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CELL SCIENCE AT A GLANCE

Bacterial cell division at a glance

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ABSTRACT

Bacterial cell division is initiated by the midcell assembly of polymers of the tubulin-like GTPase FtsZ. The FtsZ ring (Z-ring) is a discontinuous structure made of dynamic patches of FtsZ that undergo treadmilling motion. Roughly a dozen additional essential proteins are recruited to the division site by the dynamic Z-ring scaffold and subsequently activate cell wall synthesis to drive cell envelope constriction during division. In this Cell Science at a Glance article and the accompanying poster, we summarize our understanding of the assembly and activation of the bacterial cell division machinery. We introduce polymerization properties of FtsZ and discuss our current knowledge of divisome assembly and activation. We further highlight the intimate relationship between the structure and dynamics of FtsZ and the movement and activity of cell wall synthases at the division site, before concluding with a perspective on the most important open questions on bacterial cell division.

KEY WORDS: FtsZ, Bacteria, Bacterial cell biology, Cell division, Cytokinesis, Peptidoglycan

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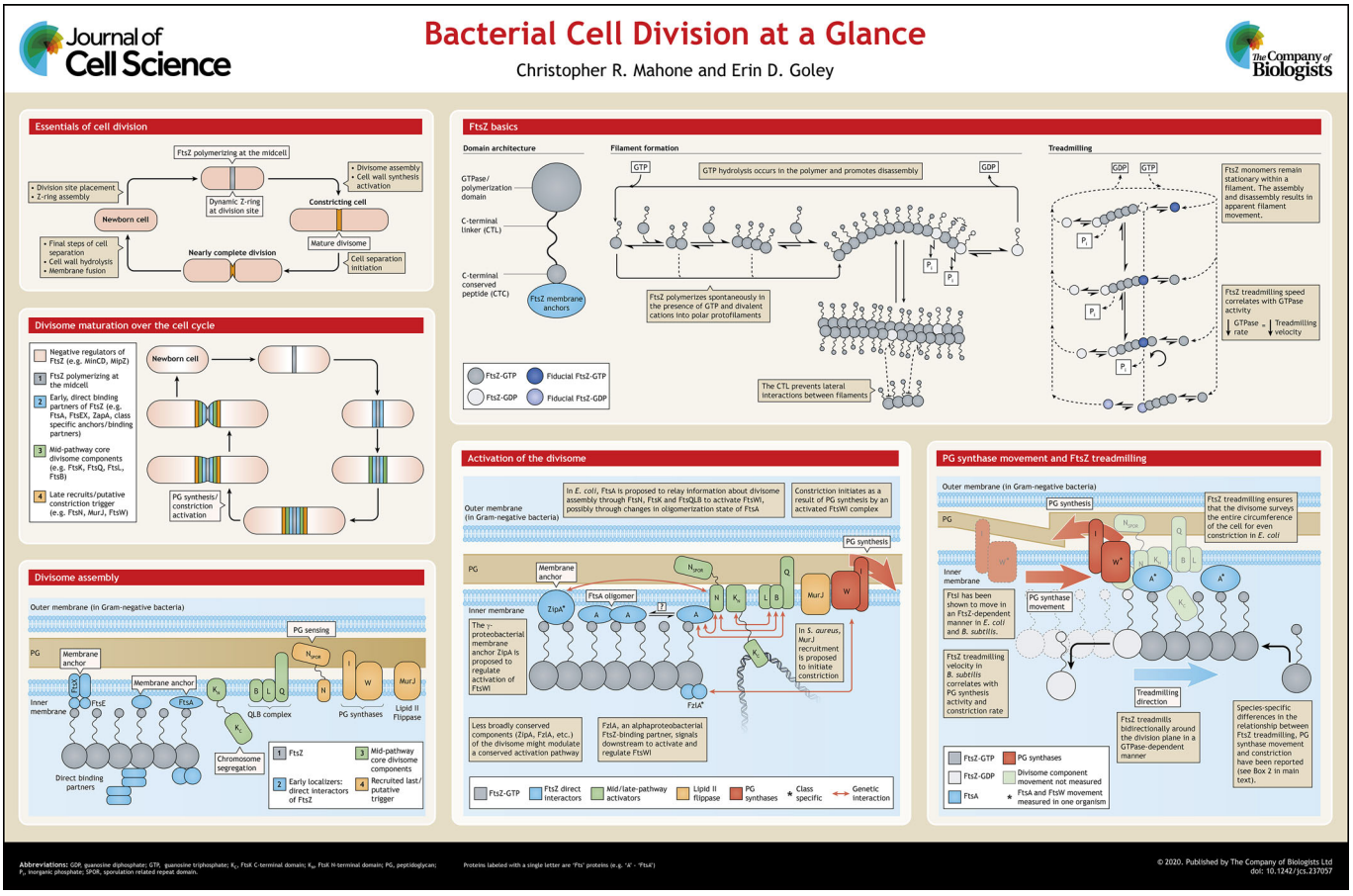
Introduction

