BMC Infectious Diseases

EPOSTER PRESENTATION Open Access

The *PknI* and *DacB2* double deletion mutant of *Mycobacterium tuberculosis* leads to alteration of cell morphology and susceptibility to antibiotics

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From 2nd International Science Symposium on HIV and Infectious Diseases (HIV SCIENCE 2014) Chennai, India. 30 January - 1 February 2014

Background

Mycobacterium tuberculosis is a slow growing infectious pathogen. It takes twenty hours for a single cell to divide into two. Its cell division is complex involving a number of proteins. Although, the physiological roles of several serine/threonine phosphorylation connected to cell division and peptidoglycan synthesis have been studied the exact mechanism is not clear. *PknI* and *DacB2* located in a same cluster have been shown to play a role in cell division and cell wall synthesis. The aim of this present study was to construct the double deletion mutant (DKO) of *PknI* and *DacB2* and study the effect on cell morphology and antibiotic susceptibility.

Methods

Specialized phage transduction method was used to construct DKO strain of *PknI* and *DacB2*. The cell morphology was observed in solid agar plate, light microscopy and electron microscopy. The MIC was determined by resazurin based microplate assay.

Results

The DKO was confirmed by PCR and southern blotting methods. The light and electron microscopy study revealed that DKO showed irregular shape and smoother colonies in comparison to *M. tuberculosis* H37Rv. The DKO was more susceptible to isoniazid compared to *M. tuberculosis* H37Rv and DKO showed the same sensitivity pattern as H37Rv strain to other drugs.

Conclusion

In the present study, we have successfully constructed a novel DKO strain of *Mycobacterium tuberculosis*. *PknI* and *DacB2* were found to have a role in maintaining cell morphology and isoniazid resistance.

Published: 27 May 2014

doi:10.1186/1471-2334-14-S3-E22
Cite this article as: Kandasamy and Narayanan: The *PknI* and *DacB2* double deletion mutant of *Mycobacterium tuberculosis* leads to alteration of cell morphology and susceptibility to antibiotics. *BMC Infectious Diseases* 2014 **14**(Suppl 3):E22.

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