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

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Chlamydia trachomatis (CT) is the most common sexually transmitted bacterial infection globally. It causes pelvic inflammatory disease in genital tracts of ~10% of infected women. CT is an obligate intracellular pathogen with a biphasic developmental cycle. The outer membrane proteins OmcA and OmcB, produced from a single transcript, are only expressed in infectious elementary bodies (EBs), serving as markers of reticulate body (RB) to EB conversion. A "persistent" form is associated with failure to generate new EBs. Our lab demonstrated that genes involved in glycogen metabolism (*glgA*) and virulence (*pgp3*) are down-regulated by stress-induced conditions in CT, but not in *C. muridarum* (CM), a species that causes acute infections in the genital tracts of mice. We hypothesized that unlike CT, CM fails to modulate *omcAB* transcription in response to environmental stress. L929 cells (mouse fibroblasts) were infected with CM and treated with various stressors. CM expressing *omcA::gfp* allowed live imaging of *omcAB* transcription and staining with anti-MOMP and anti-OmcB antibodies allowed assessment of OmcB protein production. Infection forming units (IFUs) were quantified to assess RB to EB conversion. GFP was present regardless of stressor, indicating that *omcAB* transcription by was not down-regulated. However, 2-deoxyglucose (2DG) treated CM displayed reduced OmcB protein by immunostaining and fewer IFUs were recovered from 1mM, 5mM, and 10mM 2DG as well as penicillin and iron deprivation. These results indicate that post-translational pathways leading to persistence are active in CM but that *omcAB* genes are not transcriptionally regulated in CM as they are in CT. Future studies will examine whether glycogen production and Pgp3 expression are affected similarly.