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REVIEW ARTICLE

FtsZ: The Force Awakens

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Abstract | Binary fission of prokaryotic cells depends on a protein called FtsZ that self-assembles into a membrane-associated ring structure (FtsZ-ring) in the early stages of the cell division process. FtsZ is a tubulin homologue, which interacts with many additional proteins contributing to its function forming a ring at the mid-cell, essential for bacterial cell division. Whether the Z-ring is a force-generating machinery or a simple scaffold for organizing all other molecular players is poorly understood. Here, we review briefly the structure, dynamics, and interactions of FtsZ, the Z-ring and its associated proteins and weigh the evidence for and against force production by FtsZ.

1 Introduction

Division of bacterial cells is carried out by a large complex of proteins that assemble in the division plane in the form of a ring[1]. During division, constriction of the septum known as the 'Z-ring' or 'divisome' occurs resulting in two daughter cells. The key cytoskeletal component of divisome, FtsZ is a structural homologue of eukaryotic tubulin which polymerizes and assembles, forming a major component of the Z-ring[2,3]. Here, we review the structural, dynamical and macro-assembly features of the FtsZ, in the context of its role as a force generator.

1.1 Structure and Assembly of FtsZ Protofilaments

The major domains revealed by phylogenetic analysis and crystallographic studies of FtsZ from the thermophilic bacterium *Thermotoga maritima* suggest a modular structure, with striking similarity to tubulin monomer[4]. The 5 main domains of FtsZ monomer include (1) a short, unstructured and poorly conserved N-terminal peptide (NTP), (2) a globular, highly conserved core region including major parts of the N- and C-terminal domains with the GTP-binding pocket, (3) a highly conserved C-terminal linker (CTL) 4) a short, conserved C-terminal tail (CTT) 5) a C-terminal variable region (CTV). The extreme CTT region consists of about 11 amino acid residues (known as C-terminus constant region, CTC) which together with the CTV region is designated the 'grappling hook peptide' (GHP) that acts as a central hub for FtsZ-interacting proteins[5]. A flexible C-terminal linker is required for proper FtsZ assembly in vitro and cytokinetic ring formation in vivo[6].

The N-terminal region consists of approximately 320 residues and is highly conserved throughout all FtsZ. The N-terminus and the core region are essential for GTP binding and hydrolysis as well as for longitudinal interactions between subunits. Yeast two-hybrid and deletion studies have shown that N-terminal is essential for ring assembly. Several studies have shown that the core region is enough for filament formation and helps in protofilament bundling. Ftsz polymerizes in a GTP-dependent manner akin to that of tubulin. The single-subunit thick protofilament is formed by interaction of C-terminal domain of one subunit with GTP-binding N-terminal domain of another, producing a completely one-dimensional polymer. FtsZ protofilaments fit very well when modelled on the tubulin protofilament structures[7]. The FtsZ protofilaments exhibit bundling, resulting in a dynamic filament structure

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that is stable, with subunit exchange occurring throughout the length of the filament.

A flexible C-terminal linker is required for proper FtsZ assembly in vitro and cytokinetic ring formation in vivo[6]. A 15-residue region in *E. coli* FtsZ and a 17-residue region in *B. subtilis* FtsZ have been implicated to play major role in establishing interactions between FtsZ and the host modulatory proteins, such as FtsA, MinC, ZipA and EzrA, SepF in *E. coli* and *B. subtilis*, respectively. The CTV region has also shown to be essential in determining the lateral interaction potential between FtsZ polymers in vitro and maintaining the integrity of the FtsZ ring in vivo. The C-terminus of FtsZ is capped by a peptide known as "landing pad" constituted by CTT that helps FtsZ establish interactions with multiple other regulatory proteins that are constituents of the divisome[8,9].

The CTL or the C-terminal linker is a highly variable structure[5,6,10]. Chimera experiments suggest that a flexible linker region is essential for the efficient transition of subunits into an active conformation required for polymerization and cooperative assembly of the filaments of FtsZ. Whether the binding of various proteins to CTL causes any structural changes in the globular core, and has any functional implications is yet to be resolved [10].

