene clusters; however its role in maturing the Kil pilins has not been demonstrated yet [14]. Intriguingly, these pilins are partially required for Kil function, suggesting that the assembly of a Kil pilus filament, if one is made at all, is not an absolute requirement for contact-dependent killing. Alternatively,

Figure 2



Myxococcus recruits two predatory machineries at the prey contact site. **(a)** On the left: the Tad-like Kil system is essential to pause motility upon contact with prey and to kill the prey by contact. The Kil system is constituted of a secretin (KilC) inserted in the OM allowing the extrusion of a pilin fiber. The polymerization and depolymerization of the pilus are energized by the ATPase KilF, which interacts with the IM platform constituted by KilG and KilH. The KilK, KilL, and KilM major and minor pilins are supposedly constituting the pilin fiber. A prepilin peptidase (KilA) is present within the *kil* gene clusters, but its role remains to be clarified. KilD is a cytoplasmic protein of unknown function associated with the Kil and essential for predation. On the right: the needleless T3SS* (T3SS*) is also recruited at the prey contact site and is essential to efficiently lyse the prey and release nutrients in the environment. The IM platform is constituted by SctD, SctI, and SctJ. The cytoplasmic protein SctQ forms the C-ring, which is connected via SctL to the ATPase SctN, energizing the system. SctO is a key link within the T3SS* central core, transmitting mechanical forces between the ATPase and the gate of the export apparatus constituted by SctRSTUV. The LcrH-like chaperones interact with the effectors. This complex binds to SctV and, supported by the action of the ATPase, the effector, and the chaperone is disassembled, and the effector unfolds and is inserted into the secretion channel formed by the export apparatus and the hollow needle. In the T3SS*, proteins constituting the needle or the translocon structure are absent (in gray). **(b)** In a *Myxococcus* strain expressing KilF (the ATPase) fused to the NeonGreen (NG), the Kil system recruitment can be observed at the prey contact site (white arrowheads). Green arrows show the direction of movement. *Myxococcus* is outlined in green, and the prey (*E. coli*) is outlined in orange. **(c)** Similarly, in a *Myxococcus* strain expressing the T3SS* ATPase SctN fused to YFP, the T3SS* recruitment can be observed at the prey contact site (white arrowheads). Yellow arrows show the direction of movement. *Myxococcus* is outlined in yellow, and the prey (*E. coli*) is outlined in orange.

other pilins might also be involved, as it is the case for *Pseudomonas aeruginosa* T4aP, which assembly can be primed by T2SS minor pilins [52].

As revealed by fluorescence microscopy, structural components of the Kil system, KilF and KilG, form dynamic fluorescent clusters at the prey contact site,

which correlates with motility pausing and is followed by prey killing (Figure 2b) [14,23]. This suggests that the Kil system carries a lethal activity at the prey contact site. Indeed, localized degradation of prey peptidoglycan rapidly follows Kil assembly [14]. However, it remains unclear whether the Kil system functions as a toxin-secretion device or recruits a yet-to-be-characterized toxin delivery system at the prey contact site.

The T3SS* plays a crucial role in facilitating the complete lysis of the prey, leading to the release of nutrients into the environment, thereby enabling *Myxococcus* to sustain its growth [23]. In absence of the T3SS*, *Myxococcus* still kills its prey by contact in Kil-dependent way, but the prey cell remains plasmolyzed, and it does not lyse [23]. Remarkably, a *Myxococcus* T3SS* mutant cannot grow on prey, demonstrating that prey nutrients only become available if prey cells lyse [23]. The T3SS* machinery is also encoded within a large genetic cluster containing many hypothetical proteins. These genes comprise a full genetic complement encoding a T3SS IM platform (constituted of SctDIJ), a C-ring (SctQ), an export apparatus (SctRSTUV), and an ATPase complex (SctN and SctL) interacting with the central stalk (SctO), as well as several LcrH chaperone-like proteins (Figure 2a) [18,23]. However, as mentioned above, the needleless T3SS* lacks key components of the injectisome necessary for the translocation of toxic molecules into the prey [9,23,53–57]. Thus, the OM secretin is missing, and homologs to a T3SS needle or translocon structure are absent [23]. This indicates that the function of the T3SS* may differ from the secretory function of typical T3SS. The T3SS* is conserved within the *Myxobacteria*, and from an evolutionary point of view, it might represent an intermediate between the flagellar T3SS and the injectisome [18,23,55,56,58]. Similar to the Kil system, the T3SS* accumulates at the prey contact site during predation (Figure 2c) [23]. Although the role of the T3SS* in prey lysis is clear, the associated toxic effectors are still unknown at this point. Interestingly, recruitment of the Kil and T3SS* machineries is only observed upon contact with a prey cell but not with other *Myxococcus* cells [14,23], suggesting that *Myxococcus* can distinguish self from nonself by a currently unknown mechanism.

