Several reports in the literature show that stress leads to increased susceptibility to infection, but the 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 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.