1.2 FtsZ Intrinsic Curvatures, Conformational Change, and Curvature

FtsZ filament has been observed to exist in curved conformation that helps to promote deformation of the membrane in vitro but little is known about its function in vivo. Few proteins have been implicated in the regulation FtsZ curvature, such as ZapA in *B. subtilis*, FtsA in *Thermotoga* maritima and FzlA in *Caulobacter cresentus*. It has been also studied that curvature of FtsZ filaments contributes to efficient division[11].

FtsZ filaments themselves possess an intrinsic curvature that results in best alignment to support curvatures of 1 μm in radius[12] (Fig. 1). Crystal structure of FtsZ from *Mycobacterium tuberculosis* showed antiparallel protofilament pairs, with the C-terminals facing inward if it were to form a ring[13]. In contrast, some in vitro reconstitution suggests that *E. coli* FtsZ C-terminus is towards the cell wall[14,15]. The intrinsic curvatures also play into how the membrane-targeting sequence (MTS) carrying FtsZ aligns on curved substrates or drive curvatures on free-standing giant unilamellar vesicles. When the MTS is at the C-terminus, the FtsZ aligns on the negative curvatures, compared to N-terminally tagged MTS that aligns on positive curvatures. This argues that there might be species-specific structural differences on how the FtsZ precisely functions and may have non-conserved mechanisms.

Beyond the discrepancy in the intrinsic curvature, it was proposed that upon hydrolysis, the protofilament can increase its curvature. This mechanochemical cycle has been associated with force production in the Z-ring. There are discrepancies in the observed orientations and resulting curvatures that have been proposed between different species. It is yet to be determined, what is the relationship between the orientations, intrinsic curvature, and GTP hydrolysis-induced conformational change resulting in change in curvature.

1.3 FtsZ Protofilament Polarity, Dynamics, Treadmilling and Bundling

Dynamic filaments are known to have structural and kinetic polarity. The growing end of the filament is called the "Plus end" and the opposite end is referred to as the "Minus end". In microtubules, stochastic switching between growth and shrinking phases occurs. The dominant growing end, referred to as the '+' end, grows by the association of an incoming αβ tubulin dimer binding to the C-terminal domain of the protofilament. Mutation studies have provided insight into the fact that the kinetic polarity of FtsZ filaments is opposite to that of microtubules, i.e. the incoming monomer (equivalent of an αβ tubulin dimer in microtubules) binds at the '−' end structural equivalent in microtubule, binding to the exposed N-terminal domain of the FtsZ protofilament through its C-terminal domain[16] (Fig. 2).

Treadmilling is a phenomenon observed by many cytoskeletal filaments, and it occurs due to simultaneous growth and shrinkage in length at each end of the filament, respectively[9,17]. As described above, FtsZ can display dynamic growth on the '−' end and shrinkage at the '+' end. This results in a dynamic filament quasi-linear network that can effectively cover the entire circumference of the divisome region. In vivo measurements of FtsZ turnover will therefore, as a result, appear rapid and dynamic [18].

A recent study found that FtsZ treadmilling performs two major roles during cell division[19]. The first is it arbitrates the condensation of diffused filaments of FtsZ into a dense Z-ring. The second role is to guide the synthesis of septal