When observed by time-lapse microscopy, bacterial cell division looks very simple. Start with one bacterium; end up with two. As with most biological processes, however, things are more complicated under the surface. To initiate division, bacteria need to accomplish at least three tasks: mark the division site, recruit the division machinery (the divisome), and activate cell wall synthesis to drive constriction (see poster). Subsequent events, including membrane fusion and cell wall hydrolysis, are required for compartmentalization and physical separation of the cell into two daughters. This Cell Science at a Glance article and poster will focus on the initial stages of divisome assembly and constriction activation. To mark the division plane, almost all bacteria use a tubulin-like GTPase called FtsZ that polymerizes along the circumference of the cytoplasmic membrane at the future division site (Löwe and Amos, 1998; Bi and Lutkenhaus, 1991). Use of



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fluorescently labeled FtsZ has shown that FtsZ forms a ring-like structure called the Z-ring (Ma et al., 1996) that directly and indirectly recruits other division proteins to the midcell region (Adams and Errington, 2009). The Z-ring is a moving target, however, as recent studies have demonstrated that patches of FtsZ filaments treadmill at the division site and direct the dynamic movement of other division proteins (Bisson-Filho et al., 2017; Monteiro et al., 2018; Perez et al., 2019; Yang et al., 2017).

Although division begins with FtsZ, and there is *in vitro* evidence that FtsZ can deform membranes, the force generated by FtsZ alone is insufficient to initiate constriction. Instead, the driving force for constriction is thought to be cell wall synthesis (Coltharp et al., 2016; Daley et al., 2016). The peptidoglycan (PG) cell wall in bacteria comprises a meshwork of glycan strands crosslinked together by short peptides, and provides shape and protection against turgor pressure (Typas et al., 2011). The enzymes that polymerize the glycan strands are called glycosyltransferases (GTases), and the enzymes that crosslink the peptide side chains are called transpeptidases (TPases). During division, FtsW and a monofunctional penicillin-binding protein (FtsI) are the primary GTase and TPase, respectively (Adam et al., 1997; Ikeda et al., 1989; Spratt, 1975; Taguchi et al., 2019). However, localization of these enzymes to the division plane is not sufficient for PG synthesis and constriction. Instead, these enzymes require activating signals to trigger constriction (Lariviere et al., 2019; Rohs et al., 2018; Taguchi et al., 2019; Tsang and Bernhardt, 2015).

This Cell Science at a Glance article will synthesize our current understanding of the assembly, activation and dynamics of the bacterial cytokinetic machinery for the initial stages of cell division. We will pay particular attention to recent advances in understanding the links between the FtsZ cytoskeleton, and the activity and dynamics of the PG synthases that drive constriction.

FtsZ – the master regulator of bacterial division

FtsZ has three conserved domains: a polymerizing GTPase domain, a C-terminal conserved (CTC) peptide through which membrane anchors bind, and a disordered C-terminal linker (CTL) that connects the GTPase domain to the CTC (Vaughan et al., 2004) (see poster and Box 1). In addition, some organisms have a short extension at the extreme C-terminus of FtsZ called the C-terminal variable (CTV) region (Buske and Levin, 2012).

