Classic Spotlight: Discovery of *ftsZ*

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Cytokinesis (cell division, fission, and septation) in bacteria is driven by a ring-shaped organelle containing dozens of distinct proteins. Among these, FtsZ plays preeminent roles in assembly and function of the organelle. FtsZ is also the first recognized cytoskeletal protein in prokaryotes and the likely progenitor of the tubulins. The discovery of *ftsZ* was inherently important. It also nicely illustrated the power of classic bacterial and phage genetics, including the ability to clone genes without ever purifying DNA (1).

Much of what we now know about the division process is based on early work with *Escherichia coli*. One important aim in the 1960s was to understand why and how DNA damage causes cells to grow into long filaments with a (usually) transient inability to divide. This inspired Piet van de Putte and colleagues to start the search for genes involved in the division process by isolating mutants that readily passed a 5- μ m filter after growth at 30°C but failed to do so after growth at 42°C due to a division defect. He coined these genes *fts*, for filamentous growth is thermosensitive (2). In the next decade, other groups isolated additional mutants and began to fine-map *fts* mutations on the *E. coli* chromosome. By the end of the 1970s, it was clear that the 2-min region contains several genes involved in murein synthesis (*ddl* and *murC*, *murE*, and *murF*) and envelope permeability (*envA* or *lpxC*) and at least two *fts* genes, *ftsA* and *ftsI* (also called *sep* and *pbpB*).

Around this time in Willie Donachie's lab in Edinburgh, Joe Lutkenhaus succeeded in isolating specialized transducing λ phages carrying the *ftsA* region. He used these to detect the FtsA protein for the first time (3) and to set the stage for a classic paper published in the *Journal of Bacteriology* (1). In the study, the authors isolated mutant [*ftsA*(Ts)] and deletion derivatives of *ftsA*-transducing λ phages and used these to determine if a subset of established *fts* strains could all be corrected by wild-type *ftsA* or if perhaps additional *fts* genes reside near *ftsA* on the chromosome.

Several of the original van de Putte strains turned out to indeed carry temperature-sensitive alleles of *ftsA*. However, strain PAT84 from Francois Jacob's lab was shown not to carry *ftsA*(Ts), as previously assumed (4), but to be affected instead in a novel gene located between *ftsA* and *lpxC*. As the alphabet was being usurped by sometimes questionable *fts* assignments in a confusing literature, naming the gene *ftsZ* was a safe bet (1). Evidence that it was of special importance followed soon after with the discoveries by Lutkenhaus and others that FtsZ is a direct target of the SOS response to DNA damage (solving the old riddle) and that an increase in FtsZ concentration leads cells to form extra septa. Of course, numerous studies have firmly established the central roles of FtsZ in the fission process since.

REFERENCES

1. Lutkenhaus JF, Wolf-Watz H, Donachie WD. 1980. Organization of genes in the *ftsA-envA* region of the *Escherichia coli* genetic map and identification of a new *fts* locus (*ftsZ*). *J Bacteriol* 142:615–620.
2. van de Putte P, van Dillewijn J, Roersch A. 1964. The selection of mutants of *Escherichia coli* with impaired cell division at elevated temperature. *Mutat Res* 106:121–128.
3. Lutkenhaus JF, Donachie WD. 1979. Identification of the *ftsA* gene product. *J Bacteriol* 137:1088–1094.
4. Hirota Y, Ryter A, Jacob F. 1968. Thermosensitive mutants of *E. coli* affected in the process of DNA synthesis and cellular division. *Cold Spring Harbor Symp Quant Biol* 33:677–693. <http://dx.doi.org/10.1101/SQB.1968.033.01.077>.

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