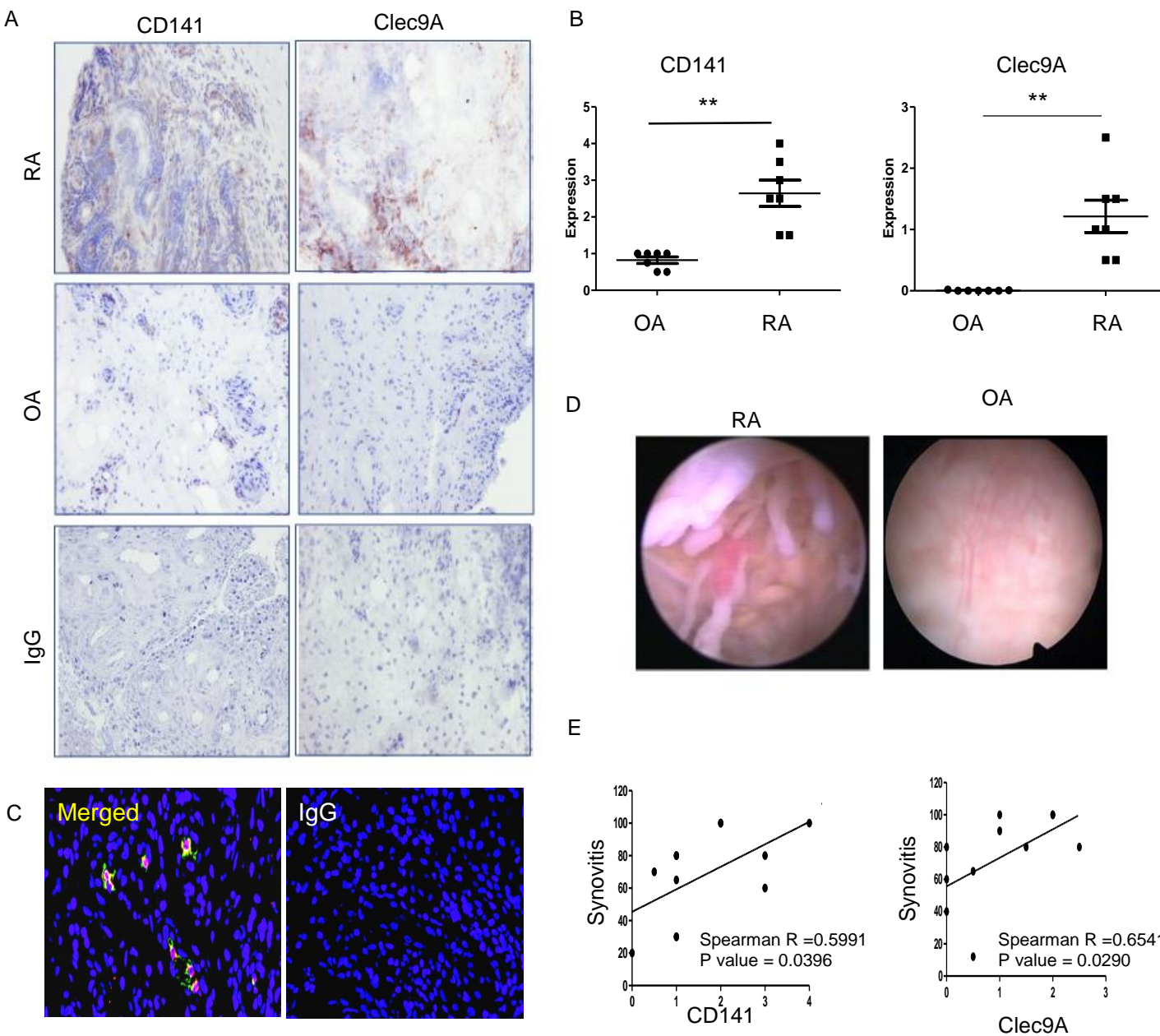
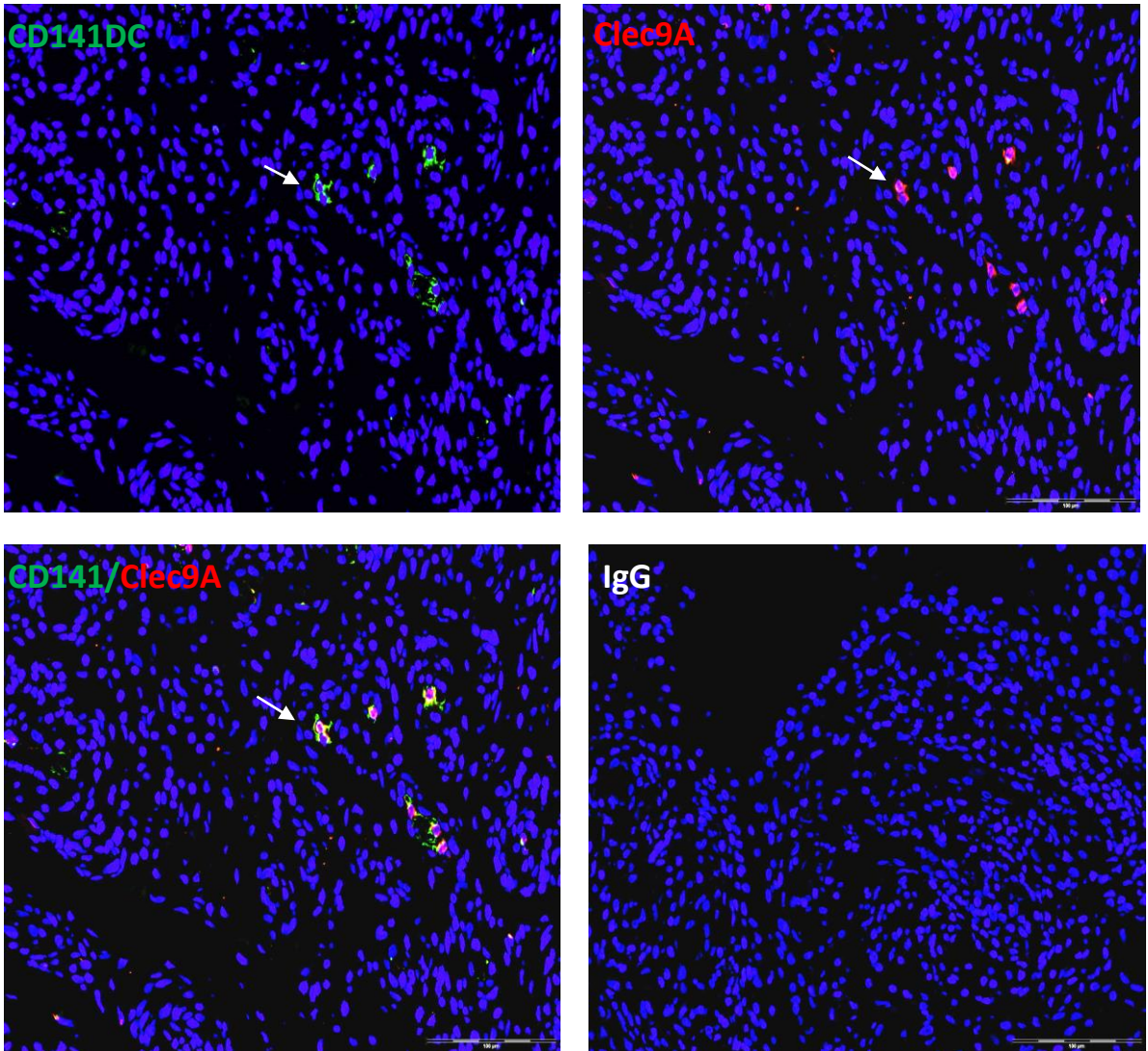