The GTPase domain of FtsZ is required for polymerization and, like tubulin, the nucleotide-bound state influences the polymerization dynamics and structure of FtsZ filaments (Bramhill and Thompson, 1994; Erickson et al., 1996; Mukherjee and Lutkenhaus, 1994). In the presence of GTP and divalent cations, FtsZ spontaneously assembles into polymers *in vitro* (Bramhill and Thompson, 1994; Erickson et al., 1996; Mukherjee and Lutkenhaus, 1994). Once in a filament, FtsZ is competent to hydrolyze GTP, and nucleotide hydrolysis serves to favor depolymerization and to take the FtsZ filament from a more straight to a more curved conformation (Erickson et al., 1996) (see poster). Mutations or chemical perturbations that slow the GTP hydrolysis rate of FtsZ stabilize the polymer *in vitro* and in cells; as a consequence, the completion of constriction is slowed or prevented (Bisson-Filho et al., 2017; Monteiro et al., 2018; Perez et al., 2019; Stricker et al., 2002; Yang et al., 2017). FtsZ assembly *in vitro* occurs when it is above a critical concentration of ∼1 µM, and assembly is cooperative (Mukherjee and Lutkenhaus, 1998, 1999; Romberg et al., 2001). Recent structural work has demonstrated that the FtsZ monomer undergoes a conformational change from closed to open upon polymerization, which provides an explanation for the cooperative nature of FtsZ polymerization and the ability of filaments to treadmill (Wagstaff et al., 2017) (see poster).

In vitro under physiological pH and salt conditions, FtsZ forms mostly single protofilaments that are ∼120–200 nm long (Erickson et al., 2010; Romberg et al., 2001). Multiple FtsZ filaments can associate laterally into thicker bundles *in vitro* depending on the presence of crowding agents or binding partners, salt concentration and pH (Erickson et al., 1996; Huang et al., 2013). However, the structure(s) of FtsZ filaments in cells is not fully resolved. When imaged by electron cryotomography *in vivo*, FtsZ filaments appear to be of a width that reflects that of a single monomer, are ∼100 nm long and are at a stereotypical distance of ∼16 nm from the membrane (Li et al., 2007; Szwedziak et al., 2014; Yao et al., 2017). Super-resolution light microscopy of labeled Z-rings highlights loose filament clusters distributed around the division plane in a discontinuous structure that extends ∼50–100 nm radially into the cell (Fu et al., 2010; Holden et al., 2014; Strauss et al., 2012). Although bundling of FtsZ is reported under a variety of conditions *in vitro*, thick bundles of FtsZ have not been observed in wild-type bacteria, and induction of stable, large-scale bundling *in vivo* can lead to detrimental phenotypic changes (Buske and Levin, 2013; Durand-Heredia et al., 2012; Sundararajan et al., 2015; Barrows et al., 2020). However, mutation of residues that are implicated in lateral interactions between FtsZ filaments disrupts division, suggesting that transient lateral interactions are important to division progression (Guan et al., 2018). The CTL that connects the GTPase and CTC domain appears to have a major role in modulating lateral interactions (see Box 1).

In addition to its intrinsic polymerization properties, FtsZ has a host of interacting proteins that can modulate its assembly (Huang et al., 2013). Importantly, these include inhibitors of FtsZ that spatially and/or temporally regulate Z-ring assembly and proteins

Box 1. Contribution of the C-terminal linker of FtsZ to polymer structure, dynamics and function

Although the GTPase domain is the primary determinant of FtsZ assembly, the C-terminus of FtsZ has recently been demonstrated to impact FtsZ function and polymerization. The CTL is a disordered region that connects the GTPase domain and the CTC (see poster) and is highly variable in sequence and length across species (Vaughan et al., 2004). *E. coli* and *B. subtilis* are each tolerant of changes in the CTL sequence, but it must be disordered and close in length to the native CTL to function in division, implying a role as a flexible link to the membrane (Buske and Levin, 2013; Gardner et al., 2013). *C. crescentus* cells that produce FtsZ lacking its CTL (ΔCTL) exhibit dominant lethal cell bulging and lysis (Sundararajan et al., 2015), and *B. subtilis* cells producing ΔCTL rapidly lyse (Buske and Levin, 2013), suggesting downstream effects on PG metabolism. FtsZ variants bearing alterations to or deletion of the CTL have altered polymerization properties *in vitro* and altered filament superstructure in cells. *C. crescentus* ΔCTL forms hyperstable filament bundles *in vitro* and large non-ring assemblies in cells, implicating the CTL in regulating lateral interactions and polymer stability (Sundararajan and Goley, 2017; Sundararajan et al., 2015, 2018; Barrows et al., 2020). *B. subtilis* ΔCTL forms large extended bundles in cells, and *in vitro* the CTL impacts *B. subtilis* FtsZ interfilament spacing, suggesting a conserved role for the CTL in impacting FtsZ polymer superstructure (Buske and Levin, 2013; Huecas et al., 2017). The phenotypic outcomes of deleting or altering the CTL implicate this domain in regulation of PG metabolism (Buske and Levin, 2013; Gardner et al., 2013; Sundararajan et al., 2015). In addition to the CTL, the CTV of *B. subtilis* FtsZ is sufficient to induce lateral interactions *in vitro* that are important for formation of the Z-ring and, therefore, division in that organism (Buske and Levin, 2012).