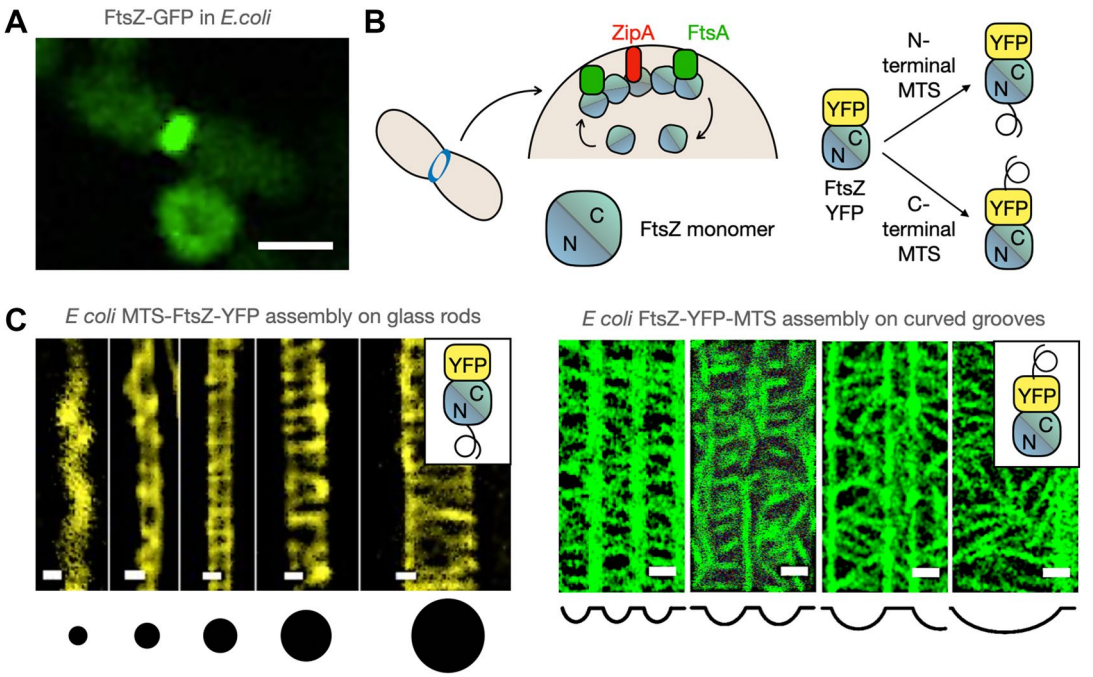

Figure 1: FtsZ protofilaments possesses intrinsic curvature. **a** Image of FtsZ-GFP in the Z-ring in bacteria. **b** Schematic of central FtsZ-interacting cytosolic components of the Z-ring FtsA and ZipA. To assess intrinsic curvature, two artificial constructs, with membrane-targeting sequence at the N-terminus, and at the C-terminus were used. **c** The two constructs align on positively and negatively surfaces coated with membranes, revealing the preferred intrinsic curvature – with the N-terminal domain towards the inner side and C-terminal domain facing the membrane in the context of the Z-ring. The protofilaments show a preference for a diameter of 1 μm in both cases, the diameter of *E. coli.* Scale bars: **a** 1 μm, **c** 500 nm.

