

Production of CD4+ T Cell Subsets in a Beta2-Adrenergic Receptor Knockout Stress Mouse Model during *Chlamydia muridarum* Genital Infection Kaitlyn Cook*, Tesfaye Belay, Ph.D*, *Department of Applied Science and Mathematics, Bluefield State College, Bluefield WV 24701

Chlamydia is the most widely spread sexually transmitted disease, with more than one hundred million new cases occurring every year worldwide. Studies show the effect of stress promoting infection, but little is known about Chlamydia and the negative impact of stress. We use cold-induced stress (CIS) to simulate stress in mice to understand the relationship between the stress hormone norepinephrine (NE) and the immune system. This study aims to determine the role of beta2-adrenergic receptor (β 2-AR) under stressful conditions during infection. We hypothesize that the β 2-AR suppresses T helper cell 1 (Th1) and promotes the production of Th2 cytokines. After stressing, infection, and sacrificing of mice, the spleen, lymph node, and genital tract proliferated for 72 hours. ELISA data for IL-13 concentration shows stressed knockout mice had 273.6pg/mL compared to stressed wildtype mice with 120.6pg/mL. Treatment of CD4+ T cells with chemicals resulted in production of TNF- α , ranging from 595pg/mL to 398.7pg/mL. Viability of CD4+ T cells treated with β 2-AR agonist Fenoterol, and antagonist ICI 118 551 were analyzed. The viability of CD4+ T cells treated with Norepinephrine was 70.7% in knockout (KO), compared to 69.1% and 70.2% for the agonist and antagonist, respectively. Norepinephrine, Fenoterol and ICI treatment resulted in 72.2%, 83.6% and 84.4% respectively for wildtype (WT). No significant difference is found between the agonist and antagonist. *Chlamydia muridarum* isolation from the genital tract of treatment groups is underway. Our ELISA data implies that β 2-AR mice restored increased production of protective cytokines more than their WT counterparts.