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Overview of cell shape: cytoskeletons shape cells

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Introduction

Bacterial cells come in a variety of shapes although the best studied and most commonly encountered species are either spherical or rod-shaped. Phylogenetic analysis indicates that spherical-shaped bacteria arose periodically during evolution from rod-shaped precursors, probably due to a loss of genes [1]. Consistent with this, rod-shaped bacteria can be converted to a spherical morphology by deletion of certain genes [2]. Other bacteria with more elaborate shapes, such as curved or spiral, have additional genes that are responsible for their distinctive shape.

The distinct shape of most bacteria is due to their cell wall or peptidoglycan (PG), which retains the shape of the cell it is isolated from. Thus, the study of bacterial morphogenesis is focused on this component of the cell envelope. Over the years, the effort to explain cell shape has shifted emphasis from the architecture of the PG to spatial regulation over the insertion of new material [3]. Rod-shaped bacteria are viewed to have a general PG synthetic system, sufficient for overall increase in cell size [4]. Imposed upon this general system are two distinct modifiers, one causing elongation and one causing septation. In *E. coli*, genes were identified that are required for septation (mostly designated *fts*) and other genes (*mreB*, *mreC*, *mreD*, *rodA* and *pbpB* [PBP2]) that are required for elongation. But how do these genes influence the biosynthetic PG machinery to exert their effect on cell shape?

A mechanism to account for spatial regulation of PG synthesis arose from the studies of cell division in *E. coli*. Septation involves localized (septal) PG biosynthesis which requires cell division genes and is sensitive to a subgroup of β -lactams [5]. Over the past 15 years it has become clear that this septal PG synthesis requires a cytoskeletal element, the Z ring, composed of polymerized FtsZ, the ancestral homologue of eukaryotic tubulin [6]. The Z ring recruits at least a dozen proteins required for cell division, including FtsI (PBP3), the target of septal specific β -lactams. Although many details remain to be worked out, the results laid the foundation for a cytoskeletal element as the foundation for localized PG synthesis.

Errington's group found that MreB, known to be required for rod shape, assembled into helical cables that extend between the poles of the cell [7]. Furthermore, the structural similarity of MreB to eukaryotic actin, including its ability to assemble in vitro, confirmed that it was a *bonafide* cytoskeletal protein [8]. These studies raised the possibility that MreB could influence PG synthesis to affect a rod shape and have reignited efforts to understand how cell shape is determined.

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Septation

The Z ring, present in nearly all bacteria, recruits proteins specifically required for cell division. Among these proteins are FtsI and FtsW which have been shown to be essential for septal PG synthesis. In addition, the Z ring also causes the re-localization of enzymes that are less specialized and also required for lateral PG expansion such as PBP1B, a transglycosylase [9]. Transglycosylases are part of the general PG synthetic machinery and are not specific for cell division. The Z ring also recruits PG hydrolases necessary for progression of septation and for the separation of the daughter cells after division [10–12]. Importantly, mutations in *ftsZ* that markedly reduce the GTPase activity produce a spiral FtsZ resulting in a spiral septum indicating that the morphology of the septal FtsZ structure dictates the pattern of PG growth [13,14].

Regulation of Z ring formation plays a role in cell size determination. In rich media bacteria grow faster and divide at a larger size than in poor media but the mechanisms that coordinate cell size with growth rate are relatively unexplored. A recent report [15] shows that UgtP, an enzyme involved in glucolipid biosynthesis in *B. subtilis*, also has the ability to inhibit FtsZ polymerization. Cells depleted for this enzyme (or ones upstream in the same pathway) assemble Z rings and divide at a shorter cell length than the wild type. Thus, an active UgtP reduces FtsZ activity, delaying cytokinesis. These findings suggest that this dispensable pathway is utilized as a metabolic sensor for nutritional regulation of cell length. In absence of this metabolic sensor, however, timely formation of Z rings is still regulated at slow growth rate. This implies that other control systems, possible Min and Noc, are responsible for linking formation of the Z ring to the cell cycle at slower growth rates [6].