**Supplementary Figure 1:** (A) The percentage of CD141<sup>+</sup> mDC and CD1c<sup>+</sup> mDC in healthy control (HC) and inflammatory arthritis (IA) and PBMC as a percentage of Lineage<sup>-</sup> HLADR<sup>+</sup> cells (n=6) and (B) the absolute numbers of these cells relative to beads. \*P<0.05 significantly different to control.

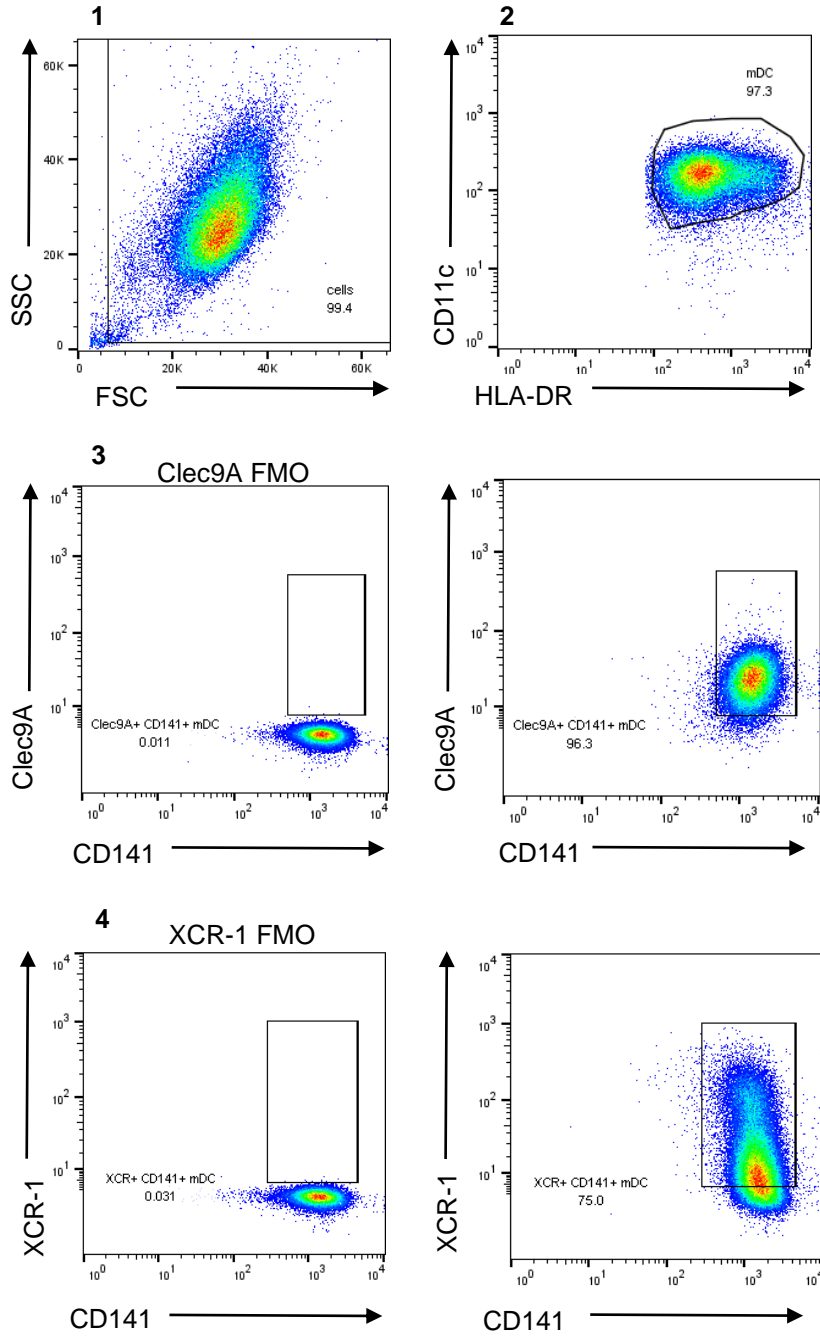


**Supplementary Figure 2:** (A) and (B)) Representative images and semi-quantification of immunohistochemical staining for CD141 and Clec9A, in synovial tissue from RA and OA patients (n=7) (Original magnification 20x). **Haematoxylin staining was used for counterstaining.** (C) Representative immunofluorescence images of CD141 Clec9A dual staining in RA synovial tissue. Nuclei are stained with DAPI (blue), CD141 is stained with Alexa-Fluor-488-conjugated goat anti-mouse superclonal™ secondary antibody (red), Clec9A is stained with Cy™3-conjugated goat anti-rabbit secondary antibody (green) and colocalisation of CD141<sup>+</sup> Clec9A<sup>+</sup> cells is depicted in yellow, original magnification of photomicrographs x 20. (D) Representative images of *in vivo* images of macroscopic synovitis from an RA and OA patient. (E) Correlation between CD141 expression or Clec9A expression and macroscopic synovitis. \*P<0.05, \*\*P<0.01 significantly different to control.

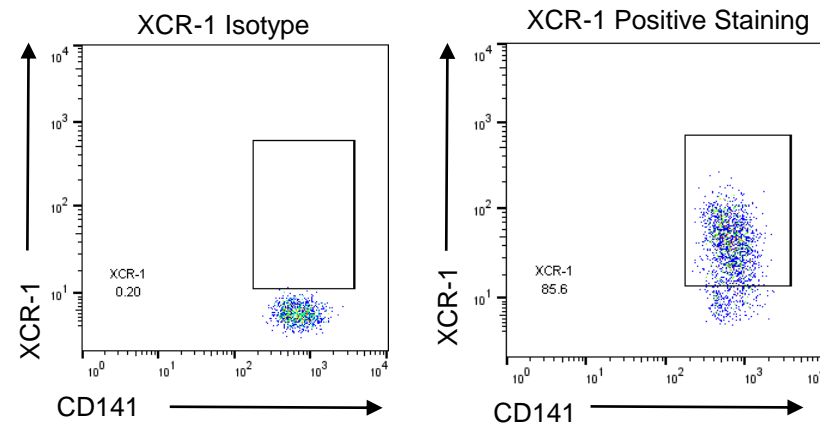


**Supplementary Figure 3.** Immunofluorescence staining for CD141 (green), cle9A (red), merged image of CD141/Cle9A (yellow) and IgG control. Nuclei were counterstained with DAPI. Original magnification 20X.

