CD45, CD3, CD4, CXCR3, CCR6, HLA-DR, CD38, CD25 and CD127. Levels of 12 cytokines in plasma were measured using a cytokine kit (MACSplex Cytokine 12 Kit, human, Miltenyi Biotec Ltd., UK). Clinical data from patients were collected to determine treatment outcome.

Results: Mean level of TNFα was higher in our RA patients (6.2 pg/ml) compared to our PsA patients (4.6 pg/ml). Initial analysis of clinical data suggested that our PsA patients without psoriasis (n = 2) responded better to TNFα than those with active psoriasis (n = 4) at 3 months and 6 months after treatment, with a clear reduction in the number of tender joints (‘psoriasis positive’: 6.65 vs 1.67; ‘psoriasis negative’: 11.5 vs 0.67) and swollen joints (‘psoriasis positive’: 2 vs 6.67; ‘psoriasis negative’: 3.50 vs 0). The mean proportion of activated Th17 cells was higher in our patients with active psoriasis (5.1% of Th17 cells) than those without psoriasis (3.6% of Th17 cells). The mean level of IL-12 and INFγ was higher in the ‘psoriasis positive’ group (respectively 129.2 pg/ml and 266.1 pg/ml) than the ‘psoriasis negative’ group (respectively 52.7 pg/ml and 115 pg/ml).

Conclusion: We hypothesize that PsA patients have a lower level of TNFα because of other pro-inflammatory cytokines potentially involved in the inflammatory process. IL-12 can induce INFγ production by Th1 cells and is involved in the IL12/IL23 axis in psoriasis pathogenesis. INFγ can induce Th1 and Th17 cells expansion through myeloid dendritic cells activation. In PsA, the high levels of IL-12 and INFγ involved in psoriasis pathogenesis might be one of the reasons for a poor response to treatment, but larger sample sizes are needed to find a biomarker that could be used routinely by clinicians, and to determine if the same biomarker can also be used for other types of arthritis such as rheumatoid arthritis.

REFERENCES: