Decreased FOXP3 protein expression in patients with asthma.

T-regulatory cells (T(reg)) are important in balancing immune responses and maintaining peripheral tolerance. Current evidence suggests that asthma is characterized by a relative deficiency in T(reg), allowing T helper 2 cells to expand. In this study, we aimed to evaluate circulating T(reg), defined by the protein FOXP3, in both control subjects and patients with stable asthma. Peripheral blood mononuclear cells (PBMC) of control (n = 14) and asthmatic patients (n = 29) were labeled for CD4, CD25, and intracellular FOXP3 and analyzed using flow cytometry. In CD3/CD28 stimulated PBMC, the effects of dexamethasone on the transcription factors T-bet, GATA-3, FOXP3, and RORc2 and representative cytokines were studied. In control subjects and asthmatic patients, numbers of peripheral blood CD4(+)/CD25(high) and CD4(+)/CD25(high)/FOXP3(+) T-cells were similar. However, FOXP3 protein expression within CD4(+)/CD25(high) T-cells was significantly decreased in asthmatic patients. There was a tendency for increased FOXP3 expression within CD4(+)/CD25(high) T-cells in glucocorticosteroid-treated patients when compared to steroid-naive asthmatic patients. In stimulated PBMC, dexamethasone treatment increased the anti-/proinflammatory transcription ratios of FOXP3/GATA-3, FOXP3/T-bet, and FOXP3/RORc2. Asthmatic patients have decreased FOXP3 protein expression.
expression within their CD4(+)CD25(high) T(reg). Our findings also suggest that treatment with inhaled glucocorticosteroids in asthmatics might increase this FOXP3 protein expression within the CD4(+)CD25(high) T-cell population.