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Th2 and IL-17 responses in Chlamydia pneumoniae-stimulated PBMC in asthmatic and non-asthmatic subjects

Rationale: Chlamydia pneumoniae (C. pneumoniae) is a gram-negative intracellular bacterium that causes respiratory infections in humans, including asthmatic and non-asthmatic subjects. C. pneumoniae activates cells in vitro and produces cytokines that may contribute to the inflammatory responses observed in asthma. Different asthma endotypes are described, including Th2-high and Th2-low. Th2-low endotypes are characterized by IL-17 production and neutrophilic inflammation. The aim of this study was to investigate the role of C. pneumoniae in regulating Th2 versus Th17 responses in peripheral blood mononuclear (PBMC) from subjects with or without asthma.

Methods: PBM) (1×106/mL) from asthmatic (N=6) and non-asthmatic subjects (N=14) were infected +/- C. pneumoniae TW-183 at a multiplicity of infection (MOI) = 0.1, using dose responses (1:10, 1:100), and cultured 48 hrs. Cytokine responses (Interferon (IFN)-gamma, Interleukin (IL)-2, IL-4, IL-17 A/F) were measured in supernatants (ELISA).

Results: Comparison of cytokine responses (the mean differences in non-asthmatic versus asthmatic subjects) were significant for IFN gamma (unstimulated; $P < 0.0001$), IL-2 (unstimulated and 1:100; $P < 0.0001$, $P = 0.0002$, respectively), IL-4 (unstimulated, 1:10, 1:100; $P = 0.0001$, $P < 0.0001$, $P < 0.0001$, respectively) and IL-17 A/F (unstimulated, $P < 0.0001$) (Wilcoxon signed-rank test). Cytokine levels were higher in asthmatic subjects for IFN-gamma (unstimulated,1:10, 1:100), IL-2 (unstimulated), and IL-17 A/F (unstimulated) compared with non-asthmatic subjects. However, IL-4 levels were higher in non-asthmatics (unstimulated, 1:10, 1:100).

Conclusions: Differences in Th2 and IL-17 cytokines responses in PBMC from subjects with and without asthma may indicate the involvement of cell-mediated immunity.