The Expression of OmcA in Chlamydia muridarum is Modified in Response to Environmental Stress
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Introduction

GlpA: Virulence protein found to be downregulated under glucose stress in C. trachomatis but not C. muridarum.1

Hypothesis

Unlike Chlamydia trachomatis, Chlamydia muridarum fails to modulate OmcA expression in response to environmental stress.

Methods

• Seed L929 cells and infect them with C. trachomatis and C. muridarum at 1 MOI and treat with stressors: 2-deoxyglucose (2DG), penicillin, 2,2’-bipyridyl, deferoxamine, and mannitol
• Strains used in study: C. trachomatis E3024 and C. muridarum
• Live cell imaging at 18 hours post infection with groEL::mCherry and omcA::gfp
• Immunofluorescent staining at 40 hours post infection with anti-OmcB antibody to determine protein expression
• Infection forming unit assays (IFU)

Results

C. trachomatis downregulates omcA::gfp in response to multiple environmental stressors.

C. trachomatis expression in response to 24 hours post infection with CTE3024. NucBlue stained cell nuclei, groEL::mCherry is constitutively expressed while omcA::gfp is not.

C. muridarum expression of omcA::gfp is unaltered in response to 2DG.

Stress results in fewer infectious C. muridarum progeny despite unaltered omcA transcription.

Discussion

• omcA transcription is unaltered in response to glucose limitation, penicillin, iron limitation, and hyperosmolarity in C. muridarum.
• Reduced OmcB expression, generation of persistent RB, and reduced infectious progeny indicate that post-translational pathways leading to persistence4,5,6 are active in C. muridarum.
• Our previous study1 suggests that 2DG treated C. muridarum continue to be proinflammatory, indicating that virulence proteins are produced. Active recruitment of host responses in combination with low infectious yield could contribute to accelerated clearance in the murine genital tract.
• Future Investigations: determine if virulence is also modulated by environmental stress by monitoring the expression of pgp3 by qPCR and secretion of Pgp3 by immunostaining with anti-Pgp3 antibody to observe phenotypic changes in protein expression.

References


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