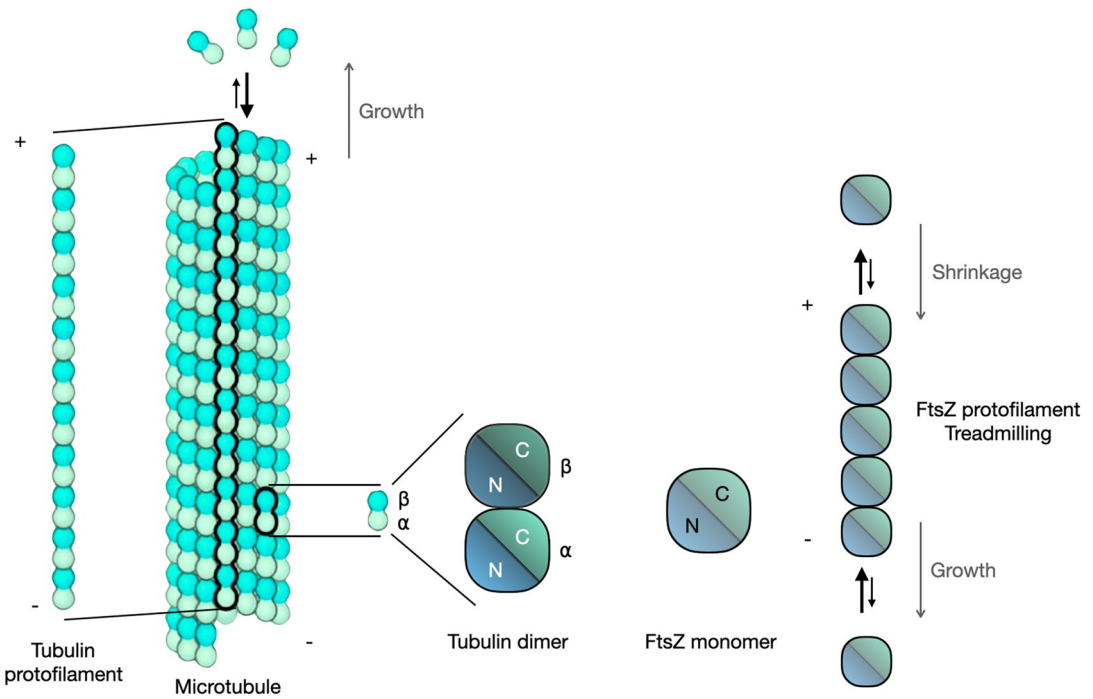

Figure 2: FtsZ filaments have the opposite kinetic polarity of microtubules. Schematic displaying the structural organization of N- and C-terminals with opposite polarity in growing ends.

peptidoglycan, which is essential during the initial phase of ring constriction (as observed in *B. subtilis* cell division). Treadmilling helps to guide the PG synthase molecules during constriction initiation around the mid-cell circumference before the septum is established[20]. FtsZ treadmilling has been revealed to be required until the initiation of constriction and becomes dispensable in the later stages. Although they are not required after constriction initiation, treadmilling has been observed to increase the rate of septal constriction[19].

1.4 The Z-ring as the Organizer

Cell division in bacteria begins with the assembly of FtsZ filaments at the mid-cell to form a structure called the Z-ring. The initial positioning and timing depend on the Min proteins oscillations, following which the ring-like structure appears[1,21]. Z-ring then recruits associated proteins—early proteins, such as FtsA, EzrA, SepF and ZapA, followed by integral membrane proteins or late proteins including Div1B, Div1C, FtsL, Pbp2B, and FtsW. Z-ring constriction occurs during cytokinesis which generates a septum that in turn leads to the division of the cell. FtsZ treadmilling has been found to be a critical step in cell division as it has been shown to directly impact the rate of septum formation. Z-ring condensation is ultimately necessary for cell division as it helps recruit the associated enzymes to the division site[19]. FtsZ bundling condenses the Z-ring and does not affect the treadmilling process. Condensation is especially important, not for the proper activation of the divisome-associated enzymes but to reduce the size of the region and concentrate their activity to that region to allow productive septum formation. In *E. coli*, about 30 proteins interact as part of the Z-ring. They include the Z-associated proteins (ZAPs) A, B, C, D, and F, Membrane tethers of FtsZ—FtsA and ZipA, PG synthases FtsW and FtsI, and their conserved regulators FtsB, FtsL, and FtsQ[22]. The cell wall remodelling proteins make indirect contacts with the Z-ring through other proteins but are integral for septum synthesis. These proteins do not contribute to stabilizing the Z-ring, but rather rely on the FtsZ ring structure itself as a scaffold. Beyond this general role of Z-ring, the question of whether FtsZ on its own is a force generator has been a matter of severe scrutiny as discussed below.

1.5 Can FtsZ Generate Force?

Whether and how bacterial Z-ring generates force for constriction of the septum without any motor proteins remains a puzzle. The three major mechanisms for force generation based on the observed dynamics, such as treadmilling, mechanochemical cycle of GTP hydrolysis, and conformational changes are:

1. *FtsZ filament bending model* [23,24] This model proposes that the force required to constrict the inner membrane and to overcome the turgor pressure wholly comes from the Z-ring. The Z-ring pulls inwards, creating a space between the inner membrane and PG which in turn activates peptidoglycan ingrowth mediated by the various enzymes recruited to the site by FtsZ.
2. *FtsZ condensation model* [19,25] This model suggests that the FtsZ filaments exhibit intrinsic affinity to bundle with each other and this in turn leads to the condensation of the Z-ring into denser structures, generating a force of constriction on the attached membrane.
3. Concerted constriction[26,27] This model considers the concerted contribution of both the PG- pushing and Z-ring pulling. Z-ring generates a constrictive force that helps in directionality/efficiency of the process or it might help regulate the PG remodelling kinetics.