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that contribute to formation of a focused Z-ring at the midcell region (see poster). In most well-studied organisms, spatial regulation of Z-ring assembly is primarily mediated by negative regulators of FtsZ polymerization. The Min proteins function in *Escherichia coli* (de Boer et al., 1989) and *Bacillus subtilis* (Levin et al., 1992) to inhibit FtsZ polymerization near the cell poles, and a functionally analogous protein called MipZ fulfills a similar function in α-proteobacteria (Thanbichler and Shapiro, 2006; Toro-Nahuelpan et al., 2019), with the ultimate result being the accurate placement of the Z-ring at the midcell. Additional negative (e.g. nucleoid occlusion factors; Bernhardt and De Boer, 2005; Wu and Errington, 2004) or positive (e.g. Zaps; Durand-Heredia et al., 2011, 2012; Gueiros-Filho and Losick, 2002; Marteyn et al., 2014) regulators of FtsZ polymerization and organization serve with Min proteins or MipZ to coordinate Z-ring assembly in time and space (see poster). As FtsZ is studied in additional organisms, novel modes of regulation are being recognized – for example, the primary role of positive regulation of Z-ring placement by PomX, PomY and PomZ in *Myxococcus xanthus* (Schumacher et al., 2017) or by MapZ in *Streptococcus pneumoniae* (Fleurie et al., 2014; Massidda et al., 2014). Collectively, intrinsic and extrinsic factors promote the nucleotide-dependent assembly of FtsZ into a dynamic Z-ring to establish the future division site.

Assembly and activation of the divisome

Although central to the process, FtsZ is not the only protein required for cell division; there are roughly a dozen conserved proteins that are required at the midcell for constriction (see poster). Once the Z-ring assembles, the remainder of the divisome is recruited in a roughly sequential manner. In *E. coli* and *B. subtilis*, the process occurs in a two-step fashion, with direct FtsZ interactors localizing first and other division components localizing in a second step (Aarsman et al., 2005; Gamba et al., 2009). In *Caulobacter crescentus*, cell synchronization experiments enabled the classification of divisome assembly into a series of seven functional modules (Goley et al., 2011). Across bacteria, the earliest arrivals to the division plane help to assemble a focused midcell Z-ring (Aarsman et al., 2005; Fleurie et al., 2015; Gamba et al., 2009; Goley et al., 2011; Schumacher et al., 2017). A subsequent wave (or waves) of protein recruitment brings factors in as their functions are required. Divisome components that localize just prior to initiation of constriction (FtsN in *E. coli*, FtsW in *C. crescentus* and MurJ in *Staphylococcus aureus*) have been proposed to trigger constriction through activation of cytokinetic cell wall synthesis (Aarsman et al., 2005; Goley et al., 2011; Monteiro et al., 2018) (see poster).

Simply localizing PG synthases to the midcell is not sufficient for constriction to begin, which implies a requirement for regulatory input into constriction activation. This makes sense, as improper timing of constriction could have drastic consequences for the cell. The GTPase FtsW requires its partner TPase FtsI to act as a PG polymerase *in vitro*, suggesting that it is inactive until engaged in a complex (Taguchi et al., 2019). Moreover, the fully assembled divisome in *C. crescentus* can be held in an inactive state by SidA or DidA, small protein inhibitors of constriction that bind the late divisome proteins FtsW and/or FtsN upon DNA damage (Modell et al., 2011, 2014). Mutations in FtsW or FtsI that bypass inhibition by SidA and DidA hyperactivate these PG synthases such that the cells constrict faster than wild type (Lambert et al., 2018; Lariviere et al., 2019; Modell et al., 2014). Similarly, hyperactivating mutations in the divisome proteins FtsL and FtsB were described in *E. coli* to cause premature initiation of constriction and/or cell shortening (Liu et al., 2015; Tsang and Bernhardt, 2015). Collectively, these observations imply the presence of inactive and active states of the PG synthases that promote constriction.