A

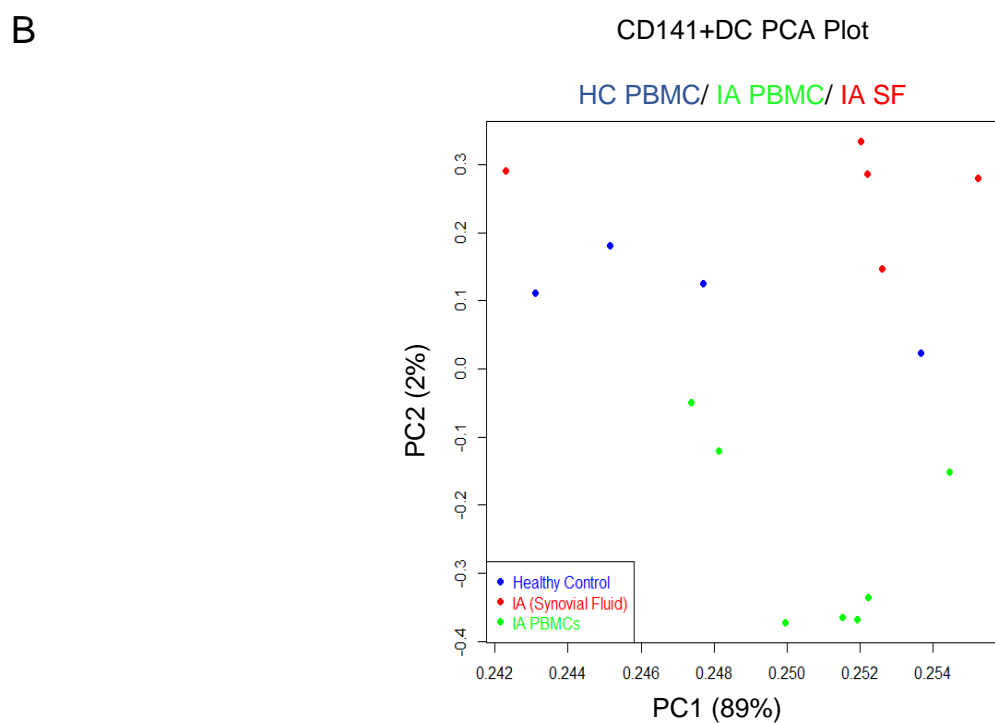
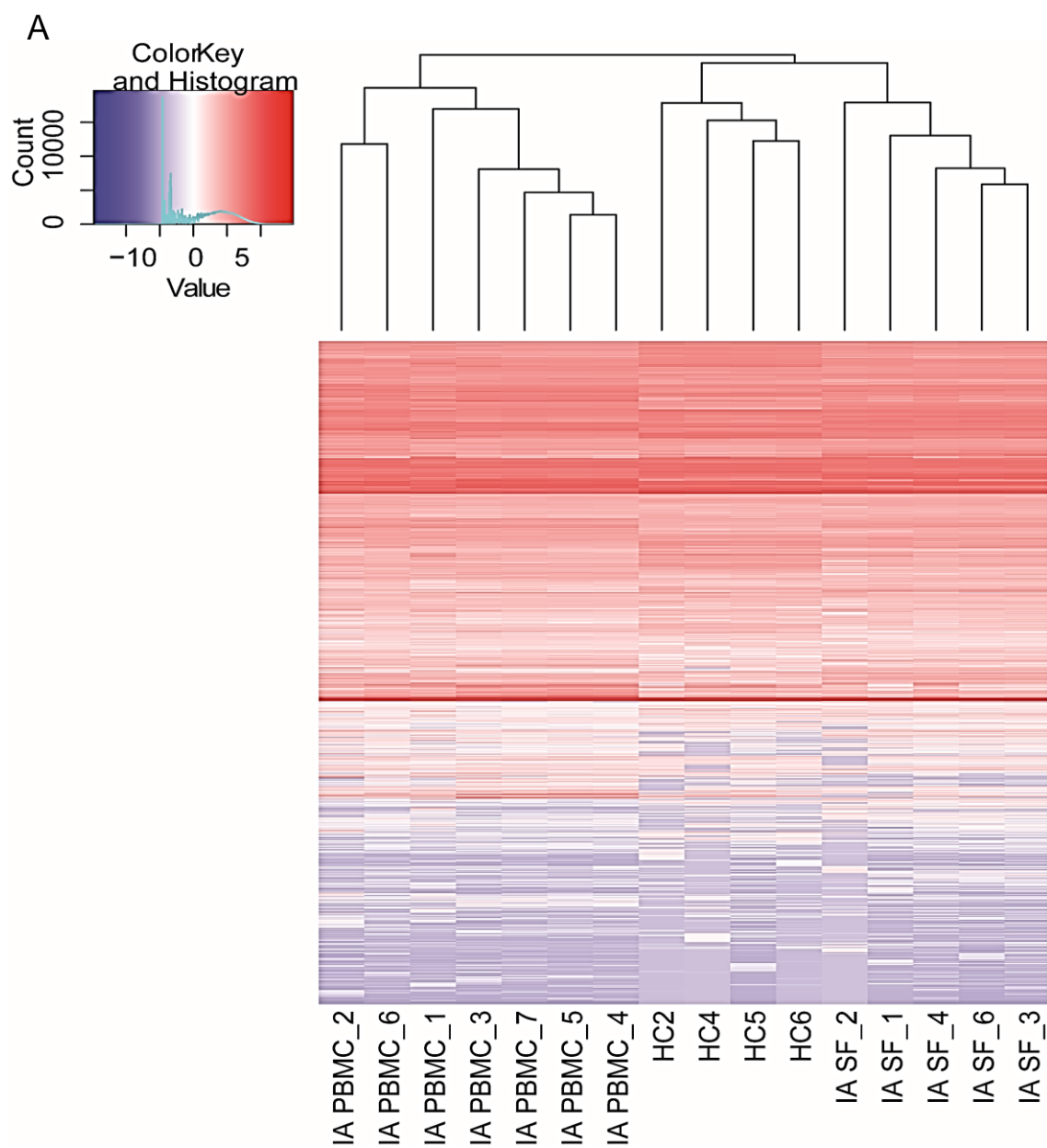