Altogether, this indicates that each predatory system fulfills specific and essential functions during the predation process, allowing *Myxococcus* to pause its motility, kill and lyse the prey, subsequently used as a source of nutrients for growth and division.

Motility is regulated by the Kil system upon contact with prey

Coupling between motility and killing has been observed directly by microscopy. Typically, A-motile cells disassemble bFA complexes when they establish lethal contacts with prey cells (Figure 1c) [14]. This pause lasts

for the entire killing process, and bFA complexes are reassembled once prey cells have lysed (Figure 1c). How prey detection and lysis act as cues to regulate the disassembly and reassembly of the bFA complexes on the substratum is still an open question. The Kil system is essential for this regulation because in the absence of the KilC secretin, the prey is still perceived (as indicated by the assembly of KilD at the prey contact site), but motility is not paused, and the prey cell survives [14]. This suggests that prey perception and killing involve a complex sequence of events, that is, prey detection, pause, lysis, and motility resumption. In this pathway, the Kil system induces both motility arrest and prey lysis (in conjunction with the T3SS*). It is not known whether the T3SS* is also involved in motility regulation.

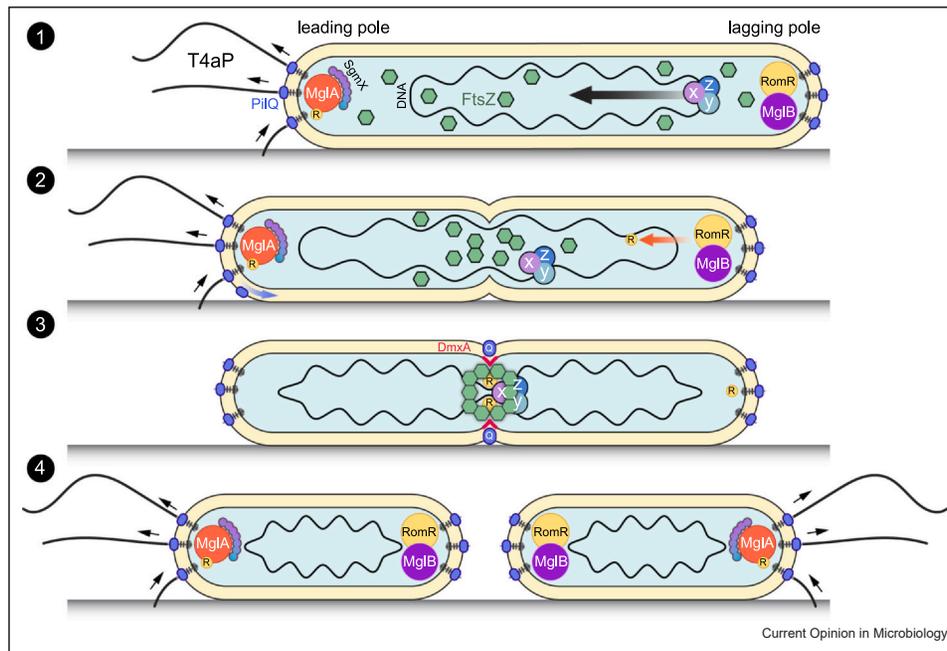
How the Kil system regulates the activity of the A-motility machinery when a prey is perceived remains mysterious. Within the *kil*, *t3ss**, or *glt* gene clusters, numerous genes encode proteins with unknown functions, some predicted to have Forkhead-associated (FHA) or Tetratricopeptide (TPR) domains (e.g. the ATPase KilF itself is fused to an N-terminal FHA domain and KilD contains multiple FHA and TPR domains) [14,23,30]. These domains are known for their role in protein–protein interactions and signal transduction [59,60] and might therefore be constituting a signaling pathway to regulate motility upon prey detection. Thus, understanding the mechanism behind prey-induced motility arrest likely lies in the characterization of these proteins.

