

Supplementary information

Tumour-retained activated CCR7+ dendritic cells are heterogeneous and regulate local anti-tumour cytolytic activity

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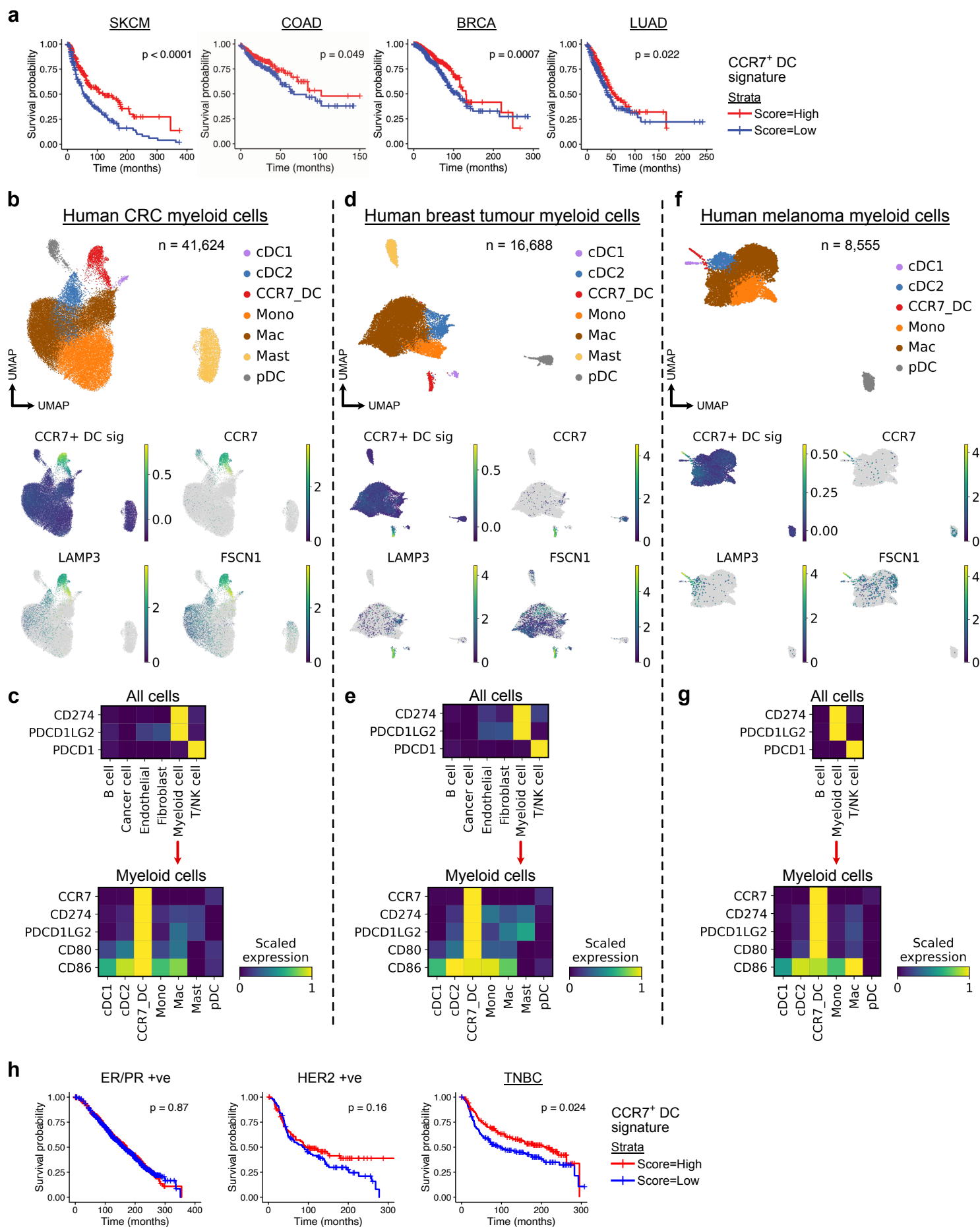
Supplementary table 1: Antibodies and reagents