B

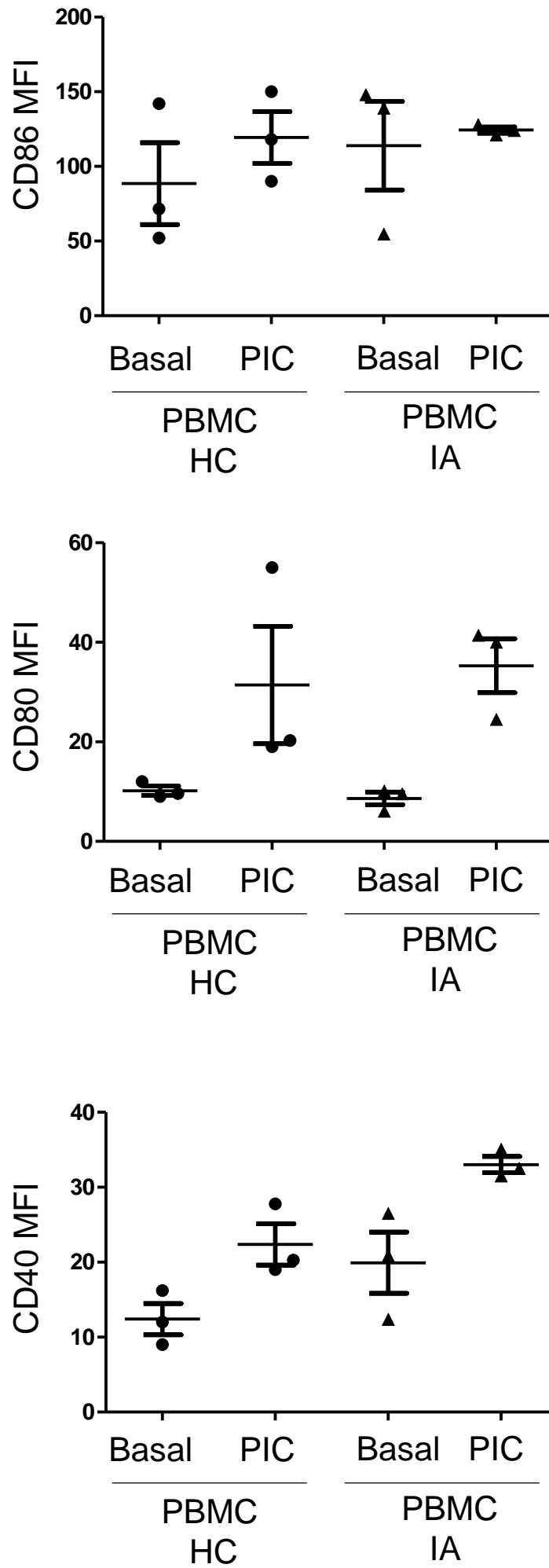


**Supplementary Figure 4:** (A) Gating strategy of sorted CD141<sup>+</sup>DC. Following removal of debris, doublets and dead cells, myeloid DC cells were gated on CD11c and HLA-DR. CD141, Clec9A and XCR-1 were all subsequently examined (using FMO controls to determine gating boundaries) to identify/confirm CD141<sup>+</sup>DC. (B) Due to the previously reported difficulty in staining XCR-1, we also performed staining using an appropriate isotype control to confirm positive XCR-1 staining.

