Cold-induced stress alters the dynamics of T helper 1 and T helper 2 cytokine production in a mouse model during Chlamydia muridarum genital infection.

Chlamydia caused by Chlamydia trachomatis is the leading cause of bacterial STDs worldwide and the USA. Although there are more clinical and epidemiological studies of chlamydia genital infection, the effect of stress on chlamydia disease is not well explored. Previous results from our lab show that cold-induced stress results in increased intensity of chlamydia genital infection in mice; however, the mechanisms are unknown. Thelper 1(Th1) is known to be protective against chlamydia, whereas Th2 leads to infection. However, the differentiation of Th1 and Th2 during exposure to cold stress is not well understood. Several studies show GATA-3 and T-bet are associated with Th1 and Th2 cytokine production. The purpose of this study was to evaluate the effect of cold water on the activity of Th1 and Th2 during Chlamydia muridarum genital infection. We hypothesized that cold stress alters the differentiation and variation in gene expression profile of transcription factors and cytokine productions in T cells. T cells isolated from the genital tract were proliferated for 48 h incubated at 37°C and 5% CO2. The mRNA levels and detection of cytokines in culture supernatants was determined by quantitative real time PCR and ELISA, respectively. Our results showed increased IL-4 production, which was associated with upregulated expression of transcription factor GATA-3. In contrast, decreased gamma interferon production was associated with decreased T-bet gene expression in T cells. Based on this data, cold stress modulates the dynamics of signature Th1 and Th2 cytokine productions that are regulated by expression of GATA-3 and T-bet. This work was supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence and NIH Grant P20GM103434 awarded to Bluefield State College.