GAVIN TANNER LILLY & TESFAYE BELAY, Bluefield State College, Characterizing Bone Marrow-derived Monocytes in a Stressed beta2 Adrenergic Receptor Knockout Male Mice during *Chlamydia muridarum* Lung Infection

Several reports in the literature show that stress leads to increased susceptibility to infection, but relationship of stress and chlamydia remains to be explored. Monocytes play a significant role in protective immunity against chlamydia genital infections; however, the effect of stress on the function of monocytes is not known. During chronic stress conditions, the beta2 adrenergic receptor (b2-AR), as a ligand to norepinephrine (NE) hormone, shows to regulate immune function in the host. However, the effect and mechanism of b2-AR on chlamydia lung infection is not well known. The purpose of the study was to examine the susceptibility of a b2-AR knockout (KO) mouse model to *Chlamydia muridarum* lung infection; determine the function of bone marrow-derived dendritic cells (BMDMs). We hypothesize that deficiency in b2-AR leads to a decreased *C. muridarum* shedding from the lung of stressed b2-AR KO mice compared to stressed to wildtype (WT), C57BL/6J. After stressing mice for 21 days, mice will be infected intranasally. *C. muridarum* will be isolated from lung lysates in McCoy tissue culture, and inclusion bodies will be visualized and counted after staining with fluorescein isothiocyanate-labeled, anti-chlamydial antibody. BMDMs cells will be isolated, differentiated, and increased to determine proinflammatory cytokine production by ELISA. Results will show significant differences between stressed b2-AR KO and WT stressed of IFU/ml. Moreover, increased proinflammatory cytokine production will be observed in BMDMs stressed b2-AR KO compared to stressed WT, suggesting that mice lacking b2 AR have reverted to protective cytokine production to decrease infection.