Pausing motility to divide

Inside prey fields, feeding is revealed by continuous elongation of *Myxococcus* cells due to growth [14]. However, long predivisional cells systematically pause their movements ~20 min before division, even when they are caught in the middle of large groups of moving cells within the prey colony. Remarkably, dividing cells resume motility immediately once cytokinesis is complete. This suggests that the motility systems are probably tightly connected to cell cycle regulators or to some divisome proteins, through a yet-to-be-discovered mechanism. The reason for this coupling is not known. It is not a prey-dependent process, and it also occurs in axenic cultures [25]. Although there is no evidence for this, some components may be common for both processes, as in eukaryotic cells, where actin must be rearranged from focal adhesions to enter mitosis and assemble a cytokinetic ring [61]. It is also possible that motility complex disassembly is necessary to ensure that the daughter cells inherit properly assembled motility systems following cell division (see below).

In *Myxococcus*, our understanding of cell division is still rather incomplete. Like the vast majority of bacteria, cell division in *Myxococcus* cells is initiated by the polymerization of the

Figure 3



Reorganization of the motility proteins upon cell division in *Myxococcus*. **(1)** In a motile predivisional cell, the T4aPs are activated at the front pole by SgmX, while the motility-associated protein RomR marks the lagging pole. At this stage, FtsZ is soluble and diffuse in the cytoplasm. The DNA-bound PomX-PomY-PomZ complex is positioned to one end of the nucleoid. **(2)** While *Myxococcus* is elongating and still moving, the PomXYZ complex is seen navigating along the nucleoid to reach the center of the cell. **(3)** In the latest stage of cell division, about 20 min before septation, *Myxococcus* pauses and the PomXYZ complex triggers FtsZ-ring polymerization at the constriction site. RomR and the PilQ proteins are seen relocalizing at the site of constriction where they might help co-ordinating motility regulation and division. The membrane-bound protein DmxA was recently shown to localize at the septum for a short period before and after septation while it appears to be diffuse during the rest of the cell cycle. **(4)** The two daughter cells start moving in opposite directions immediately after cytokinesis. Their new lagging pole has been enriched in RomR while the septum was established. Both leading and lagging poles have assembled S-motility apparatus, ready to polymerize T4aPs.

tubulin-like protein FtsZ in the middle of predivisional cells (Figure 3) [62]. The Z-ring serves as a scaffold to recruit all the proteins involved in cell constriction, peptidoglycan remodeling, and synthesis where the poles of the future daughter cells are being formed (see Ref. [63] for details on the bacterial divisome). The genetic organization of divisome genes is rather well conserved in *Myxococcus*, suggesting that the steps following Z-ring assembly are probably conserved as well [62,64]. While most bacteria inhibit FtsZ polymerization anywhere in the cytoplasm but at midcell, thanks to the rather conserved Min and NOC systems [64,65], *Myxococcus* possesses its own system, namely, the PomXYZ complex, which is proposed to drive FtsZ polymerization at the center of predivisional cells (Figure 3) [62,64,66,67]. These three proteins are important for FtsZ-ring positioning at midcell as any of the single mutants of the *pom* genes tends to mislocate the Z-ring [62,66]. Recently, PomY was shown to form liquid-like protein condensates that can trigger FtsZ polymerization *in vitro* [68]. *In vivo*, this system is proposed to move along the nucleoid via PomZ, a ParA-like DNA-binding ATPase, and position itself to mid-nucleoid by biased random motion [66]. Since the Pom complex stalls at mid-nucleoid position,

which coincides with midcell, it likely stimulates FtsZ polymerization there, as it does *in vitro* [68].

