THE H3 HELIX OF THE TUBULIN-LIKE PROTEIN FtsZ IS THE LIKELY TARGET FOR ZapA INTERACTION IN *Bacillus subtilis*

Bisson-Filho, A.W.[1], Varvakis, T. A.[1], Gueiros-Filho, F.J.[1]

[1]Departamento de Bioquímica, IQ, USP

FtsZ is a prokaryotic homolog of tubulins, essential for cellular division and widely conserved in bacteria. FtsZ self-associates into a ring structure (Z ring) that establishes the place where division will occur. The Z ring represents a scaffold to which other division proteins will associate to create the new septum. Z ring formation *in vivo* is affected by a number of proteins that interact with FtsZ and modulate its polymerization properties. These modulatory proteins, which either inhibit or stimulate the assembly of FtsZ, play key roles in the regulation of division. The objective of this work was to elucidate how FtsZ interacts with ZapA, a modulator that stabilizes FtsZ polymerization and Z-ring formation. We created a mutagenized *ftsZ* plasmid library by error prone PCR, and used it to select FtsZ mutants that restored the interaction with ZapA^{N62A}, a ZapA mutant which had lost the ability to bind to FtsZ and stabilize its polymers. To do this we exploited a situation in which the interaction between ZapA and FtsZ is essential for bacterial survival. More specifically, we looked for mutants that rescued the ability of ZapA^{N62A} to suppress the lethal effect of overexpressing an FtsZ inhibitor (the protein MinD) in *B. subtilis* cells. Using this genetic selection we have screened 70000 transformants and found one FtsZ mutant that survives MinD overexpression in the presence of ZapA^{N62A}. The mutant, FtsZ^{E91V} , has a substitution in the N-terminal H3 helix and restored the interaction between FtsZ and ZapA, as seen by the localization of GFP-ZapA^{N62A} on Z rings. Thus, our data suggests that the H3 helix is the probable target region of ZapA in FtsZ. We are currently randomizing other aminoacids of the H3 helix and investigating the properties of these mutants.

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