RAENEL CRENSHAW & TESFAYE BELAY. Bluefield State College. Decreased *Chlamydia muridarum* genital infection and improved immune response in a β2-Adrenergic Receptor Knockout Mouse Stress Model.

Chlamydia genital infection caused by *Chlamydia trachomatis* shows a high prevalence in low socioeconomic populations; however, whether stress is associated with the high prevalence of infection is not known. During chronic stress conditions, the interaction of beta2-adrenergic receptor (β2-AR) with norepinephrine (NE) is known to suppress the immune system in humans and animal models. However, the mechanism of suppression that leads to chlamydia infection is not well known. This study aims to measure the susceptibility of mice and cytokine production of a β2-AR knockout (KO) during *Chlamydia muridarum* genital infection. Stressed and non-stressed C57BL/6J wildtype (WT) and β2-AR gene KO mice will be infected with *C. muridarum* intravaginally post 21-day stressing period. Swabbing of mice at the 3-day interval for 42 days will be performed to determine *C. muridarum* count*s* and isolation. Moreover, splenic CD4+ T cells, bone marrow-derived dendritic cells at day 7 post-infection will be performed for immune response analysis by ELISA. We hypothesize that the deficiency of β2-AR leads to significantly less *C. muridarum* shedding from the genital tract of stressed KO and leads to an increase and decrease in the production of protective and suppressive cytokines, respectively. We anticipate results obtained will align with our central hypothesis that stressed WT mice have increased *C. muridarum* shedding of genital tracts and increased production of protective cytokines in stressed β2-AR KO, suggesting the importance of stress in β2-AR stimulation.