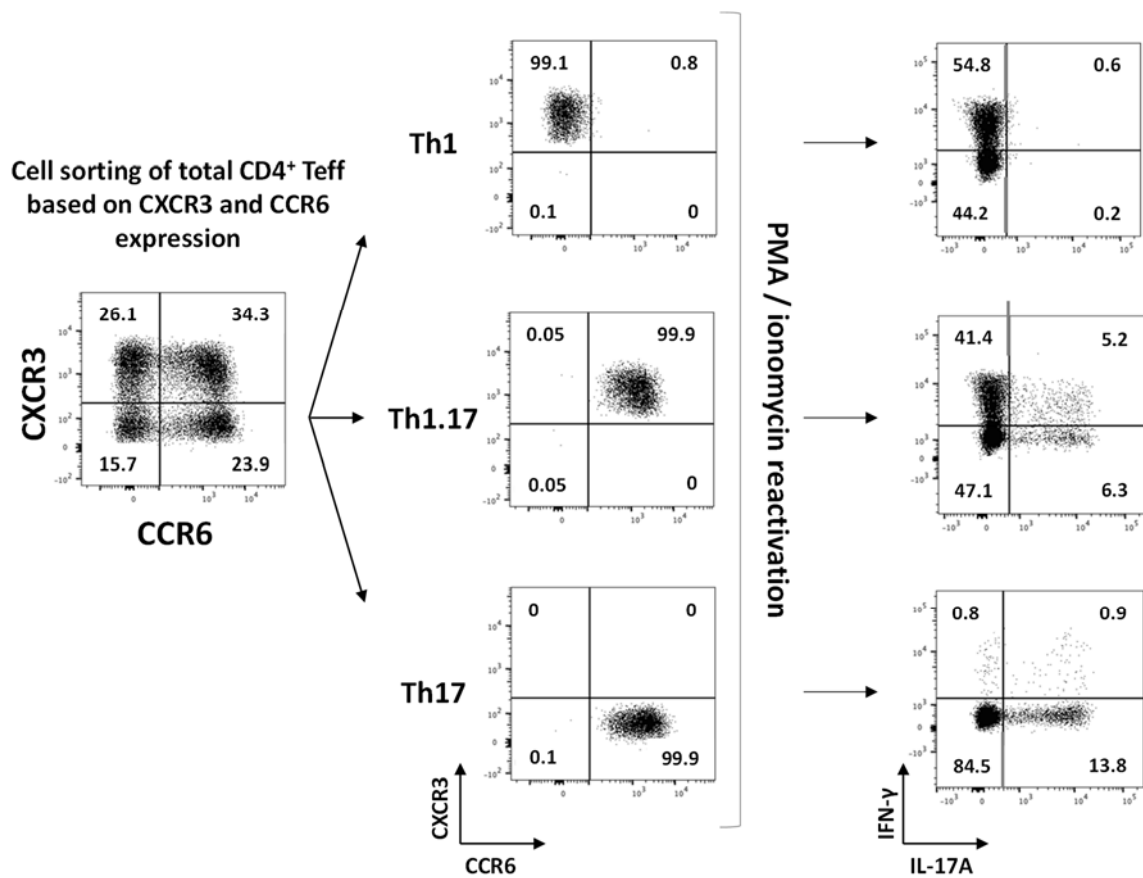
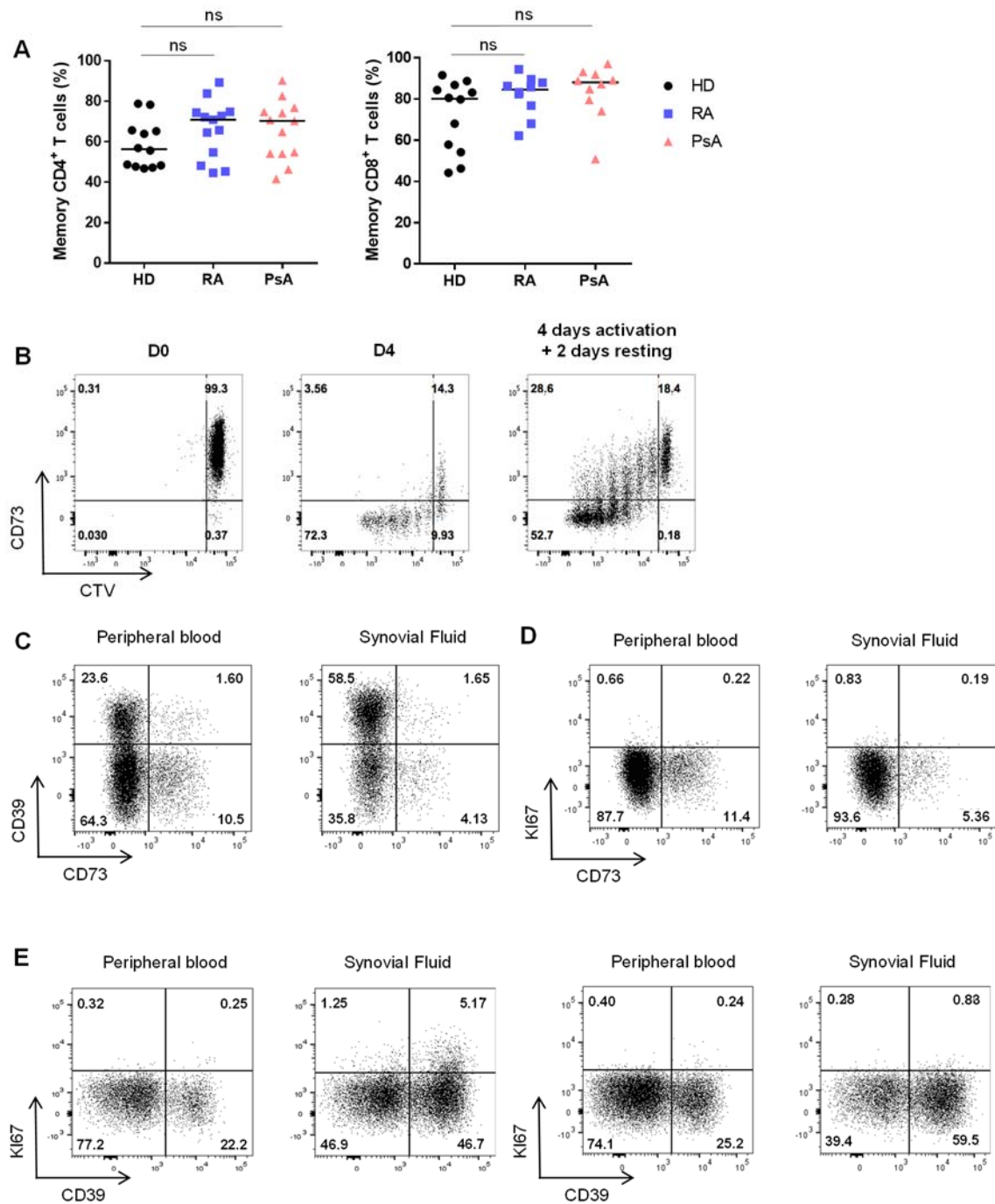


