

Analysis of immune response of stressed male mice during *Chlamydia muridarum* lung infection. *Nathasha Woart, Tesfaye Belay. Dept of Applied Sciences and Mathematics. Sch of STEM. Bluefield, WV*

Abstract

Chlamydia lung infection caused by *Chlamydia trachomatis* is a serious lung infection particularly in infants but there are a few studies about lung infection. Chlamydial genital and lung infections in mice is frequently done with *Chlamydia muridarum*. The purpose of our study was to explore the effect of stress in lung chlamydia infection in male mice. We hypothesize that stress changes the level of cytokine production in monocytes during lung infection. As a result from these studies include counting of live cells, differentiation of proliferation of bone marrow derived monocytes where the result showed that non-stressed dendritic cells (DCs) WT mice and stressed macrophages (MO) WT mice had sufficient live cells to study. No difference in IL- β production of macrophages of stressed and non-stress mice was observed. Beta2-adrenergic receptor (β 2-AR agonist (fenoterol) and antagonist (ICI 118 551) treatment resulted in difference in cytokine production the effect of agonists and antagonists. TNF-alpha production was high in stressed mice compared to non-stress mice for MO. LPS stimulated TNF-alpha production in DC but showed no difference in stressed and non-stressed mice. However, TNF-alpha production in macrophages of non-stressed mice was decreased. The production of IFN- γ , in Con A- and LPS-treated splenic T cells. In contrast IL-5, IL-10, and IL-23 production was high in T cells of stressed mice. T cells treated with norepinephrine and bet2-adrenergic receptor agonist, Fenoterol. Overall, the production of cytokines in stressed and non-stressed mice shows variation that may have roles in enhancing protection or increased lung infection. (*Supported by the pilot CNPR of WV-INBRE and BSU*)