The most advanced – but still incomplete – model for constriction activation is derived from genetic studies in *E. coli* and includes a number of broadly conserved divisome components. In this model, the activating signal is proposed to initiate with FtsA, an actin homolog and conserved membrane anchor for FtsZ. Specifically, FtsA is thought to relay information about divisome assembly status by converting from an 'off' to an 'on' state through a mechanism that may involve FtsA transitioning from polymeric to monomeric state (Pichoff et al., 2012, 2015) (see poster). Although FtsA has been demonstrated to polymerize *in vitro* (Krupka et al., 2017; Szwedziak et al., 2012), its physiological polymerization state is not clear. Regardless of the mechanism, FtsA is proposed to be in a constriction activation pathway that includes FtsN and the complex formed by FtsQ, FtsL and FtsB (FtsQLB) (Liu et al., 2015; Pichoff et al., 2018). Variants of FtsA and FtsN identified in *E. coli* are also able to bypass loss of an essential, but γ-proteobacteria-specific, membrane anchor for FtsZ called ZipA that is thought to modulate interactions between FtsA, FtsZ and downstream signaling proteins like FtsN (Geissler et al., 2003; Pichoff et al., 2012, 2015; Schoenemann et al., 2018) (see poster). FtsN, which contains a sporulation-related repeat (SPOR) domain responsible for recognizing denuded glycans (the result of amidase activity) (Yahashiri et al., 2015), could direct the FtsW–FtsI PG synthetic complex (FtsWI) to locations where new PG material should be incorporated. FtsN has also been proposed through genetic studies to relay the polymerization status of FtsA downstream to FtsQLB (Liu et al., 2015; Pichoff et al., 2012; Tsang and Bernhardt, 2015). FtsQLB is a multimeric complex that is genetically implicated in activating FtsWI (Liu et al., 2015; Tsang and Bernhardt, 2015), though the details of its role in activation of FtsWI are unknown. Finally, FtsK, a bifunctional protein involved in division and chromosome segregation, is also genetically implicated in activation of constriction through its N-terminal domain (Dubarry et al., 2010). Such a role for FtsK could link chromosome segregation to PG synthase activation to ensure DNA is not trapped as the cell envelope constricts.

Although many of the proteins implicated in constriction activation in *E. coli* are broadly conserved, the divisome has also diversified across bacteria. This includes the incorporation of less broadly conserved participants in constriction activation that are, nevertheless, essential in their cognate organisms, such as ZipA in *E. coli*. In *C. crescentus*, recent work identified an activating signal that originates from a Z-ring-associated protein that is found only in α-proteobacteria. In that organism, an essential, direct binding partner of FtsZ called FzlA participates in activation of the downstream PG synthases (Goley et al., 2010; Lariviere et al., 2018, 2019) (see poster). Although *fzlA* is normally essential for division, hyper-activating mutations in *ftsW* and/or *ftsI* allow deletion of *fzlA*, indicating that the essential function of FzlA is to activate FtsWI (Lambert et al., 2018; Lariviere et al., 2019). Many of the proteins described in the *E. coli* activation pathway are present in *C. crescentus*; it is therefore likely that FzlA ultimately signals through FtsA, FtsK, FtsN and/or FtsQLB. We suspect that other bacteria similarly modulate a conserved core constriction activation pathway to suit their needs. Consistent with this prediction, in *Staphylococcus aureus* the MurJ lipid II flippase is proposed to trigger constriction, and its recruitment to the division site relies on the DivIB–DivIC–FtsL complex (the FtsQLB homologs in this organism) (Monteiro et al., 2018). In summary, the initiation of constriction during bacterial division requires assembly of the polymeric FtsZ ring, sequential recruitment of other divisome

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proteins and a poorly understood constriction activation step that promotes PG synthesis to drive constriction.

