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## Natural regulatory T cells in some parasitic diseases

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Parasitic infection in human alimentary tract causes a significant change in immune system through its continuous antigens secretion. The aim of this study was to estimate the change in natural regulatory T cell population in peripheral blood of patients infected with different types of alimentary tract parasites. Regulatory T cells (CD4+CD25+Foxp3+) were detected in eighty patients infected with intestinal parasites and forty healthy volunteers using flow cytometry technique. Statistical analysis showed a significant increase in regulatory T cell percentages in infected patients compared to healthy group ($P<0.001$). Patients infested with Giardia showed significantly higher CD4+CD25+Foxp3+ cell percentages than those infested with other parasites ($P<0.001$). Also, mixed infestation showed significantly higher CD4+CD25+Foxp3+ cell percentages than single infestation. In conclusion, natural regulatory T cell frequencies (CD4+CD25+Foxp3+) increase significantly in patients with parasitic diseases compared to healthy controls. The higher levels were associated with mixed infection compared to single infection and in older than younger patients.

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## PD-L1 regulation by PspA antigen during *S. pneumoniae* infection

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Pneumonia is a major cause of mortality in children under the age of five worldwide resulting in close to 20 percent of all deaths in this age group. Consequently, investigations into the host-pathogen interactions during *S. pneumoniae* infection are keys to devising strategies towards the development of better vaccines and drugs. To that end in this study it was investigated the role of *S. pneumoniae* and its antigen pneumococcal surface protein A (PspA) in modulating the expression of the co-inhibitory molecule Programmed Death Ligand -1 (PD-L1) on bone marrow derived dendritic cells (DCs) and the subsequent effects of increased PD-L1 levels on immune responses. PD-L1 is a co-stimulatory molecule that largely induces suppressor responses and causes T cell inactivation and anergy. Obtained data indicates that stimulation of DCs with PspA increases the surface expression of PDL1 in a time and dose dependent manner. Characterization of intracellular signalling molecules indicate that PspA induced expression of PDL1 was dependent on MAPK pathway and routes of calcium influx. While calcium release from intracellular stores positively regulated PDL1 expression, calcium influx from external milieu negatively regulated PDL1 expression. Knockdown of intermediates in the TLR pathway showed that the expression of PDL1 was dependent on MyD88. Knockdown of PD-L1 promoted apoptosis of DCs and increased autophagic responses together with increase in the levels of pro-inflammatory cytokines like IL-12 and IL-6. These results indicate that increased expression of PD-L1 mediated by PspA could be an immune evasion strategy adopted by *S. pneumoniae* to establish a long term infection. Further characterization of the responses is underway.

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