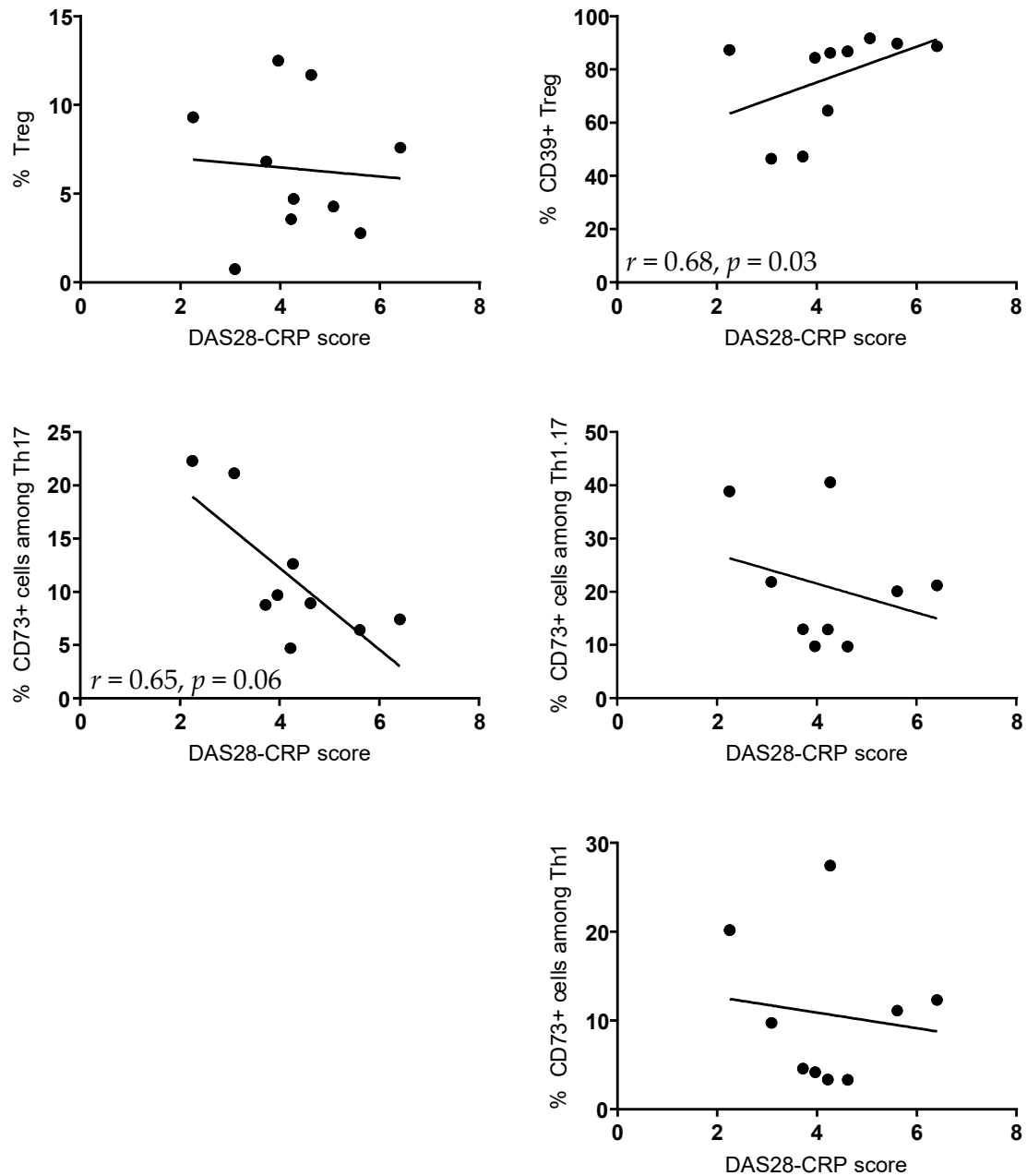
Supplementary Materials



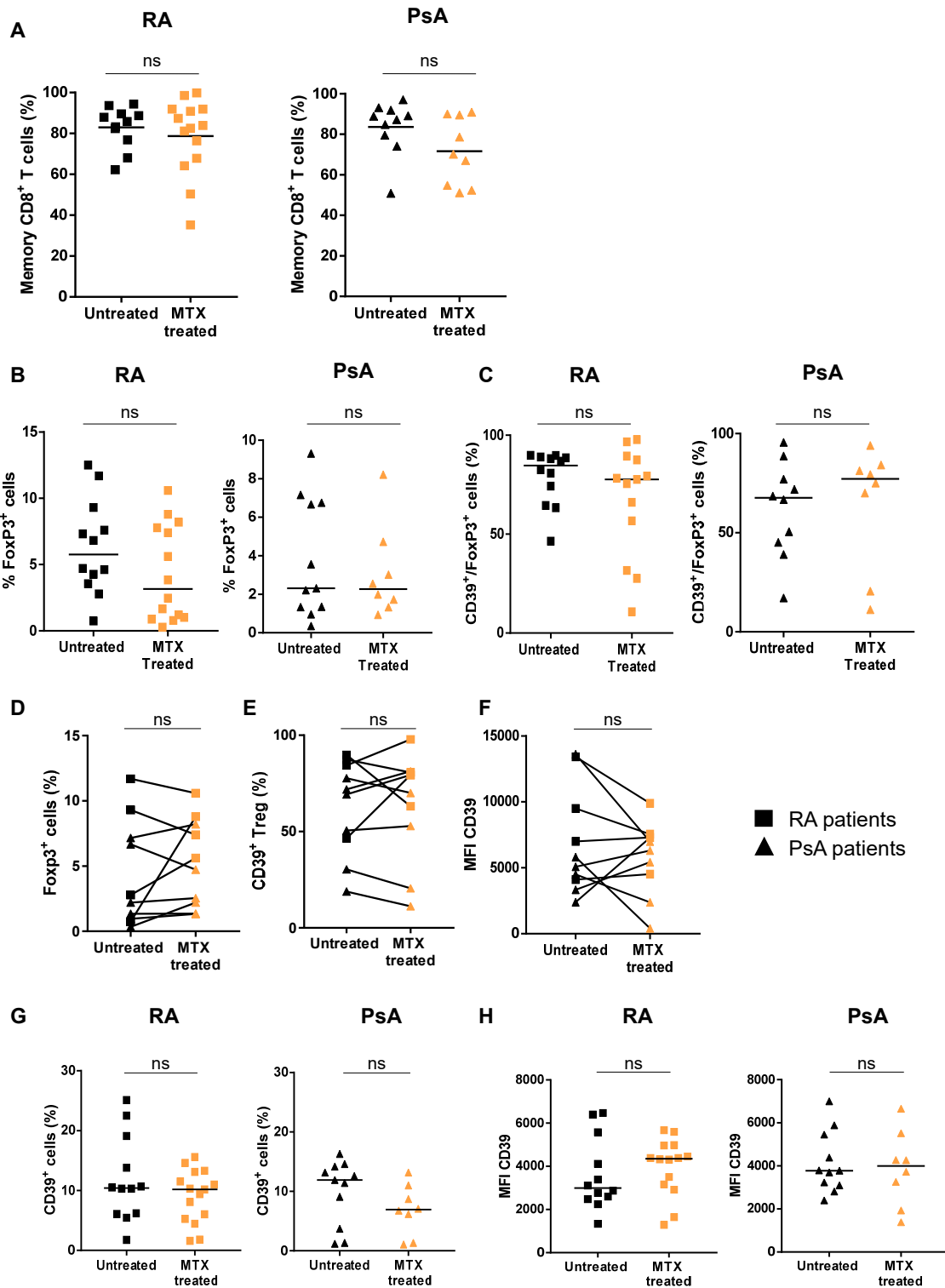
Supplementary Figure S1. Co-analysis of phenotypic and functional characteristics of Th subsets. Th1, Th1.17 and Th17 subpopulations based on CXCR3 and CCR6 expression were sorted from total memory CD4⁺ T cells and IL-17A and IFN-γ production capacity was analyzed after short term reactivation with PMA-Ionomycin.