Provided separately as Supplementary Data files

Supplementary data 1: Gene sets used

Supplementary data 2: Cluster DEGs in scRNA-seq of tumour DCs

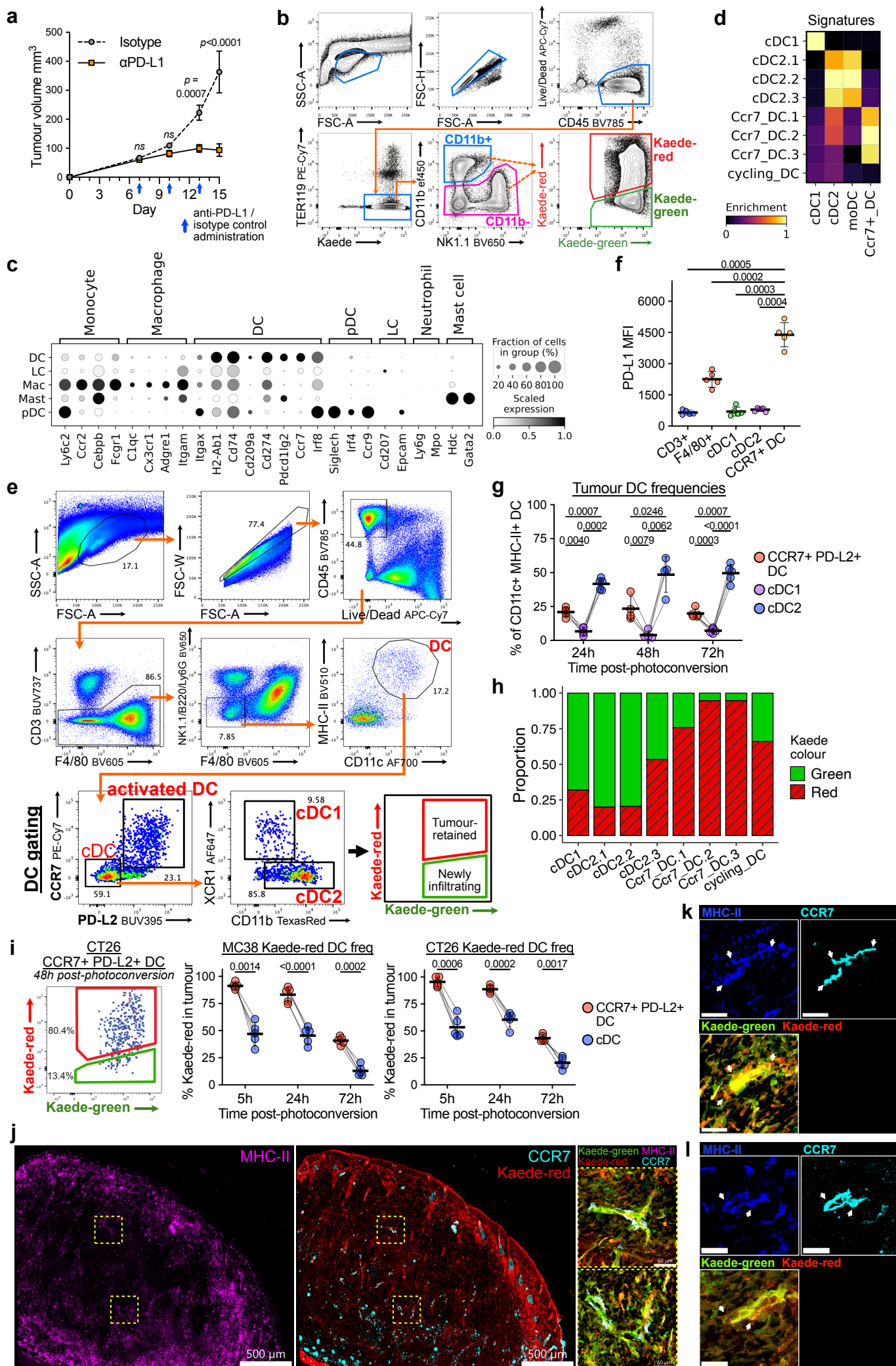
Supplementary Figure 1



Supplementary Figure 1 | CCR7⁺ DCs in human cancer.

(a) Kaplan-Meier analysis of overall survival rate in skin cutaneous melanoma (SKCM, $n = 473$), colorectal adenocarcinoma (COAD, $n = 521$), breast invasive carcinoma (BRCA, $n = 1226$), and lung adenocarcinoma (LUAD, $n = 598$) from TCGA, stratified by enrichment of CCR7⁺ DC signature genes. (b) Uniform manifold approximation projection (UMAP) of myeloid cells from scRNA-seq of human CRC²⁶ ($n = 62$ patients) and expression of CCR7⁺ DC genes (CCR7⁺ DC signature score⁵, *CCR7*, *LAMP3*, *FSCN1*). Mac, macrophage; Mast, mast cell; Mono, monocyte; pDC, plasmacytoid DC. (c) Expression of *CD274* (PD-L1), *PDCD1LG2* (PD-L2) and *PDCD1* (PD-1), *CCR7*, *CD80* and *CD86* by cell-type (all cells, top; myeloid cells only, bottom) in scRNA-seq of human CRC. (d) UMAP of myeloid cells from scRNA-seq of human breast cancer²⁴ ($n = 29$ patients) and expression of CCR7⁺ DC genes. (e) Expression of selected genes by cell-type in scRNA-seq of human breast cancer. (f) UMAP of myeloid cells from scRNA-seq of human melanoma²⁵ ($n = 25$ patients) and expression of CCR7⁺ DC genes. (g) Expression of selected genes by cell-type in scRNA-seq of human melanoma. (h) Kaplan-Meier analysis of overall survival rate in hormone receptor-positive (oestrogen receptor, ER, or progesterone receptor, PR, $n = 1369$), human epidermal growth factor receptor 2 (HER2)-positive ($n = 185$), or triple-negative breast cancer (TNBC, $n = 299$) from METABRIC cohort. Log-rank test for Kaplan-Meier survival was used (a, h).

