Chlamydia genital infection is the most frequent bacterial STD across the world caused by Chlamydia trachomatis. Studies on Chlamydia muridarum infection of male mice with through pulmonary routes have been reported. Growing evidence shows chronic stress suppresses the immune that leads to infection. Stimulation of beta2-adrenergic receptor (β₂-AR) is known to create an immune response through its known ligand, norepinephrine. We have shown that norepinephrine is released when mice are exposed to cold water stress, but its interaction with β2-AR during chlamydia lung infection is not known. The purpose of this project was to determine the susceptibility of β2-AR knock out male mice to Chlamydial muridarum lung infection. We hypothesized that deficiency in β2-AR leads i) a decreased C. muridarum shedding in the lung of male mice; ii) increased gene expression and production of protective cytokines during lung infection. In our preliminary experiments, we seeded 70 µM-filtered lung lysate to monolayers of McCoy tissue culture following standard methods. After 32 h of infection and staining with fluorescein isothiocyanate-labeled, anti-chlamydial antibody. Inclusion forming units/ml were not determined because cell debris covered monolayers we were able to visualize and count inclusion bodies in monolayers. To improve results, 30 µM-filtered lung lysate will be seeded to monolayers of McCoy tissue culture following standard methods. Gene expression and production of protective cytokines during lung infection is underway. We expect that stressed and non-stressed β2-AR knockout male mice will have about the same IFUs/ml in the lungs because of the lack of β2-AR gene expression. This work was supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence and NIH Grant P20GM103434 awarded to Bluefield State College.