The dance of the divisome – dynamics of FtsZ and PG synthases

Cell division is a dynamic process, with the shape of the cell changing dramatically as constriction progresses. Perhaps unsurprisingly, the components of the divisome are also highly dynamic both before and during constriction. Early observations using fluorescence recovery after photobleaching indicated that FtsZ turns over rapidly within the Z-ring, with half-times of recovery for fluorescently tagged FtsZ on the order of tens of seconds (Anderson et al., 2004; Stricker et al., 2002). Subsequently, work in several bacterial systems including *E. coli* (Yang et al., 2017), *B. subtilis* (Bisson-Filho et al., 2017), *S. aureus* (Monteiro et al., 2018) and *S. pneumoniae* (Perez et al., 2019) demonstrated that FtsZ treadmills circumferentially around the division plane. That is, FtsZ patches appear to move directionally around the circumference of the cell, but individual monomers are stationary for their lifetime within the patch (Bisson-Filho et al., 2017; Perez et al., 2019; Yang et al., 2017). The apparent movement of FtsZ is mediated by net addition of FtsZ to one end of each patch and net loss from the other (see poster). The velocity of FtsZ treadmilling, at ∼30 nm/s on average, is remarkably consistent across species and is dependent on the GTPase activity of FtsZ (Bisson-Filho et al., 2017; Perez et al., 2019; Yang et al., 2017). Diminished FtsZ GTPase activity correlates with slower treadmilling speeds (Bisson-Filho et al., 2017; Perez et al., 2019; Yang et al., 2017). Conversely, FtsZ treadmilling is independent of PG synthesis; treatment of cells with PG synthesis inhibitors had no effect on treadmilling velocity (Bisson-Filho et al., 2017; Perez et al., 2019; Yang et al., 2017). At least in *B. subtilis*, the membrane anchor FtsA also moves along with FtsZ filaments (Bisson-Filho et al., 2017).

What is the purpose of FtsZ treadmilling? Two possibilities have gained recent experimental support. The first is that FtsZ treadmilling helps to organize divisome complexes, including the PG synthases, around the division plane for evenly distributed PG synthesis and constriction. Tracking of single molecules of PG synthases in the divisome in diverse species revealed that they move directionally around the division plane (Bisson-Filho et al., 2017; Yang et al., 2017) (see poster). In *E. coli* and *B. subtilis*, movement of the PG synthases is dependent upon FtsZ treadmilling, with the rates of movement correlating with treadmilling speed of FtsZ (Bisson-Filho et al., 2017; Yang et al., 2017). In *E. coli*, changing the treadmilling velocity of FtsZ did not change the rate of constriction, but changed the spatial distribution of PG synthesis such that septa were distorted when treadmilling was slowed (Yang et al., 2017). Collectively, these observations indicate that treadmilling FtsZ is important for the spatial distribution of PG synthesis during constriction. A second possibility, with support in *B. subtilis*, is that FtsZ treadmilling provides input into the activity of PG synthases. In that organism, changing treadmilling velocity causes a change in the rate of PG metabolism and subsequent constriction (Bisson-Filho et al., 2017). Genetic evidence implicating FtsA and FtsZ in regulation of PG metabolic activity is also consistent with the idea that FtsZ actively modulates PG metabolism, in addition to acting as a dynamic scaffold (Buske and Levin, 2013; Gardner et al., 2013; Mura et al., 2017; Sundararajan et al., 2015; Barrows et al., 2020; Varma and Young, 2004). Whether the differences in the relationship between FtsZ treadmilling and PG synthase activity reported in different bacterial species are due to technical or biological differences remains to be resolved (see Box 2). Nevertheless, it is clear that FtsZ polymer dynamics are linked to divisome dynamics and/or function across diverse bacterial species.