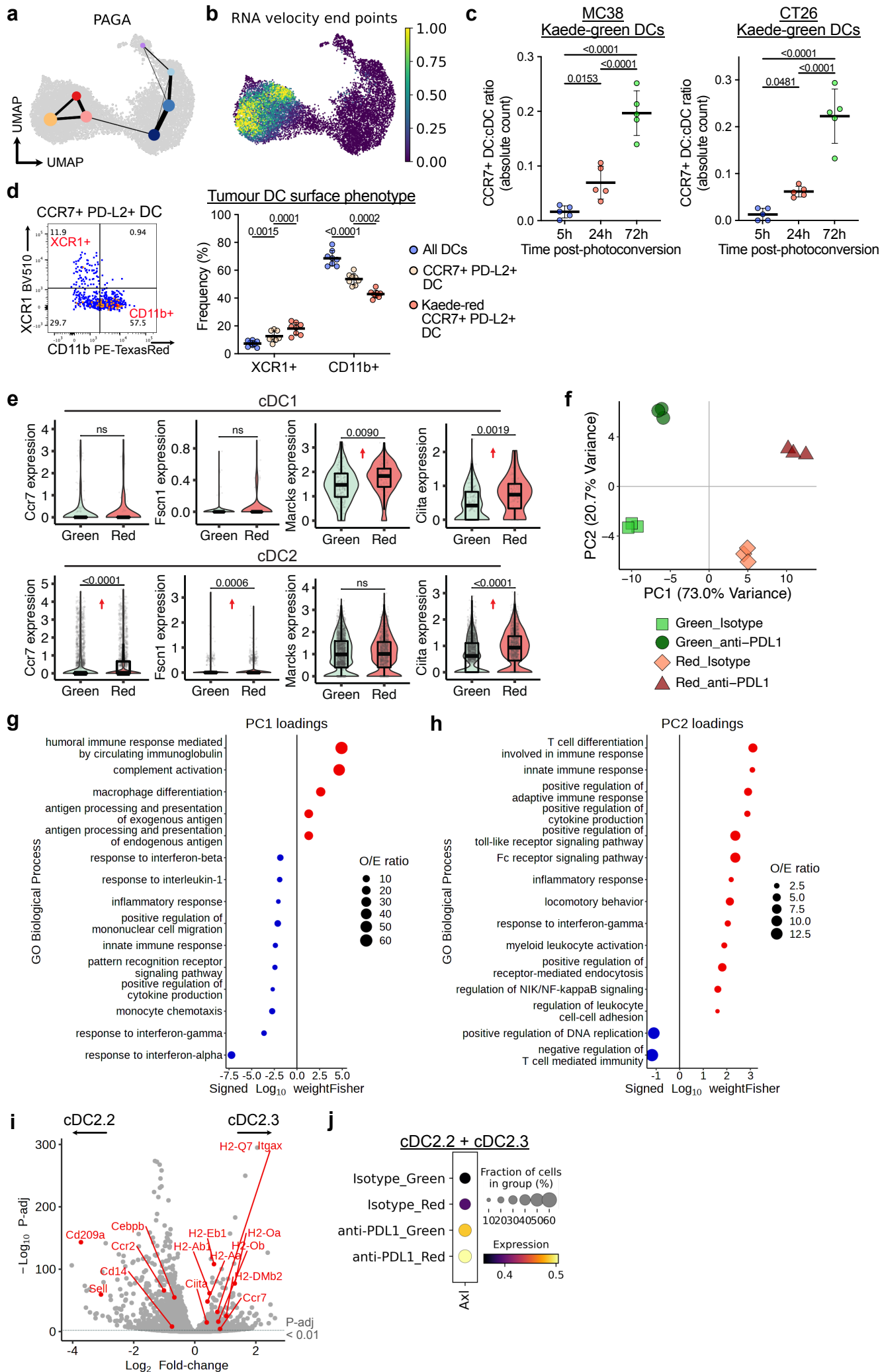
Supplementary Figure 2



Supplementary Figure 2 | Single cell profiling of mouse subcutaneous tumours.

(a) Tumour growth curves of three-dose anti-PD-L1 versus isotype control antibody treatment in MC38-Ova tumours. Data from scRNA-seq experiment (Fig. 1) and includes 5 mice in the isotype control group and 6 mice in the anti-PD-L1 group. (b) Fluorescence-activated cell sorting (FACS) strategy for isolation of tumour immune cells for scRNA-seq. CD45⁺ cells were sorted to CD11b⁺ or CD11b^{-/low} fractions to ensure appropriate representation of various cell types in the scRNA-seq data. Both fractions were combined for scRNA-seq analysis and annotated based on their transcriptome. (c) Canonical marker gene expression in myeloid cell subsets from Fig. 1b. (d) Gene signature scores of published transcriptomic reference signatures in DC clusters from Fig. 1c. Ccr7⁺_DC signature obtained from mRegDC genes in Maier *et al*⁵. Monocyte-derived DCs (moDC) signature scored highly in cDC2 clusters, consistent with known challenges in distinction of cDC2 and moDC⁹. (e) Representative flow cytometry gating strategy for tumour and LN DCs (Fig. 1-3). Activated DCs were defined as live, CD45⁺ lineage⁻ (CD3, NK1.1, B220, Ly6G, F4/80) CD11c⁺MHC-II⁺ PD-L2⁺CCR7⁺ cells (PD-L2 expression was more specific for activated DCs than PD-L1). Where Kaede transgenic mice were used, Kaede⁺ cells were gated. (f) Flow cytometry of surface PD-L1 expression on immune cells from MC38-Ova tumours. (g) Flow cytometry of DC composition in MC38-Ova tumours. (h) Kaede fluorescence by DC cluster in the scRNA-seq data. (i) Flow cytometry of Kaede fluorescence in DCs from MC38 and CT26 tumours; photoconversion time course. (j) Representative microscopy of MC38 tumours 72h after tumour photoconversion. Insets highlight selected regions with tumour-residing (Kaede-red⁺) CCR7⁺MHC-II⁺ DCs. (k-l) Zoomed-in representative microscopy of MC38 (k) and CT26 (l) tumours 72h after tumour photoconversion. Arrows point to Kaede-red⁺CCR7⁺MHC-II⁺ DCs. Scale bar, 30µm. Two-way analysis of variance (ANOVA) and Šidák's multiple comparisons test (a), one-way ANOVA and Šidák's multiple comparisons test (f), or paired two-sided student's t-test (g, i) were used. Points represent independent mice (f, g, i). Data are shown as means ± s.e.m. (a), or means ± s.d. (f, g, i). The results shown in (f) are from one experiment (*n* = 5 animals), representative of three independent experiments; (g) are from one experiment (24h/72h *n* = 5; 48h *n* = 4 animals), representative of three independent experiments; (i) are from one experiment (*n* = 5 animals per group), representative of two independent experiments; and (j-l) are representative of three independent experiments (*n* = 7 animals) for MC38 tumours and two independent experiments (*n* = 4 animals) for CT26 tumours.

