Characterization of C-terminal structure of MinC and its implication in evolution of bacterial cell division

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Abstract

Bacterial cells have a fundamental need to divide by binary fission through accurate spatial and temporal regulation of septum formation, producing two daughter cells of equal size [1]. Proper cell division at the mid-site of Gram-negative bacteria reflects stringent regulation by the *min* system (MinC, MinD and MinE) [2]. Herein we solved the crystal structure of the C-terminal domain of MinC from *Escherichia coli* (*Ec*$MinC_{CTD}$). *Ec*$MinC_{CTD}$ forms a dimer between the two β-sheets in each subunit, as observed in the *Thermotoga maritima* $MinC_{CTD}$ structure (*Tm*$MinC_{CTD}$). However, both *Ec*$MinC_{CTD}$ and *Tm*$MinC_{CTD}$ lack an α-helix (helix3) at their C-terminal tail, in comparison to *Aquifex aerolicu* $MinC_{CTD}$ (*Aa*$MinC_{CTD}$) which forms an extra interaction interface with MinD. By fusing helix3 to the C-terminus of *Ec*MinC, we studied its effect on cell morphology and cell growth, revealing that *Aa*helix3 impaired normal cell division in *E. coli*. Furthermore, results of a co-pelleting assay and binding free energy calculation suggested that *Aa*helix3 plays an essential role in *Aa*MinCD complex formation, under the circumstance of lacking MinE in *A. aerolicu*. Combining these results with sequence analysis of MinC and MinD in different organisms, we propose an evolutionary relationship to rationalize different mechanisms in cell division positioning in various organisms.

Reference

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2. Deboer PAJ, Crossley RE, Rothfield LI. A division inhibitor and a topological specificity factor coded for by the minicell locus determine proper placement of the division septum in *E. coli*. *Cell*. 1989;56:641–649.