Modelling is an efficient way to quantitatively assess the features of a hypothetical model. Features and outcomes of various mechanisms have relied on such approaches, that have been utilized by various groups to propagate one force generation over the other. Lately, integrated models considering many features of FtsZ dynamics have been developed. The general approach needs to be integrative rather than simply emphasizing on one favoured mechanism. There are no reasons why multiple factors may not be at play in these systems. A very comprehensive model can be found here[28]. However, one of the major caveats in modelling is, that the outcome vastly depends on initial parameters, many of which are estimated or are variable parameters. This is because it is very difficult to precisely measure these parameters experimentally. The major modelling efforts with FtsZ as the major force generator rely on either the bending or the filament sliding-based mechanisms.

A more powerful way to prove the functional capacity of a protein machinery, complex or assembly to perform a certain process is to reconstitute with the minimal number of components[29]. This allows for controlling the components as well as other parameters, such as temperature, buffer composition, etc. that allow systematic investigation. One such approach was pioneered by Osawa and Erickson to reconstitute the Z-ring in vitro[30]. While the FtsZ with an artificially tagged MTS could form a complete ring in thick-walled liposomes, it still lacked clarity on whether it generated force. This is because a high number of amphipathic helices inserting into a membrane themselves can bend the membrane, and the observed mild constrictions may not be from FtsZ-derived force. Similarly, the second approach that included FtsA as the membrane anchor instead of the artificial MTS also suffered from thick-walled vesicles and had one single example[31]. A step forward in the reconstitution was to incorporate de novo synthesis of limited bacterial division components in vesicles [32]. This approach convincingly showed ring-like structures that resulted in vesicle budding. Interestingly, the minimal components required were only FtsA and FtsZ which colocalize in the bacterial Z-ring[33,34]. This puts forward an exciting hypothesis that FtsZ–FtsA, two-component system, might constitute the minimal force producing unit.

How might the FtsZ–FtsA be coordinating to produce force? ZipA and FtsA*, a mutant of FtsA that bypasses the requirement of ZipA[35,]] both have been shown to stabilise FtsZ in GDP-bound mini-ring conformations[36]. If filament bending was the major mode of action here, then it is imperative that the GDP-bound FtsZ protofilament be 'more stably' incorporated in a bundle to relay the force. In simpler reconstitutions, FtsZ–GDP leaves the filament bundle, suggesting that the FtsZ–GDP interface is highly unstable. An active stabilising and anchoring function of FtsA would effectively harness the mechanical energy from GTP hydrolysis of FtsZ and relay to the membrane. There are still existing discrepancies around the following: (1) what is the relationship of intrinsic curvature to GTP-hydrolysis generated curvature? (2) Which way does the hydrolysis bend the filament—away from the membrane, or towards the membrane? (3) How does the dynamic property of FtsZ protofilaments, i.e. treadmilling, operate along with protofilament bending-based mechanisms to generate force, which needs the filament to be stabilised for a certain amount of time? (Fig. 3). The assembly and disassembly rates of a treadmilling FtsZ filament are identical at about 16.8 monomers per second[17]. This dynamics is powered by the GTP hydrolysis activity, which for wild-type FtsZ is about 7 GTP/ FtsZ/ min[37]. The cell division occurs over 25 min, implying that the dynamic filaments undergo multiple rounds of work pulling in the membrane, questions remain regarding the rates at which the membrane is pulled inwards, and what role membrane linking as well as ftsZ-remodelling proteins FtsA and ZipA play. An interesting observation made by Ramirez-Diaz is by switching the membrane binding directionality from C-terminus to the N-terminus, the direction of rotation, an observation as a result of treadmilling[38]. This implies that FtsZ filament is not just curved in a single direction, but rather has a helical geometry[12,38]. How this feature manifests in force generation is still unknown. Perhaps, more can be learnt from functional dissection of FtsA and its interactions[39–44]. The most updated simulations do consider now filament bending in both directions and prove more realistic and in agreement with available data [28].