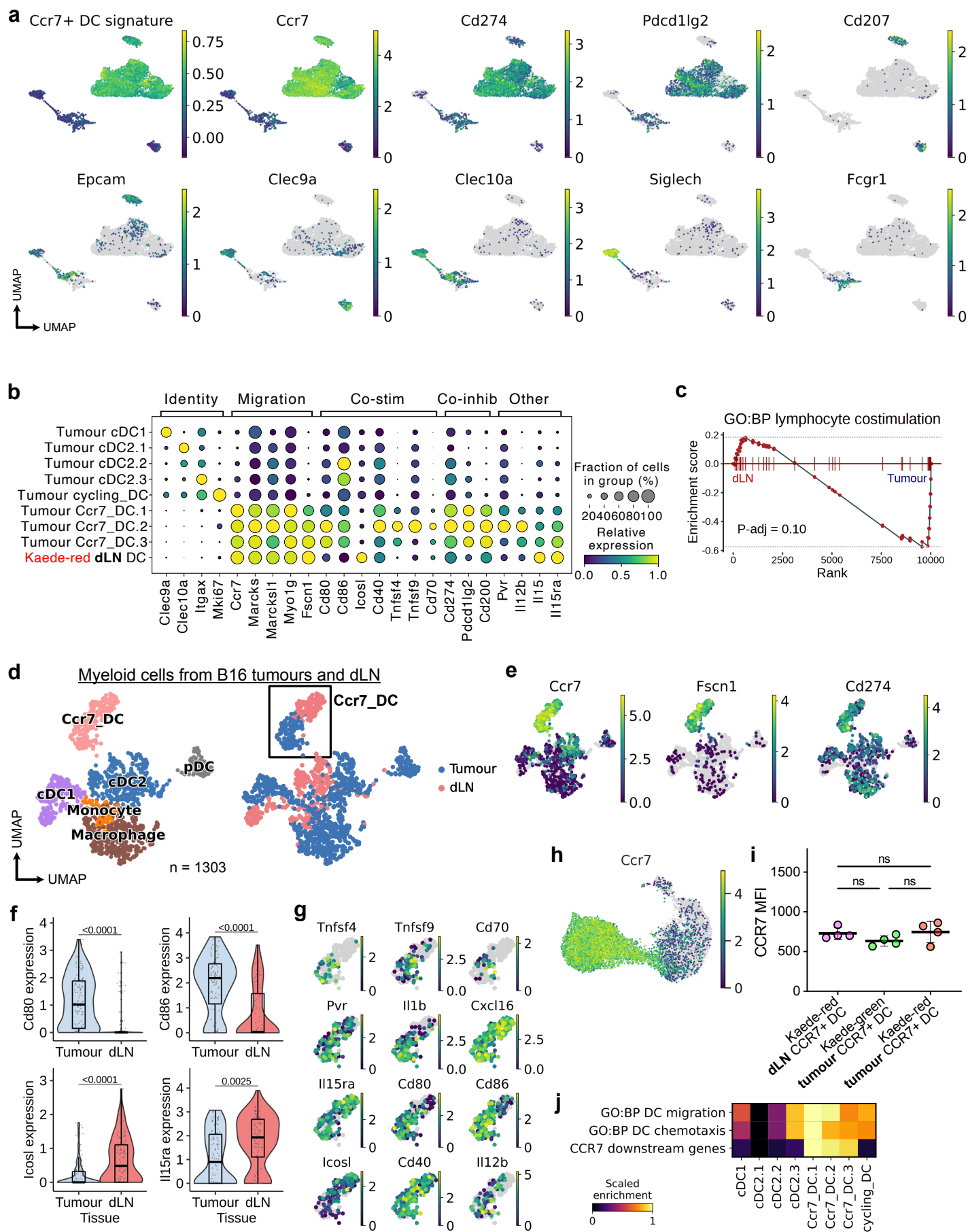
Supplementary Figure 3



Supplementary Figure 3 | Activation trajectory in tumour DCs.

(a) Partition-based graph abstraction (PAGA) of scRNA-seq of tumour DCs. (b) Terminal states (end points) of RNA velocity analysis. (c) Flow cytometry of CCR7⁺ DC to cDC ratio in Kaede-green DCs from MC38 and CT26 tumours; time course post-photoconversion. (d) Flow cytometry of surface cDC1 (XCR1⁺) and cDC2 (CD11b⁺) marker expression on CCR7⁺ DCs from MC38-Ova tumours 48h post-photoconversion. (e) Expression of selected DC migration genes and *Ciita* in scRNA-seq data of cDC1 and cDC2. Arrows indicate relative expression in Kaede-red versus Kaede-green cells. (f) Principal component (PC) analysis of scRNA-seq of cDC2s, pseudo-bulked to 3 artificial replicates per condition. PC1, variance associated with Kaede profile; PC2, variance associated with treatment. (g-h) Gene ontology (GO) term enrichment test for the top 100 loading genes of PC1 (g) and PC2 (h). O/E, observed/expected ratio; signed Fisher weight indicates direction of gene loading. (i) Differential gene expression between cDC2.2 and cDC2.3. Selected differentially expressed genes (DEG) relating to DC maturation, antigen presentation, MHC-II and migration are highlighted in red (all $P\text{-adj} < 0.01$). (j) Expression of *Axl* in cDC2.2 and cDC2.3 combined. One-way analysis of variance (ANOVA) and Šidák's multiple comparisons test (c), paired two-sided student's t-test (d), two-sided Wilcoxon rank-sum test with Benjamini-Hochberg multiple-testing correction (e), Fisher's exact test (g-h), or two-sided Wald test with Benjamini-Hochberg multiple testing correction (i) were used. Points represent independent mice (c-d). Data are shown as means $\pm$ s.d. (c-d), or box (median; box, 25th percentile and 75th percentile; whiskers, 1.5*inter-quartile range) and violin plots (e). The results shown in (c) are from one experiment ($n = 5$ animals per group), representative of two independent experiments; and (d) are from two independent experiments ($n = 8$ animals).

