Site-specific mutations of FtsZ - effects on GTPase and in vitro assembly

ABSTRACT

Some mutants containing reduced GTPase still complement ftsZ84, suggesting that the high level of GATPases observed in vitro is not necessary for in vivo function. However, all of the lateral mutant fail to complement it, indicating that these surfaces of various protofilaments may play a role in association with Ftz protofilements, which may be different from the structures assembled in DEAE dextran or calcium.

INTRODUCTION

The FtsZ gene is a transcription factor that has been associated with a number of diseases, including diabetes, atherosclerosis, type II diabetes, and multiple sclerosis.

As the gene has been associated with a number of diseases, it is important to understand the mechanisms involved in the interaction between FtsZ and the other transcription factors.

To date, there is no consensus on the mechanisms involved in the interaction between FtsZ and the other transcription factors, and a number of different studies have been carried out to try to understand the effects of FtsZ mutations on other transcription factors.

In this study, we will focus on the effects of FtsZ mutations on the FtsZ gene in terms of the effects on other transcription factors and to understand how FtsZ mutations lead The cytoskeletal framework of the bacterial cell division machine of prokaryotes is thought to be formed by FtsZ recombinantly expressed in vitro. The FftzZ protein is homologous to tubulin, and the orientation of FntSZ subunits in the protofilament can be determined by comparing their atomic structures. If tubular, rectangular shape is considered a cube, it would have six faces: the top and bottom faces of each Fb, which are also the right and We constructed 16 mutations to identify essential amino acids for FtsZ function, mainly by reversing conserved aspartate and glutamate residues in favor of alanine. Although our initial choice of amino acid for mutation was based on conservation across species, we later created additional mutation types to address specific structural questions. The main objective of this study was to characterize the in vitro assembly and GTPase of the mutant proteins, but we also conducted preliminary analysis of their in vivo function by testing ftz84. Our analysis of the structurally important side chains of FtsZ includes over a dozen mutations that have been previously described in previous studies.

CONCLUSION

Remarkable conclusions The 16 mutants were specifically designed to assemble and GTPase in vitro, and their ability to function in vivo was tested. They were found to be mostly benign, with normal assembly on the front and back of the FtsZ protofilament. However, some mutations significantly reduced their in-vitro GpaSe levels, indicating that these mutation types could not interfere with DEAE dextran's assembly or cloned structures. Furthermore, they mapped to the sides of PROFin