

AMBEL HARTWELL, TESFAYE BELAY, Dept. of Applied Sciences and Mathematics, Bluefield State College, Bluefield WV, 24701. Deficiency in beta2 adrenergic receptor in mice leads to decreased *Chlamydia muridarum* shedding in genital tract.

Chlamydia caused by *Chlamydia trachomatis* is the most common bacterial STD that causes pelvic inflammatory disease and infertility, especially in childbearing women. We have shown that stressing mice in cold water for 5 minutes daily for 21 days leads to increased production of stress hormones. It is known that beta2 adrenergic receptor (β 2-AR) as a ligand to norepinephrine hormone is shown to regulate immune function in mice. However, the effect and mechanism of β 2-AR on chlamydia infection is not well known. The purpose of the study was to examine the susceptibility of a β 2-AR knock out model to *Chlamydia muridarum* genital infection. We hypothesized that deficiency in β 2-AR leads i) a decreased *C. muridarum* shedding from the genital tract of stressed mice; ii) increased gene expression of protective cytokines in the genital tract during infection. Mouse cervico-vaginal swabbing at 3-day intervals for 28 days was performed. *C. muridarum* was isolated from genital swabs in McCoy tissue culture according to standard methods, and inclusion bodies were visualized and counted after staining with fluorescein isothiocyanate-labeled, anti-chlamydial antibody. Quantitative real time PCR and ELISA methods, respectively, were used to measure cytokine mRNA expressions and secretions from genital tract lysates. Results showed no significance differences between stressed and non-stressed of IFU/ml. Moreover, no significant gene expression of β -AR subsets in stressed and non-stressed mice was observed. Interestingly, up-regulation of interferon gamma was observed in stressed compared to non-stressed suggesting that mice lacking β 2 adrenergic receptor have reverted to protective cytokine production to decrease infection. *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.*