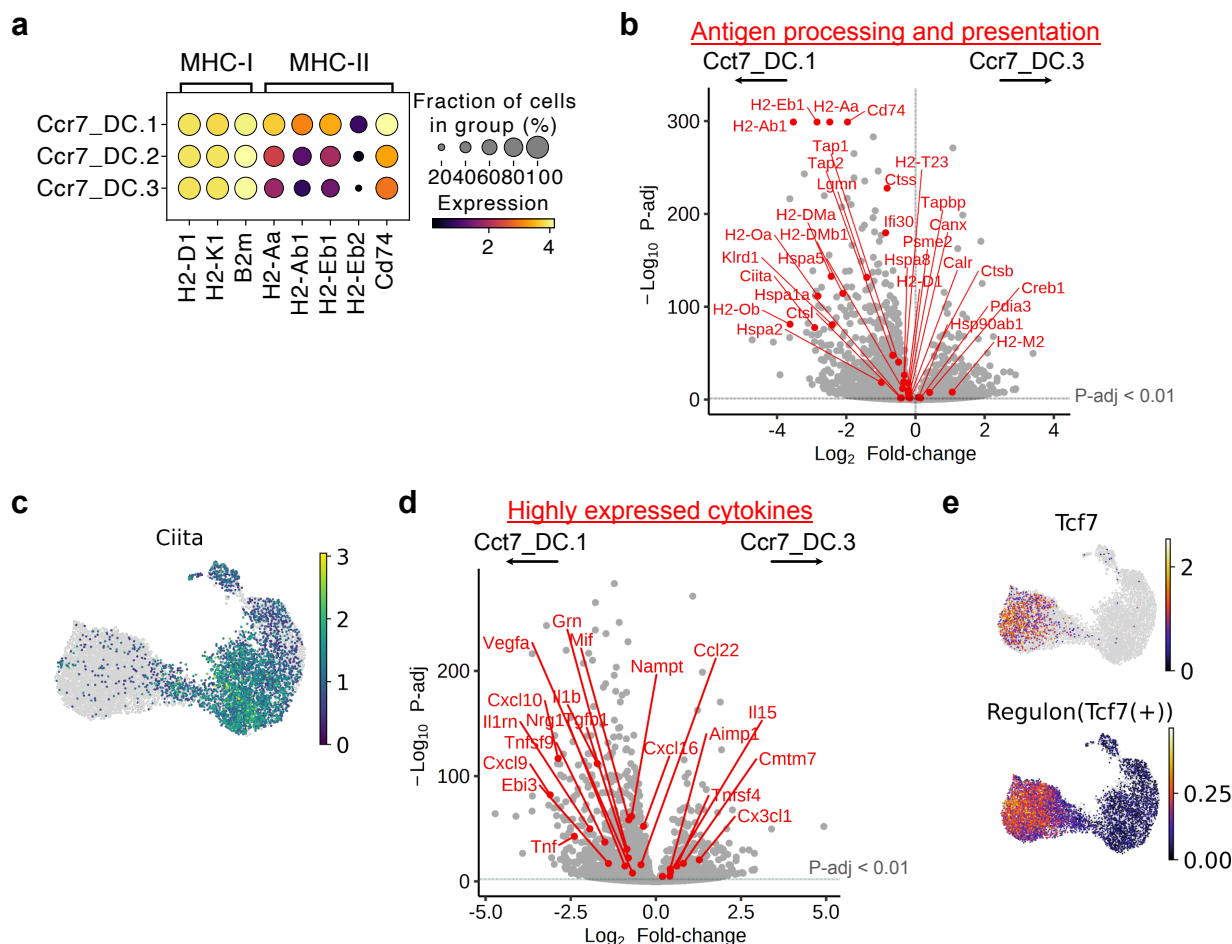
Supplementary Figure 4



Supplementary Figure 4 | Comparison of tumour-residing CCR7⁺ DCs versus LN CCR7⁺ DC emigrants.

(a) Expression of CCR7⁺ DC or myeloid cell canonical marker genes in scRNA-seq of LN myeloid cells (Fig. 2d). (b) Expression of selected genes in tumour DCs (isotype control-treated) and Kaede-red DC emigrants in tumour-dLNs. (c) GSEA of “*GO:biological process (BP) lymphocyte co-stimulation (GO:0031294)*”, comparing Kaede-red CCR7⁺ DCs from the dLN versus tumour CCR7⁺ DCs. P-adj, Benjamini-Hochberg-adjusted p values. (d) UMAP of myeloid cells from scRNA-seq of murine subcutaneous B16 tumours and tumour-dLNs, by cell-type and tissue. (e) Expression of CCR7⁺ DC genes in (d). (f) Violin plots of selected genes in scRNAseq of CCR7⁺ DCs from B16 tumours and tumour-dLNs. (g) Expression of selected DEGs between in tumour and dLN CCR7⁺ DCs from (d, box). (h-i) Expression of CCR7 in DCs from MC38-Ova tumours in the scRNA-seq data (h) and by flow cytometry (i), 48h post-photoconversion. (j) Gene signature enrichment of pathways relating to DC movement and CCR7 downstream signalling. Two-sided Wilcoxon rank-sum test with Benjamini-Hochberg multiple-testing correction (f), or one-way analysis of variance (ANOVA) and Šidák’s multiple comparisons test (i) were used. Points represent independent mice (i). Data are shown as box (median; box, 25th percentile and 75th percentile; whiskers, 1.5*inter-quartile range) and violin plots (f), or means ± s.d. (i). The results shown in (i) are from one experiment (*n* = 4 animals per group), representative of three independent experiments.