Conclusions and perspectives

Bacterial cell division requires regulated polymerization of FtsZ into a dynamic cytokinetic ring that recruits roughly a dozen other division proteins, ultimately leading to activation of cell wall synthesis for constriction. The Z ring and other components of the divisome are highly dynamic, and these dynamics are apparently critical for efficient and accurate division. These recent exciting advances in understanding bacterial cell division have spurned new or renewed interest in old questions. With respect to activation of PG synthesis for constriction, it is not clear what is being sensed to license constriction initiation. Is it clearance of the chromosomal termini from the division plane, accumulation of a limiting component of the divisome or the substrate for PG synthesis, or changes in the assembly properties of FtsZ or FtsA? Once sensed, how are signals relayed between components of the division machinery to cause activation of FtsW and FtsI? Who are the players and how do they communicate with each other?

We are only at the beginning of understanding the relationship between FtsZ and both the movement and activity of the rest of the divisome. In *E. coli* (Yang et al., 2017) and *B. subtilis* (Bisson-Filho

Box 2. FtsZ treadmilling and PG synthase movement across species

Although FtsZ treadmilling and PG synthase movement or activity have been observed in four bacterial species to date, each species is reported to exhibit distinct properties from the others with respect to relationship between FtsZ treadmilling and movement or activity of PG synthases. In both *E. coli* and *B. subtilis*, FtsZ treadmilling velocity directly correlates with the rate of movement of the TPase FtsI (Bisson-Filho et al., 2017; Yang et al., 2017). However, there are key differences in the link between FtsZ dynamics and PG synthase activity. When FtsZ treadmilling is reduced in *E. coli*, the rate of PG incorporation is unchanged but the slowed movement of FtsI results in distorted, asymmetric septa (Yang et al., 2017). In contrast, in *B. subtilis*, PG synthase movement and activity correlates with FtsZ treadmilling – whether treadmilling velocity is decreased through mutation of FtsZ or increased by expression of an FtsZ inhibitor (Bisson-Filho et al., 2017). As FtsZ treadmilling velocity changes, the rates of PG incorporation and subsequent cell constriction change correspondingly.

S. aureus does not appear to fully follow the examples set above. FtsZ treadmilling and PG synthase activity appear to be correlated until the initiation of constriction. Specifically, pharmacological inhibition of FtsZ treadmilling prevents division if applied in the early stages of the cell cycle. However, if the cell has accumulated MurJ – the lipid II flippase and proposed division trigger in *S. aureus* – at the midcell prior to application of the inhibitor of FtsZ treadmilling, then the cell can carry out constriction independently of FtsZ movement (Monteiro et al., 2018). *S. pneumoniae* presents the most divergent picture of the link between FtsZ dynamics and PG synthases to date, in that the two processes appear to be independent of each other in that organism. PG synthases move at a slower speed than FtsZ and their speed is dependent on lipid II PG precursor availability rather than FtsZ treadmilling speed (Perez et al., 2019). Consistent with the FtsZ-independence of PG synthase movement, the PG synthesis rate during division in *S. pneumoniae* is also independent of FtsZ treadmilling rate, similar to what is seen in *E. coli* (Perez et al., 2019; Yang et al., 2017). Given the early stages in our understanding of divisome dynamics in any system, we cannot yet definitely say whether each of the species-specific differences reported are the result of biological diversification or arise from technical differences between studies.

et al., 2017), the PG synthases rely on FtsZ treadmilling for their movement (see poster and Box 2). However, FtsZ and PG synthases have not been shown to directly interact, implying that PG synthase dynamics rely on additional molecules in the pathway. What are the protein–protein and protein–envelope interactions that control the dynamics of PG synthases and other divisome proteins? Does the divisome move as a whole or are there subcomplexes independently moving within it? Are processively moving PG synthase molecules actively synthesizing PG or are they being passively distributed? Is there more than one motile population representing different activation states of the divisome?

Although we are likely years away from a complete reconstitution of the divisome and its activity *in vitro*, there are short-term advancements that will shed light on the mechanisms of division. Continued technical advances in imaging, such as techniques allowing imaging of the divisome in cross-section or monitoring dynamics of multiple divisome components in the same cells by single-molecule tracking, will allow us to define division dynamics with greater precision and higher throughput. PG synthesis assays *in vitro* with reconstituted division components will demonstrate which divisome components activate or inhibit PG synthesis, and the order in which they provide their signals. Finally, continuing to study the mechanisms of cell division across diverse bacterial species will distinguish the conserved core mechanisms of division and identify the myriad ways bacteria adapt these mechanisms to replicate with distinct morphologies and in diverse niches.

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Competing interests
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