Chemokine response induced by Chlamydia trachomatis in prostate derived CD45+ and CD45- cells.

Abstract:
The role of innate cells and their receptors within the male genital tract remains poorly understood. Much less is known about the relative contribution of different genital tract cells such as epithelial/stromal cells and resident leucocytes. In this study, we examined innate immune responses to Chlamydia trachomatis by prostate epithelial/stromal cells and prostate resident leucocytes. Murine prostate primary cultures were performed and leucocyte and epithelial/stromal cells were sorted based on surface protein expression of CD45 by magnetism-activated cell sorting or fluorescence-activated cell sorting. Prostate derived CD45- and CD45+ cells were infected with C. trachomatis and chemokine secretion assayed by ELISA. Similar experiments were performed using prostate CD45+ and CD45- cells from myeloid differentiation factor 88 (Myd88(-/-)) mice or toll-like receptor (Tlr2(-/-)) and Tlr4(mutant) double-deficient mice. Moreover, a TLR-signalling pathway array was used to screen changes in different genes involved in TLR-signalling pathways by real-time PCR. Prostate derived CD45- and CD45+ cells responded to chlamydial infection with the production of different chemokines. Both populations expressed genes involved in TLR signalling and required to respond to pathogen-associated molecular patterns and to C. trachomatis.
infection. Both populations required the adaptor molecule MYD88 to elicit chemokine response against C. trachomatis. TLR2-TLR4 was essential for chemokine production by CD45+ prostate derived cells, but in their absence, CD45- cells still produced significant levels of chemokines. We demonstrate that C. trachomatis is differentially recognised by prostate derived CD45+ and CD45- cells and suggest that diverse strategies are taking place in the local microenvironment of the host in response to the infection.