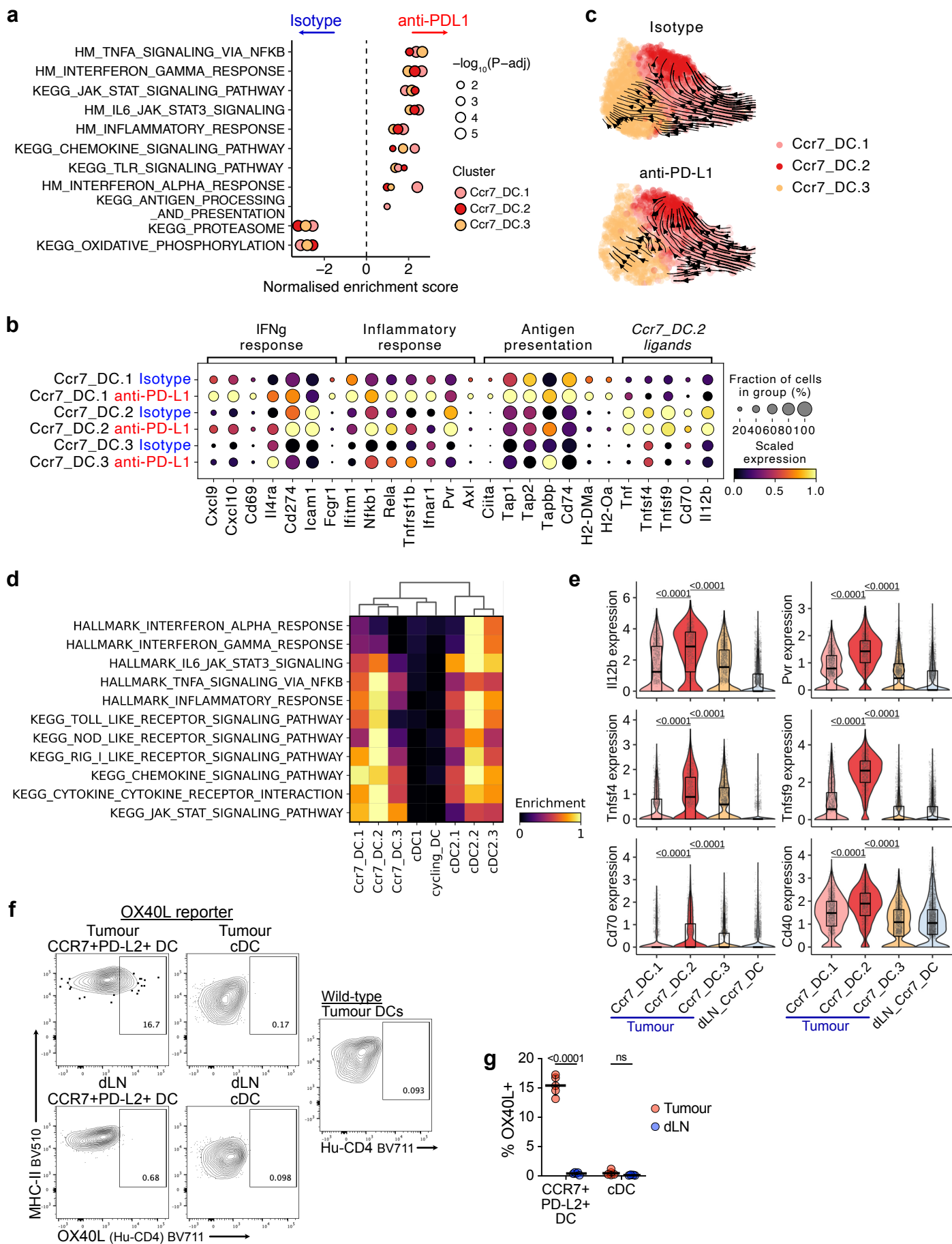
Supplementary Figure 5



Supplementary Figure 5 | Transcriptional changes in CCR7⁺ DCs with prolonged tumour residence.

(a) Dot plot of class-I and class-II MHC gene expression (log-transformed, unscaled) in CCR7⁺ DCs. (b) Differential gene expression between Ccr7_DC.1 and Ccr7_DC.3. Significant DEGs from “KEGG antigen processing and presentation” are highlighted in red ($P\text{-adj} < 0.01$). (c) Expression of *Ciita*. (d) Differential gene expression between Ccr7_DC.1 and Ccr7_DC.3. Significant DEGs from “GO: molecular function (MF) cytokine activity (GO:0005125)” are highlighted in red ($P\text{-adj} < 0.01$). (e) *Tcf7* expression and regulon activity score. Two-sided Wald test with Benjamini-Hochberg multiple testing correction (b, d) was used.