2 Conclusion

FtsZ from different species seems to have different characteristics. For example, crystal structures from *E. coli and M. tuberculosis* show opposite orientations and different bundling propensities. Similarly, a specialised protein—FzlA has been discovered in *Caulobacter crescentus*, which possesses the ability to remodel FtsZ protofilaments by increasing the curvature and twist them[45]. This protein also controls the constriction rate in vivo. One possibility is that FtsZ might be central to force generation, and many associated modulators may be divergent across species. Phylogenetic analysis of FtsZ has highlighted extremely divergent FtsZ sequences and may signify the plethora of Z-ring-associated protein composition differences across that may serve to modulate the Z-ring to a functional state. An extreme example of variety can be found in *Candidatus thiobon ineisti* which has swapped division planes along the longitudinal axis. Much remains to be discovered from these microorganisms, and identification and proof-of-activity by minimal functional reconstituted systems may lead us to create chemically the minimal living organism. For now, the minimal system approaches have shown that FtsZ, with FtsA, can generate force.

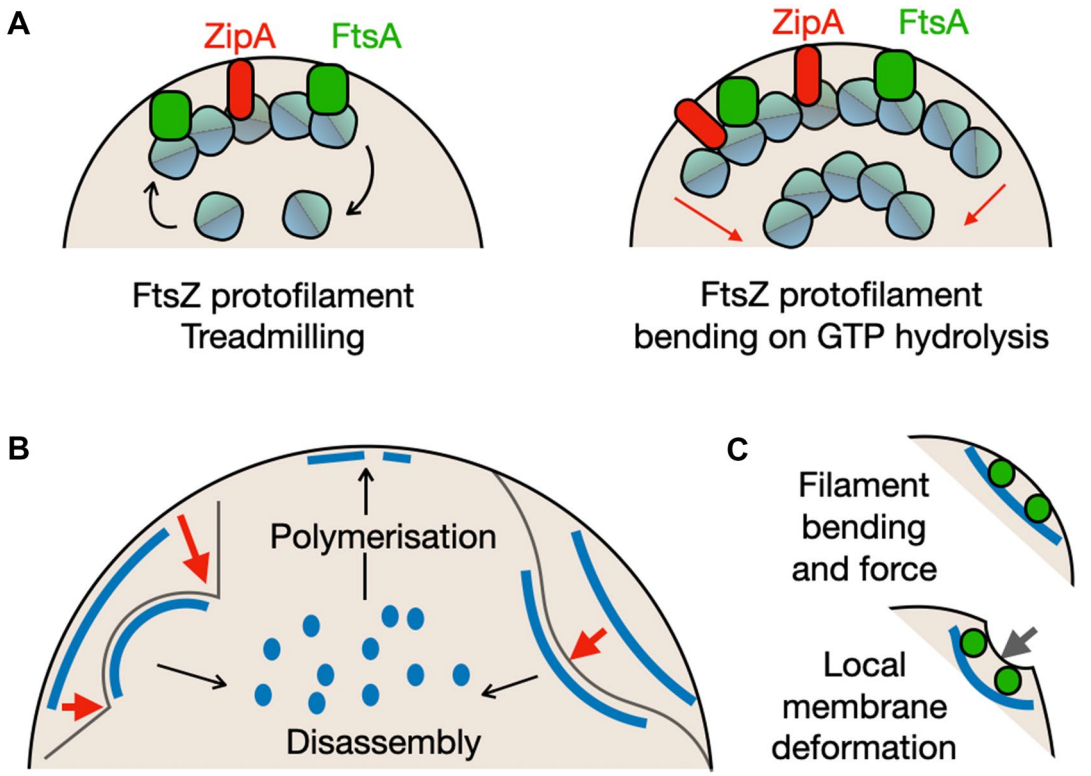

Figure 3: Open questions in FtsZ–FtsA as force-generating units. **a** How can we reconcile between the dynamics observed in treadmilling and the stability required to mechanically pull the cell membrane inwards? **b** What is the orientation of the filaments, the directionality of the intrinsic curvatures? **c** What is the function of FtsA? One putative function is to transduce the force by produced the mechanochemical cycle in FtsZ protofilaments to the membranes, producing local membrane deformations before the protofilaments disassemble.

