Characterizing the Role of CpsA in Mycobacterial Pathogenesis

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Characterizing the Role of CpsA in Mycobacterial Pathogenesis

**Mycobacterium tuberculosis**

**CpsA**

- **What is M. tuberculosis?**
  - Causative agent of tuberculosis
  - Infects 1/3 of the world’s population
  - 1.5 million deaths worldwide in 2014
  - M. tuberculosis survives in macrophages by arresting phagosome maturation and altering cellular trafficking.

- **What is CpsA?**
  - Virulence factor secreted by M. tuberculosis
  - Member of the LytR-CpsA-Psr (LCP) family of proteins
  - M. tuberculosis uses CpsA to alter cellular trafficking and disrupt host immunity mechanisms (see Dr. Sandeep Upadhyay Poster).

**Methods**

- Determine if the LytR domain is sufficient for CpsA binding to Ndp52.
- Determine if the LytR domain is important for virulence in M. tuberculosis, using CpsA deletion constructs.
- Investigate the mechanism by which CpsA confers enhanced intracellular survival to M. smegmatis.

**Results**

- CpsA promotes virulence in M. tuberculosis
- CpsA binds to Ndp52

- ΔcpsA M. tuberculosis grows poorly in human and murine macrophages.
- It is also required for virulence in mice (data not shown).

**Conclusions**

- When introduced into M. smegmatis, M. tuberculosis CpsA confers enhanced intracellular survival to M. smegmatis, such that 5-fold more bacteria are found 72 hours after infection.
- This data demonstrates that CpsA is an important virulence factor in M. tuberculosis.

**References**

- Center for Disease Control and Prevention, TB Data and Statistics, Sept. 2015.

**Future Steps**

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