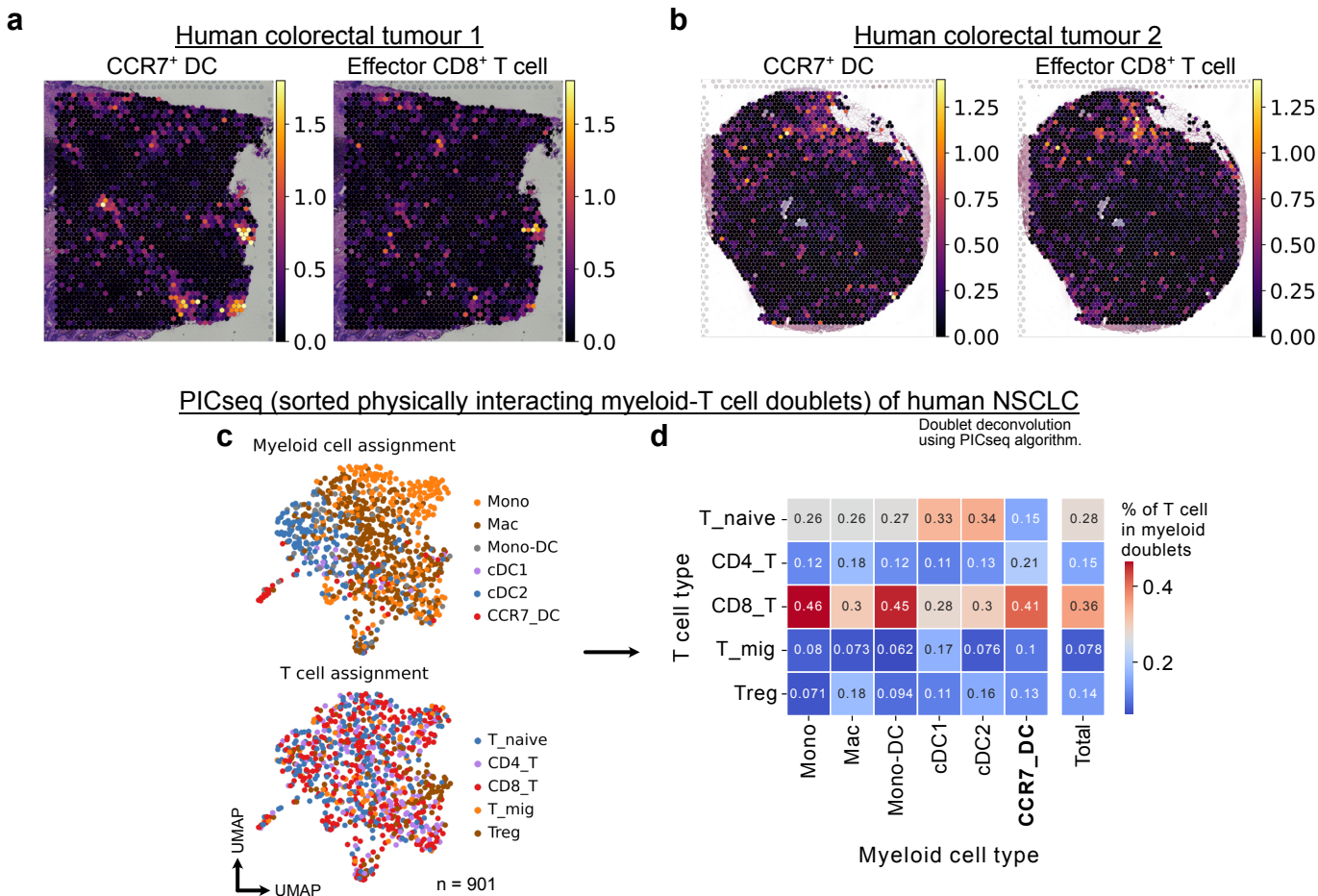
Supplementary Figure 6



Supplementary Figure 6 | CCR7⁺ DC activation following anti-PD-L1 treatment.

(a) GSEA of CCR7⁺ DCs in anti-PD-L1-treated versus isotype control-treated tumours; Hallmark (HM) and KEGG pathways. Only significant pathways (Benjamini-Hochberg-adjusted p values (P -adj) < 0.05) are shown. (b) Dot plot of selected leading-edge genes from GSEA analysis of “*Hallmark interferon gamma response*”, “*Hallmark inflammatory response*”, “*KEGG antigen processing and presentation*” pathways from (a), and others. (c) RNA velocity trajectory in tumour CCR7⁺ DCs split by treatment group. (d) Gene signature enrichment of selected pathways in scRNA-seq of DCs. (e) Expression of selected ligands differentially expressed between scRNA-seq of tumour CCR7⁺ DCs and CCR7⁺ DC tumour emigrants in the dLN (Kaede-red). (f) Representative flow cytometry of surface OX40L expression (using OX40L⁺/Human-CD4 reporter mice) on DCs from MC38-Ova tumours and dLNs. Wild-type DC (non-reporter) were used to determine OX40L⁺ gate. (g) Quantification of (f). Two-sided Wilcoxon rank-sum test with Benjamini-Hochberg multiple-testing correction (e), or paired two-sided student’s t-test (g) were used. Points represent independent mice (g). Data are shown as box (median; box, 25th percentile and 75th percentile; whiskers, 1.5*inter-quartile range) and violin plots (e), or means ± s.d. (g). The results shown in (f-g) are from one experiment ($n = 5$ animals), representative of two independent experiments.

Supplementary Figure 7

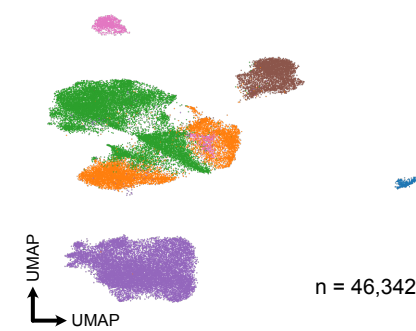


Supplementary Figure 7 | CCR7⁺ DC-CD8⁺ T cell interaction in human solid tumours.

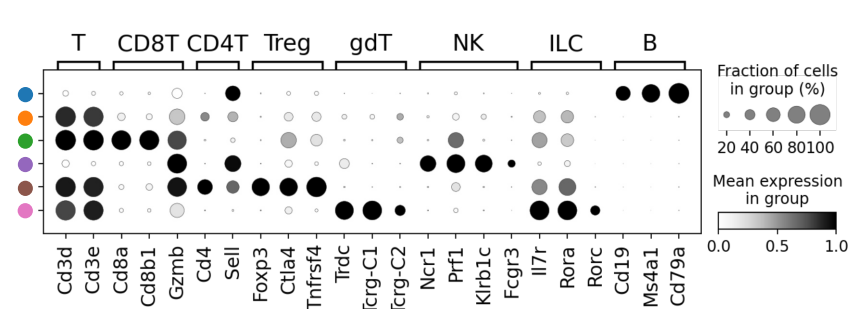
(a-b) Gene signature scores for CCR7⁺ DCs and effector CD8⁺ T cells in spatial transcriptomics (10X Genomics Visium) of independent human CRC tumour sections associated with Fig. 4d (*n* = 2). (c) Sequencing of physically interacting cells (PICseq, myeloid-T cell doublets) from human NSCLC (*n* = 10 patients). UMAP of PICs coloured by the deconvolved myeloid and T cell assignment for each doublet (PICseq algorithm). Each dot represents one myeloid-T cell doublet. (d) Heatmap of frequency (values) of myeloid-T cell doublet combinations in PICseq of NSCLC.