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Conflict of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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References

1. Adams DW, Errington J (2009) Bacterial cell division: assembly, maintenance and disassembly of the Z ring. Nat Rev Microbiol 7(9):642–653
2. Addinall SG, Bi E, Lutkenhaus J (1996) FtsZ ring formation in fts mutants. J Bacteriol 178(13):3877–3884
3. Nogales E et al (1998) Tubulin and FtsZ form a distinct family of GTPases. Nat Struct Biol 5(6):451–458
4. Lowe J, Amos LA (1998) Crystal structure of the bacterial cell-division protein FtsZ. Nature 391(6663):203–206
5. Buske PJ, Levin PA (2012) Extreme C terminus of bacterial cytoskeletal protein FtsZ plays fundamental role in assembly independent of modulatory proteins. J Biol Chem 287(14):10945–10957
6. Buske PJ, Levin PA (2013) A flexible C-terminal linker is required for proper FtsZ assembly in vitro and cytokinetic ring formation in vivo. Mol Microbiol 89(2):249–263
7. Oliva MA, Cordell SC, Lowe J (2004) Structural insights into FtsZ protofilament formation. Nat Struct Mol Biol 11(12):1243–1250
8. Krol E et al (2012) Bacillus subtilis SepF binds to the C-terminus of FtsZ. PLoS One 7(8):e43293
9. Coltharp C, Xiao J (2017) Beyond force generation: why is a dynamic ring of FtsZ polymers essential for bacterial cytokinesis? BioEssays 39(1):1–11

