J Bacteriol Parasitol 2018, Volume: 9
DOI: 10.4172/2155-9597-C1-044

6th International Congress on
Bacteriology & Infectious Diseases
May 21-22, 2018 | New York, USA

PknG supports mycobacterial adaptation in acidic environment

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Mycobacterium tuberculosis (Mtb), causative agent of human tuberculosis (TB), has the remarkable ability to adapt to the hostile environment inside host cells. Eleven eukaryotic like serine-threonine protein kinases (STPKs) are present in Mtb. Protein kinase G (PknG) has been shown to promote mycobacterial survival inside host cells. A homolog of PknG is also present in *Mycobacterium smegmatis* (MS), a fast grower, non-pathogenic *Mycobacterium*. In the present study, we have analysed the role of PknG in mycobacteria during exposure to acidic environment. Expression of pknG in MS was decreased in acidic medium. Recombinant MS ectopically expressing pknG (MS-G) showed higher growth in acidic medium compared to wild type counterpart. MS-G also showed higher resistance upon exposure to 3.0pH and better adaptability to acidic pH. Western blot analysis showed differential threonine but not serine phosphorylation of cellular proteins in MS at acidic pH which was restored by ectopic expression of pknG in MS. In Mtb H37Ra (Mtb-Ra), expression of pknG was increased at acidic pH. We also observed decreased expression of pknG in MS during infection in macrophages while the expression of pknG in Mtb-Ra was increased in similar conditions. Taken together, our data strongly suggests that pknG regulates growth of mycobacteria in acidic environment and is differentially transcribed in MS and Mtb under these conditions.

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