KATELYN GOINS & TESFAYE BELAY. Dept of Applied Science, Bluefield State College, Bluefield WV, 24701.The Role of bone marrow-derived dendritic Cells in a stressed beta2 adrenergic receptor knockout male mice during *Chlamydia muridarum* lung infection.

Recent studies have shown that *Chlamydia muridarum* causes a lung infection that resembles pneumonia of C. *trachomatis* in humans. However, the mechanism(s) of the infection is/are not well defined. Dendritic cells (DCs) play a significant role in protection against Chlamydia genital infections, but their role during stressful conditions is unknown. This study is to examine the role of bone marrow-derived dendritic cells (BMDCs) in beta2-adrenergic receptor (β2-AR) knockout (KO) male mice during *C. muridarum* lung infection. We hypothesized that the lack of β2-AR leads to decreased organ load of *C. muridarum* in mice lung infection. We also hypothesized that BMDCS from β2-AR KO mice restored the normal function of cytokine production and served as an antigen-presenting activity to CD4+T cells. Stressed and non-stressed C57BL/6J wildtype (WT) and β2-AR gene knockout mice will be infected. Lysates of lungs will be tested for isolation of *C. muridarum.* Cells will be for Differentiation and proliferation of BMDCs will be performed to detect cytokine production. Results should correspond to our working hypothesis, which includes the lack of (β2-AR) leads to decreased organ load of *C. muridarum* in lung infection. Results will show significant differences between stressed β2-AR KO and stressed of WT. Moreover, increased protective cytokine production in stressed β2-AR KO compared to decreased cytokines production in stressed WT will be observed. Our findings may suggest that mice lacking β2 AR have reverted to protective cytokine production to reduce infection, and the function of BMDCs as antigen-presenting cells will be restored in KO mice.