10. Buske PJ et al (2015) An intrinsically disordered linker plays a critical role in bacterial cell division. Semin Cell Dev Biol 37:3–10
11. Lariviere PJ et al (2018) FzlA, an essential regulator of FtsZ filament curvature, controls constriction rate during Caulobacter division. Mol Microbiol 107(2):180–197
12. Arumugam S et al (2012) Surface topology engineering of membranes for the mechanical investigation of the tubulin homologue FtsZ. Angew Chem Int Ed Engl 51(47):11858–11862
13. Li Y et al (2013) FtsZ protofilaments use a hinge-opening mechanism for constrictive force generation. Science 341(6144):392–395
14. Osawa M, Anderson DE, Erickson HP (2009) Curved FtsZ protofilaments generate bending forces on liposome membranes. EMBO J 28(22):3476–3484
15. Housman M et al (2016) FtsZ protofilament curvature is the opposite of tubulin rings. Biochemistry 55(29):4085–4091
16. Du S et al (2018) FtsZ filaments have the opposite kinetic polarity of microtubules. Proc Natl Acad Sci USA 115(42):10768–10773
17. Yang X et al (2017) GTPase activity-coupled treadmilling of the bacterial tubulin FtsZ organizes septal cell wall synthesis. Science 355(6326):744–747
18. Chen Y, Erickson HP (2005) Rapid in vitro assembly dynamics and subunit turnover of FtsZ demonstrated by fluorescence resonance energy transfer. J Biol Chem 280(23):22549–22554
19. Whitley KD et al (2020) FtsZ treadmilling is essential for Z-ring condensation and septal constriction initiation in bacterial cell division. bioRxiv 2020.07.01.182006
20. Bisson-Filho AW et al (2017) Treadmilling by FtsZ filaments drives peptidoglycan synthesis and bacterial cell division. Science 355(6326):739–743
21. Arumugam S, Petrasek Z, Schwille P (2014) MinCDE exploits the dynamic nature of FtsZ filaments for its spatial regulation. Proc Natl Acad Sci USA 111(13):E1192–E1200
22. Marmont LS, Bernhardt TG (2020) A conserved subcomplex within the bacterial cytokinetic ring activates cell wall synthesis by the FtsW-FtsI synthase. Proc Natl Acad Sci USA 117(38):23879–23885
23. Li Z et al (2007) The structure of FtsZ filaments in vivo suggests a force-generating role in cell division. EMBO J 26(22):4694–4708
24. Lan G, Wolgemuth CW, Sun SX (2007) Z-ring force and cell shape during division in rod-like bacteria. Proc Natl Acad Sci USA 104(41):16110–16115
25. Lan G et al (2009) Condensation of FtsZ filaments can drive bacterial cell division. Proc Natl Acad Sci USA 106(1):121–126
26. Ghosh B, Sain A (2008) Origin of contractile force during cell division of bacteria. Phys Rev Lett 101(17):178101
27. Surovtsev IV, Morgan JJ, Lindahl PA (2008) Kinetic modeling of the assembly, dynamic steady state, and contraction of the FtsZ ring in prokaryotic cytokinesis. PLoS Comput Biol 4(7):e1000102
28. Nguyen LT, Oikonomou CM, Jensen GJ (2019) Simulations of proposed mechanisms of FtsZ-driven cell constriction. bioRxiv 737189
29. Arumugam S, Chwastek G, Schwille P (2011) Protein-membrane interactions: the virtue of minimal systems in systems biology. Wiley Interdiscip Rev Syst Biol Med 3(3):269–280
30. Osawa M, Erickson HP (2013) Liposome division by a simple bacterial division machinery. Proc Natl Acad Sci USA 110(27):11000–11004
31. Osawa M, Anderson DE, Erickson HP (2008) Reconstitution of contractile FtsZ rings in liposomes. Science 320(5877):792–794
32. Godino E et al (2020) Cell-free biogenesis of bacterial division proto-rings that can constrict liposomes. bioRxiv 2020.03.29.009639
33. Ma X, Ehrhardt DW, Margolin W (1996) Colocalization of cell division proteins FtsZ and FtsA to cytoskeletal structures in living *Escherichia coli* cells by using green fluorescent protein. Proc Natl Acad Sci USA 93(23):12998–13003
34. Ma X et al (1997) Interactions between heterologous FtsA and FtsZ proteins at the FtsZ ring. J Bacteriol 179(21):6788–6797
35. Geissler B, Shiomi D, Margolin W (2007) The ftsA* gain-of-function allele of *Escherichia coli* and its effects on the stability and dynamics of the Z ring. Microbiology (Reading) 153(Pt 3):814–825
36. Chen Y et al (2017) ZipA and FtsA* stabilize FtsZ-GDP miniring structures. Sci Rep 7(1):3650
37. Arjes HA et al (2015) Mutations in the bacterial cell division protein FtsZ highlight the role of GTP binding and longitudinal subunit interactions in assembly and function. BMC Microbiol 15:209
38. Ramirez-Diaz DA et al (2018) Treadmilling analysis reveals new insights into dynamic FtsZ ring architecture. PLoS Biol 16(5):e2004845
39. Pichoff S, Du S, Lutkenhaus J (2018) Disruption of divisome assembly rescued by FtsN-FtsA interaction in Escherichia coli. Proc Natl Acad Sci USA 115(29):E6855–E6862
40. Pichoff S, Lutkenhaus J (2002) Unique and overlapping roles for ZipA and FtsA in septal ring assembly in *Escherichia coli*. EMBO J 21(4):685–693
41. Pichoff S, Lutkenhaus J (2007) Identification of a region of FtsA required for interaction with FtsZ. Mol Microbiol 64(4):1129–1138
42. Pichoff S et al (2012) FtsA mutants impaired for self-interaction bypass ZipA suggesting a model in which FtsA's self-interaction competes with its ability to recruit downstream division proteins. Mol Microbiol 83(1):151–167
43. Shiomi D, Margolin W (2007) Dimerization or oligomerization of the actin-like FtsA protein enhances

the integrity of the cytokinetic Z ring. Mol Microbiol 66(6):1396–1415

44. Shiomi D, Margolin W (2008) Compensation for the loss of the conserved membrane targeting sequence of FtsA provides new insights into its function. Mol Microbiol 67(3):558–569

45. Goley ED et al (2010) Imaging-based identification of a critical regulator of FtsZ protofilament curvature in Caulobacter. Mol Cell 39(6):975–987

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