domains might be too difficult to unfold or the yeast proteasome might have a very weak unfolding activity. Of course, it is also possible that the processing of Spt23 and Mga2 in yeast occurs by a different mechanism than processing in flies and vertebrates.

It will be interesting to further investigate the differences in processing determinants used by different biological systems. In addition, the number of known examples of proteasomal processing is small but growing. Whether this type of processing is a regulatory mechanism restricted to a handful of transcription factors or is instead a more general cellular function of the proteasome will be an important question for future work.

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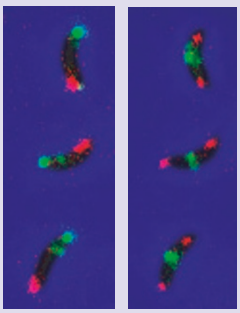
A tale of two halves

The ability of cells to position the site of the division plane so that two daughter cells with full genomes are faithfully generated from generation to generation is an intriguing aspect of cell biology. In bacteria, the site where the membrane pinches in during cell division (the midzone) is defined by the location of a ring-like structure formed by a tubulin-like protein, FtsZ. FtsZ localization was thought to be directed by two mechanisms. The first uses the membrane-tethered MinCD complex to inhibit FtsZ polymerization at the poles, thereby directing FtsZ filaments to the midzone. In the second, FtsZ assembly is prevented near the bacterial nucleoid by nonspecific chromosome-binding factors. As the nucleoid resides at the midcell until replication forces the two chromosomes to segregate to opposite poles, this second mechanism prevents FtsZ assembly from occurring before segregation but leaves the midzone free of inhibitor after segregation. However, neither of the two systems seems to operate in *Caulobacter crescentus*.



In a recent study, Martin Thanbichler and Lucy Shapiro have defined a new mechanism by which the FtsZ ring is properly positioned (*Cell* **126**, 147–162, 2006). In a screen for cell cycle-regulated genes, they isolated an essential gene encoding an ATPase of unknown function, termed MipZ. When MipZ function was repressed, cells became elongated, with the division site occurring unequally. When MipZ was overexpressed, cells again became elongated but there was little cell division, and the division that did occur was focused at the extreme ends of the cell.

Caulobacter exists in two phases: a mobile swarmer cell with a polar flagellum and an immobile stalk cell with a stalk replacing the flagellum. When a new flagellum forms opposite the stalk, the cell divides asymmetrically to yield swarmer and stalk cells. The authors observed that MipZ localizes to the flagellar pole in swarmer cells and to both poles in stalk cells before cell division, after which MipZ is found at the stalk pole. This localization pattern is reminiscent of that of the replication origin, and indeed, MipZ colocalizes with the origin, although the signals do not entirely overlap. The slight discontinuity in signals suggested that MipZ might actually associate with a cluster of sites (*parS*) for ParB, a DNA-partitioning protein that is located several kilobases from the origin. This was confirmed by colocalization and reconstitution of the ParB-MipZ interaction in *Escherichia coli*. It is important to note that although MipZ forms a focus at *parS*, mediated by its interaction with ParB, it forms a gradient toward the midcell.

When MipZ's ATPase motif is mutated, the protein becomes evenly distributed through the cell rather than focused at the origin, and the cells have a filamentous appearance similar to what occurs with MipZ overexpression. FtsZ localizes where MipZ is not present (that is, at the pole opposite the flagellum in swarmer cells and in the midcell during S phase). Therefore, to test whether MipZ has a direct effect on FtsZ assembly, MipZ expression was induced after formation of the FtsZ ring. Immediately, the ring dissolved and cell division was inhibited. By contrast, when MipZ was depleted, FtsZ formed a ring but also other foci, and the division plane was displaced. These results show that MipZ is necessary for the formation of one correctly positioned FtsZ ring.

At the start of S phase, ParB is located on an origin near the stalked pole, and FtsZ is at the opposite pole (left panel; ParB is red and FtsZ is green). MipZ is produced, and the origin, with ParB and MipZ, moves toward the opposite pole. When this happens, FtsZ is rapidly displaced from the pole and appears at the midcell, where polymerization can occur (right panel). The data suggest that FtsZ always localizes to the area containing the lowest concentration of MipZ (which maximizes its distance from ParB and the origin).

But is there a direct effect of MipZ on FtsZ assembly? The addition of GTP to FtsZ promotes its assembly *in vitro*. When MipZ is included, less FtsZ polymer is formed, the polymers are shorter and curved, and they are associated with MipZ. This result resembles what is seen when depolymerizing tubulin is bound by GDP at the ends of microtubules; in agreement with this analogy, MipZ was found to increase the GTPase rate of FtsZ, thereby affecting its assembly.

This study reveals another way in which chromosome movement can be coupled to cell division. MipZ can both interact with ParB, similarly to ParA DNA-partitioning proteins, and inhibit FtsZ ring assembly, similarly to MinC (although by a different mechanism).

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