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**Title:** Transcriptional characteristics of CD4 T cells in young Tunisian asthmatic children: RORC and FOXP3 axis

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**Body:** Asthma is a chronic inflammatory disorder, hypothetically caused by autoreactive Th2 cells, whereas Th1 and regulatory T cells may confer protection. The development of Th subpopulations is dependent on the expression of lineage-specific transcription factors. This study aimed to assess the balance of CD4(+) T cell populations in asthmatic children. Peripheral blood mononuclear cells mRNA expression was assessed in 30 asthmatic children. Real-time polymerase chain reaction quantified TBX21, GATA-3, RORC, FOXP3, and EBI3 mRNA expression. Intracellular cytokine expression of IL-2, IL-4, IL-10, and IFN-γ in CD4(+) T cells was measured by flow cytometry. IL-6 and IL-17 cytokines were assessed in serum by enzyme-linked immunosorbent assay. A significant increase was found in the percentage of CD4(+) and CD8(+) T cell-producing IL-4, IL-6, and IL-17. A decreased percentage of CD4(+) producing IFN-γ in asthmatic children was found. Expression of GATA-3 (Th2), retinoid-related orphan receptor C (RORC) (Th17), and EBI3 were increased in asthmatic patients compared to healthy controls. Expression of FOXP3 (Treg) and TBX21 (Th1) were decreased ($P < 0.0001$ and $P < 0.0001$) in asthmatic children. Young asthmatics had increased IL-4 production and low IFN-γ synthesis. The increased serum IL-17 and IL-6 levels sustained an inflammatory environment in young asthmatics. This indicates that FOXP3 and RORC mRNA expression could be associated with the sustained inflammatory process, transduced by low immune tolerance by Treg cells.