Elongation

A rod shape requires the *mre* genes (*mreB*, *mreC* and *mreD*) as well as *rodA* and *pbp2*. Although RodA and PBP2 are required for lateral PG synthesis (analogous to the role of the orthologues FtsW and FtsI, respectively in septal PG synthesis), the mechanism by which *mre* genes determines the rod shape of the cell is an area of intense investigation. Several lines of experiments indicate that they organize the PG synthetic machinery essential for elongation. As visualized in different bacteria, MreB forms a dynamic helix underneath the cytoplasmic membrane extending from one pole to the other [7,16,17]. *E. coli* and *C. crescentus* have one MreB but *B. subtilis* has 3 paralogues (MreB, Mbl and MreBH) which colocalize but are able to assemble independently and may have distinct functions [18]. In addition, MreC forms a helical structure in the periplasmic space [19,20]. The crystal structure of the periplasmic domain of MreC reveals a dimeric structure and a model for filament formation [21].

Development of fluorescent antibiotics that specifically targets PG precursors has allowed researchers to examine where they are translocated in Gram-positive bacteria [22,23]. The outer membrane in Gram-negative bacteria is not permeable to these fluorescent antibiotics, however, an alternative technique, D-cysteine labeling, allows differentiation of old and new PG [24–26]. During cell elongation, the insertion of new PG occurs along the lateral cell wall, but not at the cell poles, and appears to be helical in both *B. subtilis* [22,23••] and *E. coli* [25•]. Of course, localized PG synthesis at the division site is also observed by these techniques and as expected is Z ring dependent. For cocci, a septal model of PG synthesis appears to be the only mode of cell wall growth [27] which also appears to be the case for *E. coli* round mutants (*pbpA*, *rodA*) that have lost rod shape [28].

A second approach to examine wall growth is to localize GFP-tagged enzymes involved in PG synthesis and degradation (for review see [29]). In *E. coli*, the localization of RodA and PBP2 seems to be directed, at least in part by MreB [30]. In *C. crescentus* [19,20] PBP2 localization in a helical pattern requires MreB but is also dependent on MreC. If MreB is depleted, newly

synthesized PBP2 localizes to the septum while preexisting PBP2 molecules remain in their helical pattern associated with MreC. These results suggest that MreB distributes PBP2 along the cell length away from the septum. This location would then be maintained by MreC, which interacts with high molecular weight PBPs [20,21]. This role of MreB and MreC in regulating PBP2 localization is interesting because in both *E. coli* and *B. subtilis*, MreB organization in a spiral depends on the presence of MreC and MreD [30,31]. In addition MreB has been shown to interact with MreC which itself interacts with MreD [30] suggesting that these proteins are in a complex.

The presence of three paralogues of MreB in *B. subtilis* may have allowed for evolution of distinct functions. Mbl, has been shown to be responsible of the localization of newly synthesized peptidoglycan [22] although this result is disputed in a recent report [23]. MreBH seems to be responsible for LytE localization and has been proposed to have a specialized function in directing lateral wall hydrolysis [18].

Due to the similarities in the patterns for the localization of MreB and PBP2 and the insertion of new PG precursors along the lateral cell wall, MreB and its paralogues are thought to recruit, probably by direct interaction, the different elements of the PG synthetic machines to their proper location in the cells. This would be similar to the way in which FtsZ recruits proteins into a complex involved in septum synthesis.

Spherical-shaped organisms generally lack MreB consistent with a role in determining rod shape [22]. However, several rod shaped organisms, including *Corynebacterium diphtheriae* and *Streptomyces coelicolor*, either lack MreB or do not require it for vegetative growth. These organisms display an unusual staining pattern with fluorescent vancomycin indicating PG synthesis is occurring at the tips or poles during elongation. For *Streptomyces* formation and elongation of branches requires DivIVA, a coiled coil protein capable of self assembly [32]. Possibly DivIVA forms an additional cytoskeletal element that recruits the PG synthetic machinery to a point on the lateral wall resulting in branch growth [33]. In *Corynebacterium diphtheriae* staining is observed at the poles and the septum. One possibility is that the PG biosynthetic machinery recruited during septation is retained at the poles and remains active [22].

