Abstract

Mycobacterium tuberculosis uses several strategies to evade the innate immune response. It has been suggested that the secretion of serine protein kinase G (PknG) inhibits the phagosome-lysosome fusion, allowing the survival of the mycobacteria. The protein PknG of M. tuberculosis harbor, in addition to the kinase domain, two thioredoxin (Trx) motifs and one tetratricopeptide (TPR) motif. In this work a M. tuberculosis PknG mutant (M. tuberculosis pknGCt::hyg) was constructed which has an interruption at the C-terminal region of pknG gene. No significant difference was observed during the exponential growth phase between the mutant and parental strains. However, survival of the mutant strain was much lower than that shown by the wild type strain during the stationary phase. This phenotype could not be complemented by the wild type PknG, probably due to the accumulation of the PknGCt mutant protein in the bacterial cell. Alteration of colony morphology in the mutant strain was also observed. Finally, using a bacterial two-hybrid system, the C-terminal domain of PknG was shown to be involved in its dimerization and interactions with its substrate.

Keywords

Deletion, Kinase, M. tuberculosis, Mutant, TPR Motifs