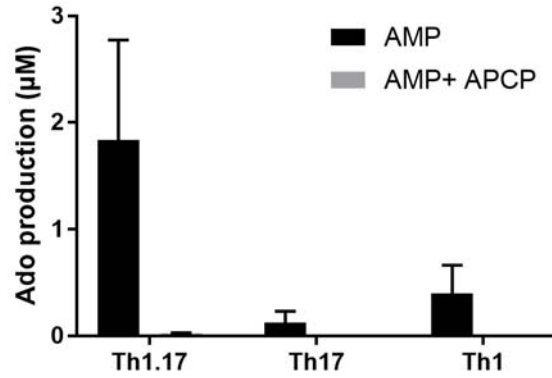
Supplementary Figure S2 The overall frequencies of memory CD4⁺ and CD8⁺ T cells are not altered in patients' blood, however the frequency of memory CD73⁺ T cells is decreased in both compartments reflecting their high level of activation. (A): Memory CD4⁺ (left) and memory CD8⁺ (right) frequencies in HD, untreated RA and PsA patients' peripheral blood. (B): CD73 dynamic of expression assessed by FC on sorted CD73⁺ blood Teff stimulated in vitro with anti-CD3/anti-CD28 coated beads for 96h. (C,D): CD73/CD39 (C) and CD73/Ki67 (D) staining on total Teff in peripheral blood and synovial fluid of a PsA patient before initiation of treatment. (E,F): Ki67/CD39 staining on total Teff from peripheral blood or SF of RA (E) and PsA (F) patients before initiation of treatment. Statistics A Kruskal-Wallis test.



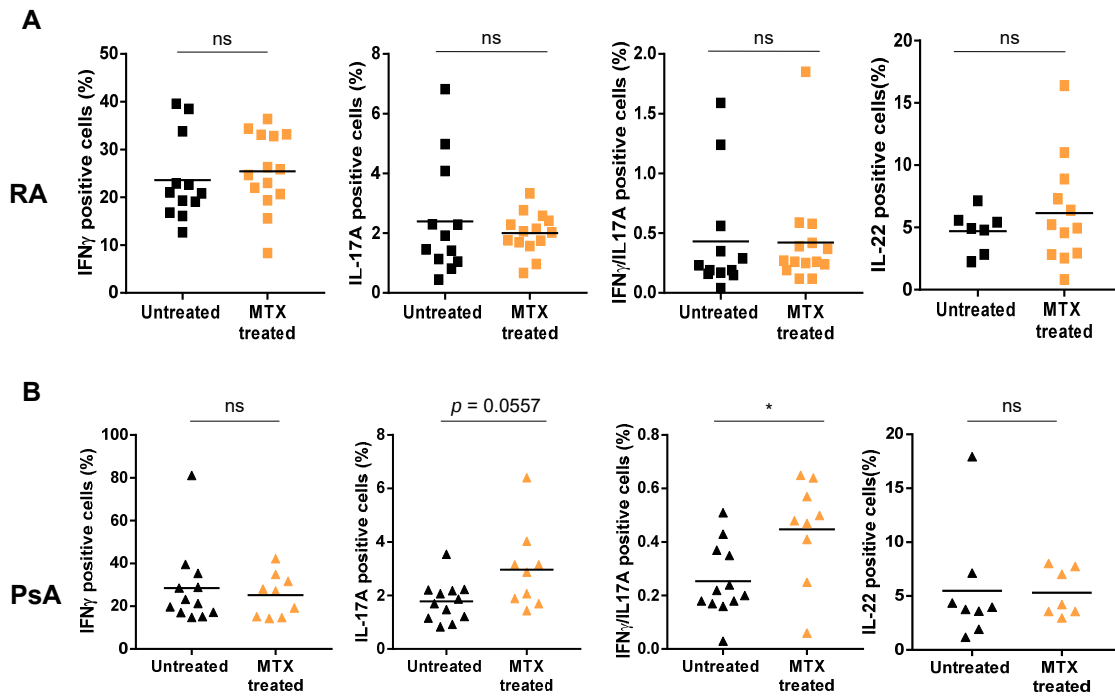
Supplementary Figure S3. Correlation between proportion of CD39 among Treg, and CD73 on Th1, Th17 and Th1.17 and clinical disease activity (DAS28-CRP) in untreated RA patients. Correlations were calculated using Spearman's test.



