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Abstract

FtsZ filaments localize at the middle of the bacterial cell and participate in the formation of a contractile ring responsible for cell division. Previous studies demonstrated that the highly conserved negative charge of glutamate 83 and the positive charge of arginine 85 located in the lateral helix H3 bend of *Escherichia coli* FtsZ are required for *in vivo* cell division. In order to understand how these lateral mutations impair the formation of a contractile ring, we extend previous *in vitro* characterization of these mutants in solution to study their behavior on lipid modified surfaces. We study their interaction with ZipA and look at their reorganization on the surface. We found that the dynamic bundling capacity of the mutant proteins is deficient, and this impairment increases the more the composition and spatial arrangement of the reconstituted system resembles the situation inside the cell: mutant proteins completely fail to reorganize to form higher order aggregates when bound to an *E.coli* lipid surface through oriented ZipA. We conclude that these surface lateral point mutations affect the dynamic reorganization of FtsZ filaments into bundles on the cell membrane, suggesting that this event is relevant for generating force and completing bacterial division.