

Roles of dendritic cells and macrophages during *Chlamydia muridarum* genital infection in stressed mice. Danielle Baker, Tesfaye Belay. Dept of Applied Science and mathematics. Sch. Of STEM. Bluefield, WV.

Abstract

It is well known that stress has a significant impact on public health and more evidence shows that stressors generally are greater in populations of lower socioeconomic status. Cold-induced stress inhibits immune cell activity and has been shown to cause different expressions of cytokine profiles during *Chlamydia muridarum* genital infection. Dendritic cells (DCs) and macrophages (MØ) are immune cells that express beta2-adrenergic receptor (β 2-AR) and with key roles during chlamydia genital infection. This study is aimed to investigate the role of β 2-AR by using a β 2-AR knockout mouse. Wildtype (WT) and β 2-AR knockout (KO) mice were stressed for five minutes daily and infected with *C. muridarum* intravaginally. Non-stressed infected mice of the groups were used as controls. Bone-marrow-derived DCs and ϕ were tested isolated, counted differentiated, and proliferated for cytokine production. The proliferation of DCs and macrophages in the presence/absence of β 2-AR agonists and antagonists was tested. Data show that non-stressed mice had a higher production of cytokines than stressed mice. Increased production of TNF- α in LPS-treated DCs and MØ of WT and β 2-AR KO was observed. The effect of fenoterol and ICI118,55 antagonist showed no significant difference in cytokine production. The data indicate that β 2-AR KO and WT had a similar pattern of cytokine production suggesting that deficiency in β 2-AR restores the function of immune cells during genital infection. Experiments are undergoing to fully understand the mechanisms involved in modulating the function of DC and MØ during chlamydia genital infection. (Supported by McNair Scholarship of Concord University and Initiation Grant of NASA WV Space Consortium, WV-INBRE and BSU).