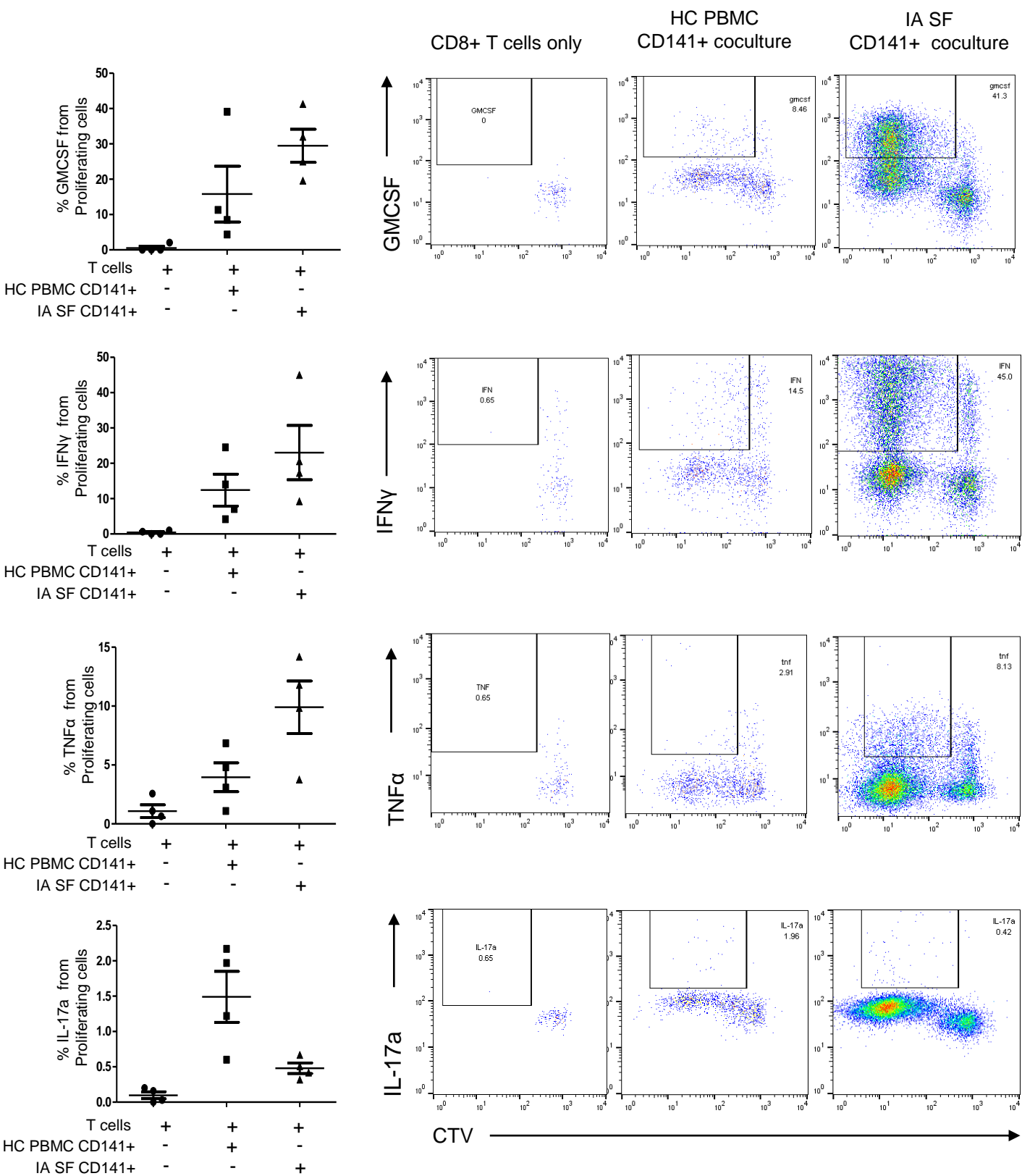




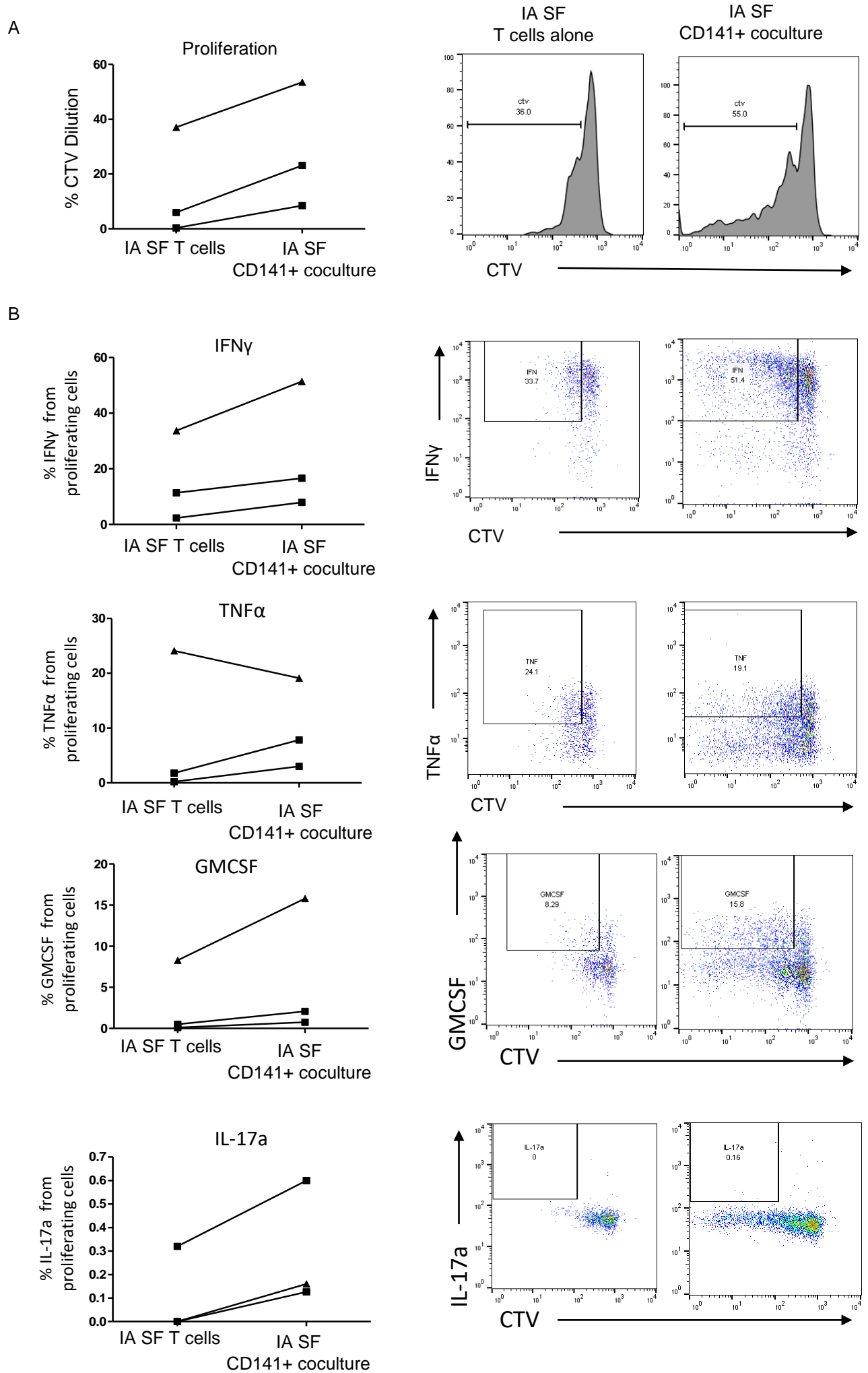
**Supplementary Figure 5:** Unsupervised Hierarchical clustering of all 18345 genes between inflammatory arthritis (IA) PBMC CD141<sup>+</sup>DC, IA SF CD141<sup>+</sup>DC and HC PBMC CD141<sup>+</sup>DC. (B) PCA analysis was performed on the total datasets of HC PBMC CD141<sup>+</sup> DC, IA SF CD141<sup>+</sup> DC and IA PBMC CD141<sup>+</sup>DC. Data shown are from 4 HC donors, 5 IA SF donors and 7 IA blood donors.



**Supplementary Figure 6:** Expression of CD86, CD80 and CD40 on sorted peripheral blood CD141<sup>+</sup>DC from HC or IA patients **peripheral blood** (n=3) basally or following stimulation with Poly:IC (1µg/ml).

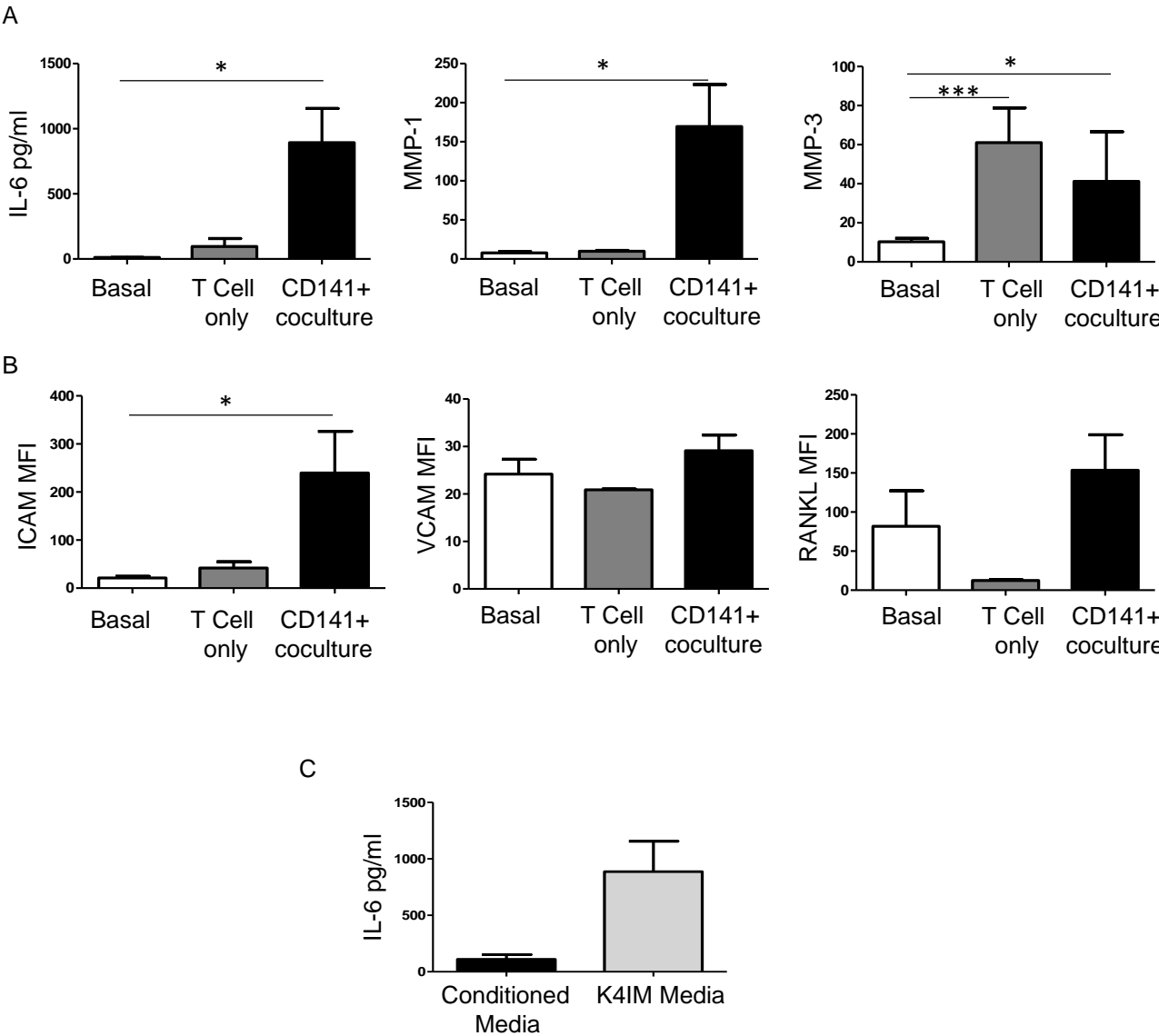


**Supplementary Figure 7:** Sorted IA synovial fluid (SF) CD141<sup>+</sup> DC or HC PBMC CD141<sup>+</sup> DC (n=4) were cocultured with allogeneic CD3<sup>+</sup> T cells from a single donor for 6 days at a ratio of 1:10 after which proliferation and cytokine production was assessed by flow cytometry. After 6 days coculture CD3<sup>+</sup> T cells were restimulated with PMA/ionomycin in the presence of brefeldin A, stained intracellularly with fluorochrome-conjugated antibodies specific for GM-CSF, IFN $\gamma$  TNF $\alpha$  and IL-17a and analysed by flow cytometry. Representative flow cytometry plots showing cytokine staining in CD8<sup>+</sup> T cells and dot plots representing the percentage of indicated cytokine within the proliferating CD8<sup>+</sup> population.

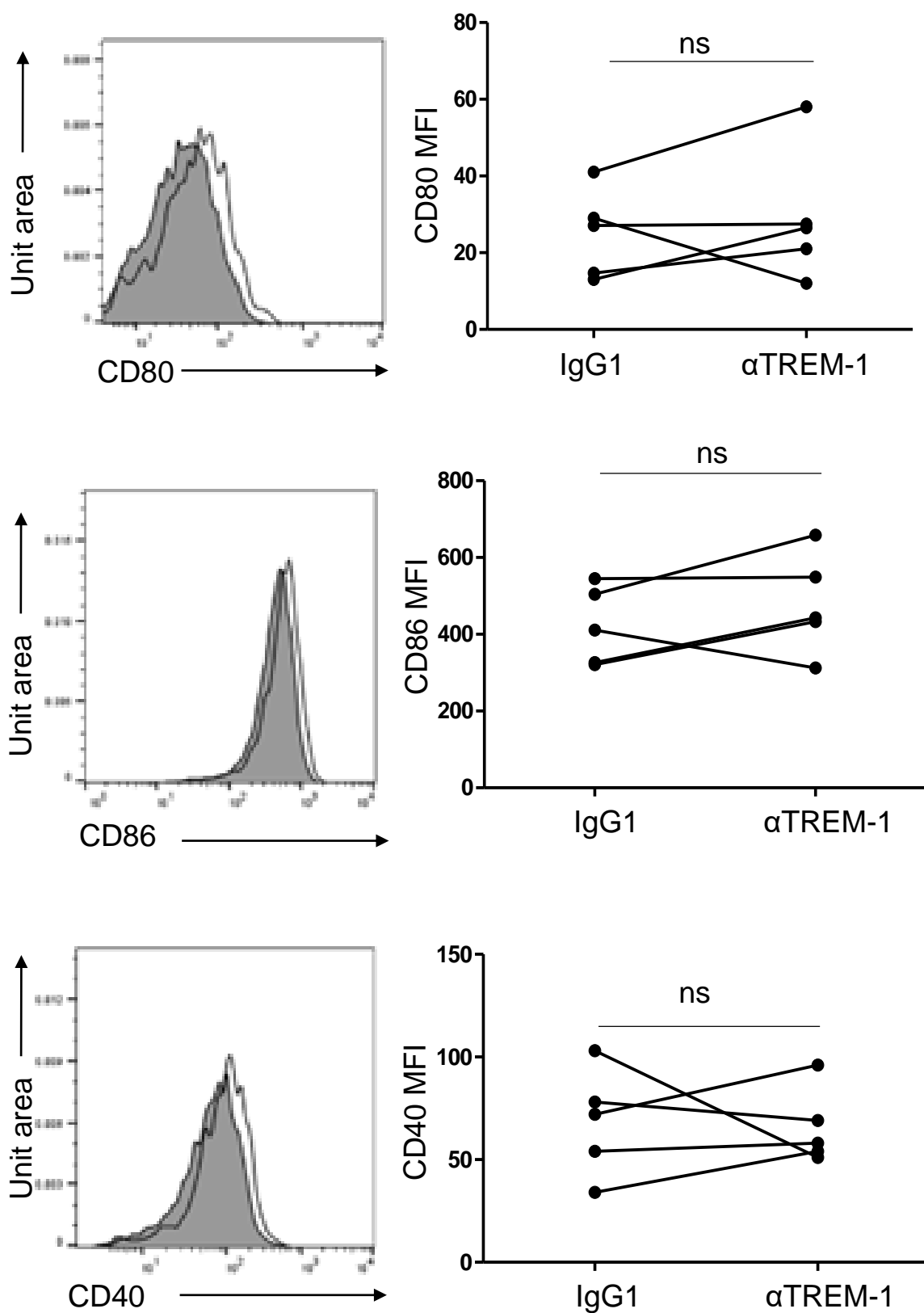


**Supplementary Figure 8:** Inflammatory arthritis (IA) synovial fluid (SF) CD3<sup>+</sup> T cells were sorted and cultured alone or with autologous synovial CD141<sup>+</sup> DC (n=3) for 4 days at a ratio of 1:10 after which proliferation and cytokine production was assessed by flow cytometry. The frequency of proliferating cells was measured by CTV dilution. After 4 days coculture CD3<sup>+</sup> T cells were restimulated with PMA/ionomycin in the presence of brefeldin A, stained intracellularly with fluorochrome-conjugated antibodies specific for IFN $\gamma$ , TNF $\alpha$ , GMCSF and IL-17a and analysed by flow cytometry. **(A)** Dot plots representing the percentage of proliferating cells in CD8<sup>+</sup> T cell populations and representative histograms showing the dilution of CTV. **(B)** Representative flow cytometry plots showing cytokine staining in CD8<sup>+</sup> T cells and dot plots representing the percentage of indicated cytokine within the proliferating CD8<sup>+</sup> population.





**Supplementary Figure 9:** (A) Levels of IL-6, MMP-1 and MMP-3 from control K4IM cells, K4IM cells stimulated with supernatants from T cells cultured on their own or K4IM cells stimulated with supernatants from CD141<sup>+</sup>DC-T cell cocultures. (B) Bar graphs representing the MFI of ICAM, VCAM and RANKL on control K4IM cells, K4IM cells stimulated with supernatants from T cells cultured on their own or K4IM cells stimulated with supernatants from CD141<sup>+</sup>DC-T cell cocultures. (C) Levels of IL-6 in CD141<sup>+</sup>DC-T cell cocultures and K4IM supernatants stimulated with these cocultures



**Supplementary Figure 10:** MFI and representative histograms displaying expression of CD80, CD86 and CD40 on HC peripheral blood CD141<sup>+</sup> DC stimulated via plate bound αTREM-1 or IgG1 control for 24 hr (n=5).