Chlamydia trachomatis is an obligate intracellular pathogen with global health and economic impact. Upon infection, *C. trachomatis* resides within a protective niche, the inclusion, wherein it replicates and usurps host cell machinery and resources. The inclusion membrane is the key host-pathogen interface that governs specific protein-protein interactions to manipulate host signaling pathways. At the conclusion of the infection cycle, *C. trachomatis* exits the host cell via lysis or extrusion. Most of our research focuses on the mechanism of host cell exit, extrusion. Extrusion depends on the phosphorylation state of myosin light chain 2 (MLC2); the extent of phosphorylation is determined by the ongoing opposing activities of myosin phosphatase (MYPT1) and myosin kinase (MLCK). Previously, it was shown that MYPT1 is recruited to the inclusion and interacts with CT228 for regulation of host cell egress. Using new genetic tools we generated a targeted chromosomal mutation of *CT228* (L2-ΔCT228) using the TargeTron system and demonstrate a loss of MYPT1 recruitment and increase in extrusion production *in vitro*. We document a delay in clearance of L2-ΔCT228 during murine intravaginal infection as well as a reduction in both systemic humoral response and reproductive tract mucinous changes, relative to L2-wild type. Additional ongoing experiments are investigating the role of additional inclusion membrane proteins with various host signaling pathways that may be contributing to regulate host cell exit or Chlamydial survival strategies.

If you would like to visit with Dr. Ericka Lutter, please contact Steph Shames at sshames@ksu.edu.

Coffee & cookies served preceding the seminar in Ackert Hall, Room 225