Z-ring assembly precedes motility arrest. Therefore, divisome proteins or their activity might also regulate the motility machineries. While the direct links are not known, RomR, a crucial motility regulator, is recruited at midcell following FtsZ assembly in predivisional cells (Figure 3) [25]. In the cell, RomR is a critical component of a regulatory system that determines the spatial activity of the motility complex by recruiting MglA-GTP directly at the pole (via an intermediate partner, RomX [69]). The recruitment of RomR at the division site could serve as a first landmark to activate the motility complexes at the appropriate pole in daughter cells. At the pole, it is known that RomR drives the polar localization of MglA-GTP which downstream, activates the Agl-Glt complex as described above. Importantly, MglA-GTP also activates T4aPs at the leading pole by recruiting SgmX, an activator of the PilB motor (Figure 3) [70,71]. However, it is important to consider that in the case of T4aPs, polar localization is independent of the RomR-Mgl system. In this case, the T4aP machineries

are directed to the pole via a septal affinity of the PilQ secretin and more specifically its so-called Amidase N-terminal domain [72]. Thus, inheritance of functional motility complexes in daughter cells might require complex interactions between motility proteins and/or their regulators with the divisome proteins and the forming peptidoglycan present at the septum.

How is RomR recruited at the septum, and is it indeed the first motility component to relocate there? RomR might only be one component of the regulation network, which likely involves a suite of partners and signals. In support of this, it was recently reported that the divisome recruits a diguanylate cyclase, DmxA, which provokes a c-di-GMP burst at the onset of constriction (Figure 3) [73]. While the exact function of this signal is not clear, it is important for motility and more specifically for the proper inheritance of correct polarity of the motility proteins in daughter cells [73]. Interestingly, RomR recruitment at division septa appeared perturbed in a *dmxA* mutant, suggesting that c-di-GMP acts upstream in the regulation cascade.

Future perspectives

In bacterial predation, the efficacy of killing is generally increased by motility, favoring cell–cell contacts and therefore improving the efficiency of short-range weapons [74]. For example, the small predatory bacterium *Bdellovibrio bacteriovorus* swims toward its prey, attaching to its surface and invading its periplasm where it can grow and divide [75,76]. In this bacterium, flagellar motility is coupled to prey attachment by a T4P and recently discovered chimeric fiber proteins to penetrate the prey OM [77–79]. In *Aureispira*, predator-prey contacts on surfaces are established by gliding motility (mediated by T9SS/SprB), which then allows the deployment of a T6SS. In this case, T6SS-associated extracellular antennae may be involved in signal transduction and triggering of firing [80]. *Myxococcus* also encodes a T6SS, but it is not essential for predation and rather appears to be involved in discriminating nonkin and less fit siblings [14,18,81–84].

Thus, prey perception by motile predatory cells and the subsequent activation of a prey killing system is a general feature of predatory bacteria.

It is truly remarkable that *Myxococcus* can detect prey at any site along its cell body, which then triggers the killing response, a *bona fide* sense of touch. How are preys perceived? how does that lead to assembly of the Kil-T3SS* at the contact site? The regulation might reside in direct cross-talk between the machineries; the over-representation of FHA- and TPR-containing proteins in these systems suggests signal transduction functions and could be linked to these cross-talks.

Motility regulation is also central to cell division. This is not a hallmark of bacterial predators and other bacteria must couple motility and cell division, again to ensure that the motility apparatus (i.e. flagella) are correctly inherited by daughter cells. For example, in *Caulobacter*, a regulator MadA ensures just-in-time expression of cell division components when flagellar assembly is completed [85]. Similar to the motility arrest observed during predation, it is not clear which cue stops motility machines and whether this is due to their disassembly or changes in their activity. It is clear that this checkpoint occurs downstream from Z-ring assembly, and therefore, this regulation must be linked to a late event of cell division.

Is this regulation occurring upstream from both Agl-Glt and the T4aP? Or are there machinery-specific regulations? In principle, the displacement of MglA-GTP from the cell pole should be sufficient to halt motility, and perhaps, this is simply related to the relocalization of RomR to the septum, a tempting hypothesis. But, as discussed above, there is evidence for additional complexity. Again, a complex cross-talk must occur between the division and the motility machineries.

In conclusion, the motile predatory cells of *Myxococcus* exhibit remarkable collective behaviors. Researchers have identified and partially characterized the individual mechanisms that enable these cells to move, kill, and divide. However, a significant challenge remains in understanding how these systems co-ordinate and lead to timely and precise cellular decisions. This perspective is broadening to include other bacteria, extending beyond predatory interactions. It is becoming increasingly apparent that integrating cellular processes is crucial for fast adaptation to rapidly changing environments.

Funding

This work was supported by the European Union (European Research Council (ERC), grant JAWS 885145), the Centre National de la Recherche Scientifique (CNRS, France), and Aix-Marseille University (France).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

No data were used for the research described in the article.

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