

ALEXIS RAY, Department of Applied Science, Undergraduate Studies, Bluefield State College. Bluefield, WV 24701, and TESFAYE BELAY PhD. Professor of Biology, Bluefield State College. Bluefield, WV 24701. Profile of CD8+ T-cells in a Stressed beta2- Adrenergic Receptor Knockout Mouse Model during *Chlamydia muridarum* Genital Infection.

Chlamydia trachomatis is the most common bacterial sexually transmitted disease. A negative correlation between the immune system and stress has been reported. We have demonstrated that cold-induced stress (CIS) increases the intensity of *Chlamydia muridarum* genital infection in a mouse model, but the status of CD8+ T cells is still unknown. This study profiles CD8+ T cell's interaction with stress hormone; norepinephrine (NE) and the beta2-adrenergic receptor (β 2-AR) in the presence of agonists and antagonists; and examines cytokines produced in the β 2-AR knockout (KO) stress mouse model during *C. muridarum* genital infection. We hypothesize that CD8+ T cells produce high-level IFN- γ and low-level TNF- α in stressed β 2-AR KO mice than C57BL/6J wildtype (WT). Two groups of mice were cold-water stressed for 21 days, infected with *C. muridarum*, and then sacrificed for CD8+ T cell isolation. Viability was tested. The proliferation of CD8+ T cells was carried out for 72 h and culture supernatant was collected for cytokine production using ELISA. To confirm genital tract infection, *Chlamydia muridarum* isolation is underway. Our data show that the viability of CD8+ T cells treated with NE was significantly reduced compared to cells treated with LPS or Con A, indicating the suppressive effect of NE on immune cells. Stressed β 2-AR KO mice displayed increased production of protective cytokines compared to stressed WT mice, indicating the absence of β 2-AR leads to the functional restoration of protective immune cells.