Do roles for FtsZ and MreB overlap?

Although the Z ring drives PG synthesis during cell division, there are several recent reports suggesting that FtsZ outside of the septation process may also have a role in elongation and maintenance of the shape of the cell wall. First, there is the Z-ring dependent, but PBP3-independent, penicillin-insensitive PG synthesis (PIPS) which produces a ring of inert PG at sites where septation is blocked [24]. Its physiological role remains uncertain, however, it suggests that FtsZ can drive PG synthesis independent of the other cell division machinery (such as FtsA and PBP-3)

More recently, work done in Young's laboratory showed that specific PBP mutants of *E. coli* grow with a spiral-like morphology when FtsZ's ability to polymerize into a ring is inhibited [34]. The authors later showed that if MreB protein directs the helical incorporation of new PG into the lateral wall, the location of this incorporation also depended on FtsZ's activity [25]. When FtsZ's ability to form a ring was impaired the incorporation of new PG occurred mainly in the central region of the cell and was significantly lower in regions close to the poles, suggesting a role for FtsZ in directing PG synthesis during cell elongation. These results come at the same time as the realization that FtsZ spirals exist throughout the cell, even in the absence of the Z ring [35,36]. Whether these spirals are responsible for FtsZ's ability to affect PG away from the division site is suspected but not proven.

Finally, in *C. crescentus*, FtsZ appears to redirect PG precursor synthesis to the midcell region well before cell constriction. It recruits MurG which is responsible of the formation of lipid II an essential intermediate in PG synthesis [26]. By doing this, the Z ring would, at least in this organism, be responsible for directing part of the cell wall elongation process. This role of the Z ring in elongation is not essential, however, as cells depleted of FtsZ are still able to increase their length at the same rate as cells containing FtsZ [26]. *E. coli* does not appear to use this mechanism as MurG is a late recruit to the division site, localizing there only after PBP3 and FtsW, around the time of the start of constriction [37].

In *C. crescentus* and *R. sphaeroide*, MreB relocates to the Z ring before division. This implies MreB has a role in the septation process in these organisms even though it doesn't in *E. coli* [38]. This relocation of MreB to the Z ring in some organisms causes us to wonder if it or possibly another type of cytoskeletal element could drive the cell division process in bacteria that lack FtsZ, such as chlamydiae. It would require that spatial regulation, normally conferred on the Z ring, be conferred to that cytoskeletal element.

Other cytoskeletons for more elaborate shapes

Other proteins, not as universally conserved as MreB or FtsZ, are able to self assemble into large polymers and have been shown to be responsible for certain bacterial shapes. A few examples include crescentin (*creS*) (an homologue of intermediate filaments) which is responsible for the croissant shape of *C. crescentus* [39] and the periplasmic flagella which causes *Borrelia* and other spirochetes to acquire their typical wavy shape in addition to providing motility (reviewed in [40]).

The presence of cytoskeletal elements inside cells leading to more elaborate shapes also raises the question of how cytoskeleton proteins mold the cells into a certain shape? Do they only direct cell wall synthesis or can they provide a force strong enough to influence how cell wall enzymes are remodelling the PG to reduce cell wall stress? Filamentous *E. coli* cells growing in microchambers take on the shape of the chamber indicating that an external force can dictate the shape of the cell [41]. Furthermore, cells released from these chambers retain their shape.

Some cytoskeletal elements have even been observed by cryotomographic microscopy (cryoTEM) [42] but are not yet associated to any known proteins. This implies that there could be more cytoskeleton-type elements to be found which may be involved in cell shape determination in organisms that lack the known cytoskeletal proteins.

Conclusions

At our current level of understanding it is becoming clear that cytoskeletal proteins are providing a scaffold for machineries involved in PG synthesis which ultimately determines bacterial cell shape. In the near future the different components of these machines should be identified and how these machines are recruited by the different types of cytoskeleton. It should also be interesting to sure out how these machines are activated at different times during the cell cycle and what are the interconnections between them.

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