

BACTERIAL PHYSIOLOGY

E. coli shape up

In addition to regulating bacterial cell division, FtsZ has a role in determining cell morphology, according to a recent report in the *Journal of Bacteriology*.

The processes of bacterial cell division and regulation of cell morphology have often been regarded as independent; however, this study shows that, in addition to being the major regulator of bacterial cell division, FtsZ also influences cell shape through interactions with the low-molecular-weight (LMW) penicillin-binding proteins (PBPs), some of which function as D-alanyl-D-alanine carboxypeptidases and are involved in peptidoglycan biosynthesis — a major determinant of cell morphology.

It has previously been observed that *Escherichia coli* cells lacking multiple PBPs have inappropriately placed, metabolically inert peptidoglycan, which distorts the geometry of the cells. Normally, the only metabolically inert peptidoglycan is found at the cell poles, and as the cell poles are created by septation, Archana Varma and Kevin Young set out to test the hypothesis that FtsZ could have an important role in determining cell morphology.

Using a temperature-sensitive *ftsZ* mutant (*ftsZ84*), which allowed the activity of FtsZ to be controlled, the authors looked at the effects of a partially active FtsZ protein (FtsZ84) and mutations in the genes encoding LMW PBPs. *E. coli* cells expressing FtsZ84 were found to exhibit an

increase in branching, and the authors reasoned that if the septation machinery and PBPs cooperated to determine cell shape, the extent of branching or morphological abnormalities would be increased in *ftsZ84* mutants lacking specific PBPs. Indeed, deletion of the gene encoding PBP5 in a *ftsZ84* mutant yielded a six-fold increase in branching. Complementing this double mutant with wild-type PBP5 eliminated the branching and abnormalities; however, the involvement of both PBP5 and FtsZ was highlighted by the observation that in cells expressing

wild-type FtsZ no branching or morphological abnormalities were observed, even when PBP5 was deleted. The effects of mutations in other PBP genes were also examined, and the authors found that in *ftsZ84* mutants lacking PBP5 the additional deletion of PBP7 or PBP4 led to an increase in branching or abnormalities, respectively. Interestingly, in the absence of PBP5, no morphological variation was observed with either fully active or inactive FtsZ, indicating that irregular cell shapes are dependent on partially active FtsZ.

The authors went on to investigate the effect of inhibition of wild-type FtsZ. Surprisingly, when FtsZ was inhibited with either SulA or MinD in cells lacking PBP5, some of the cells exhibited a left-handed spiral morphology. When PBPs 4, 6 or 7 were also deleted, the number of spiral cells increased.

Although further work is required to determine the mechanism of interaction, this study clearly demonstrates a link between the regulation of bacterial cell division and determination of cell morphology.

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References and links

ORIGINAL RESEARCH PAPER FtsZ collaborates with penicillin-binding proteins to generate bacterial cell shape in *Escherichia coli*. *J. Bacteriol.* **186**, 6768–6774 (2004)

WEB SITE

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