Supplementary Figure S5. MTX treatment induces a slight decrease of memory CD8⁺ T cells frequency in PsA patients and does not modify Treg proportion and activation status in RA and PsA patients. **A:** Memory CD8⁺ T cells frequencies in untreated vs MTX-treated RA (left) and PsA (right) patients. **B:** Frequencies of FoxP3⁺ Treg in untreated vs. MTX-treated RA (left) and PsA (right) patients. **C:** Frequencies of CD39⁺ cells among Treg in untreated vs. MTX-treated RA (left) and PsA (right) patients. **D–F:** Frequencies of FoxP3⁺ cells (**D**), CD39⁺ Treg (**E**) and MFI of CD39 on Treg (**F**) in paired samples of untreated vs. MTX-treated RA and PsA patients. **G,H:** Frequencies (**G**) and MFI (**H**) of CD39⁺ Teff in RA or PsA patients before or under MTX treatment. **A–C** and **G,H:** Mann-Whitney test, **D–F:** Wilcoxon test.



Supplementary Figure S6. Th1.17 cells are the major producers of Ado among Teff in a CD73 dependent manner. Ado produced by sorted Th1.17, Th17 and Th1 subsets from Healthy donors ($n = 3$). Cells were incubated for 2 hours with AMP^{13C15N} isotope (37.5 μ M) +/- APCP (50 μ M) before Ado quantification in supernatants by LC-MS/MS.



Supplementary Figure S7. MTX Moderately increases of IL-17A secretion by Teff in PsA but not in RA patients. Analysis of IFN- γ , IL-17A, IL-22 producing and IFN- γ /IL-17A co-producing Teff in RA (A) and PsA (B) patients before treatment initiation and under MTX treatment. * $p < 0.05$, assessed by Mann-Whitney test.

Supplementary Table S1. Panels of antibodies used for multi-parametric flow cytometry analysis throughout the study.

Panel A	Panel B	Panel C	Panel D
CD3 (UCHT1) BD Biosciences	CD3 (UCHT1) BD Biosciences	CD3 (UCHT1) BD Biosciences	CD45RA (2H4LDH11LDB9) Beckman Coulter
CCR7 (150503) BD Biosciences	CD45RA (HI100) Biolegend	CD4 (RPA-T4) Life Technologies	CD127 (M21) Life Technologies
CD45RA (HI100) Biolegend	CCR6 (11A9) BD Biosciences	CD45RA (HI100) Biolegend	CD25 (2A3) BD Biosciences
CD8 (RPA-T8) Beckman Coulter	CXCR3 (REA232) Miltenyi-Biotec	IL-17A (N49-653) BD Biosciences	CCR6 (11A9) BD Biosciences
CD4 (RPA-T4) Life Technologies	CD4 (RPA-T4) Life Technologies	TNF- α (MAb11) BD Biosciences	CXCR3 (REA232) Miltenyi-Biotec
CD39 (A1) Life Technologies	CD39 (A1) Life Technologies	IFN- γ (4S. B3), Biolegend	DAPI Life Technologies
CD73 (AD2) Life Technologies	CD73 (AD2) Life Technologies	IL-22 (22URTI) Life Technologies	
FoxP3 (PCH01) Life Technologies	FoxP3 (PCH01) Life Technologies	CD73 (AD2) Life Technologies	