Supplementary Figure 8

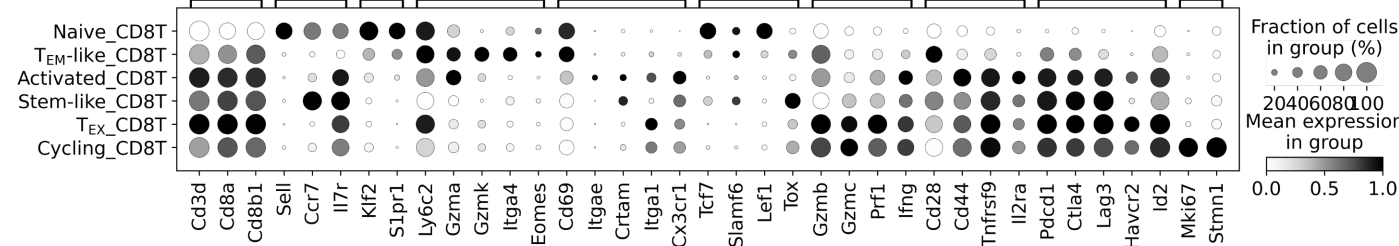
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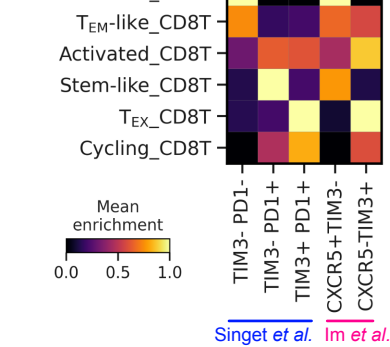
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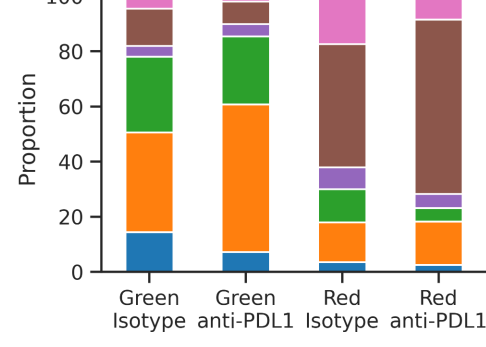
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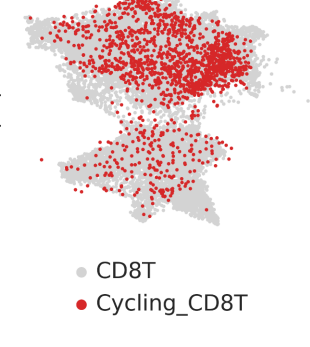
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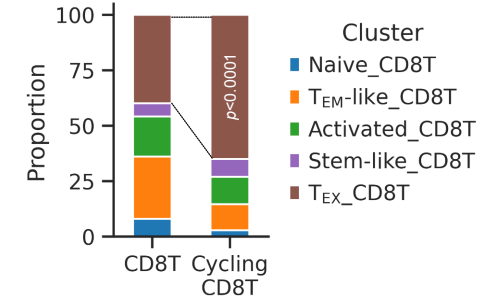
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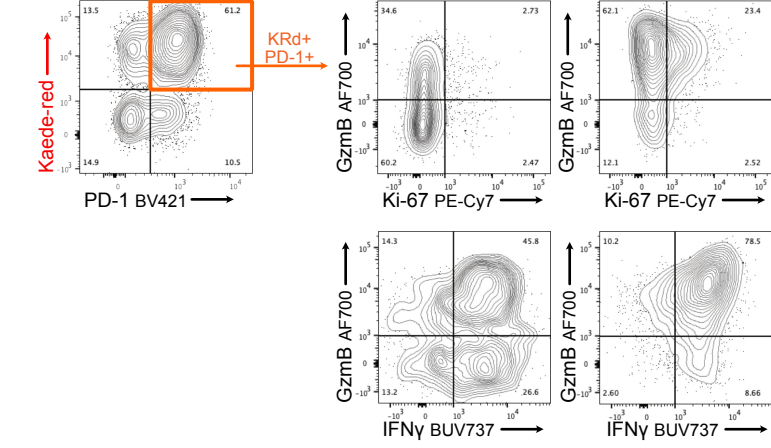
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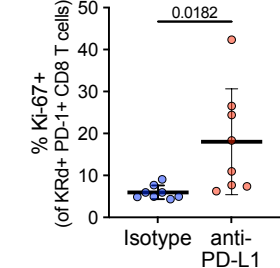
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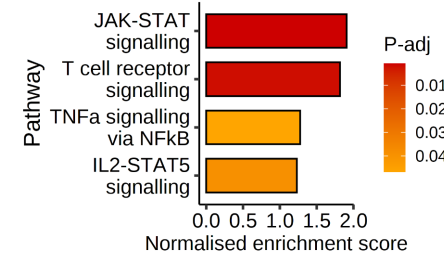
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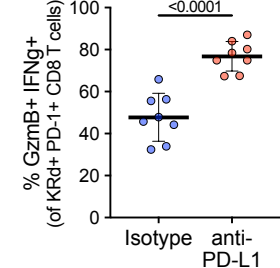
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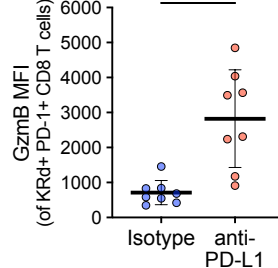
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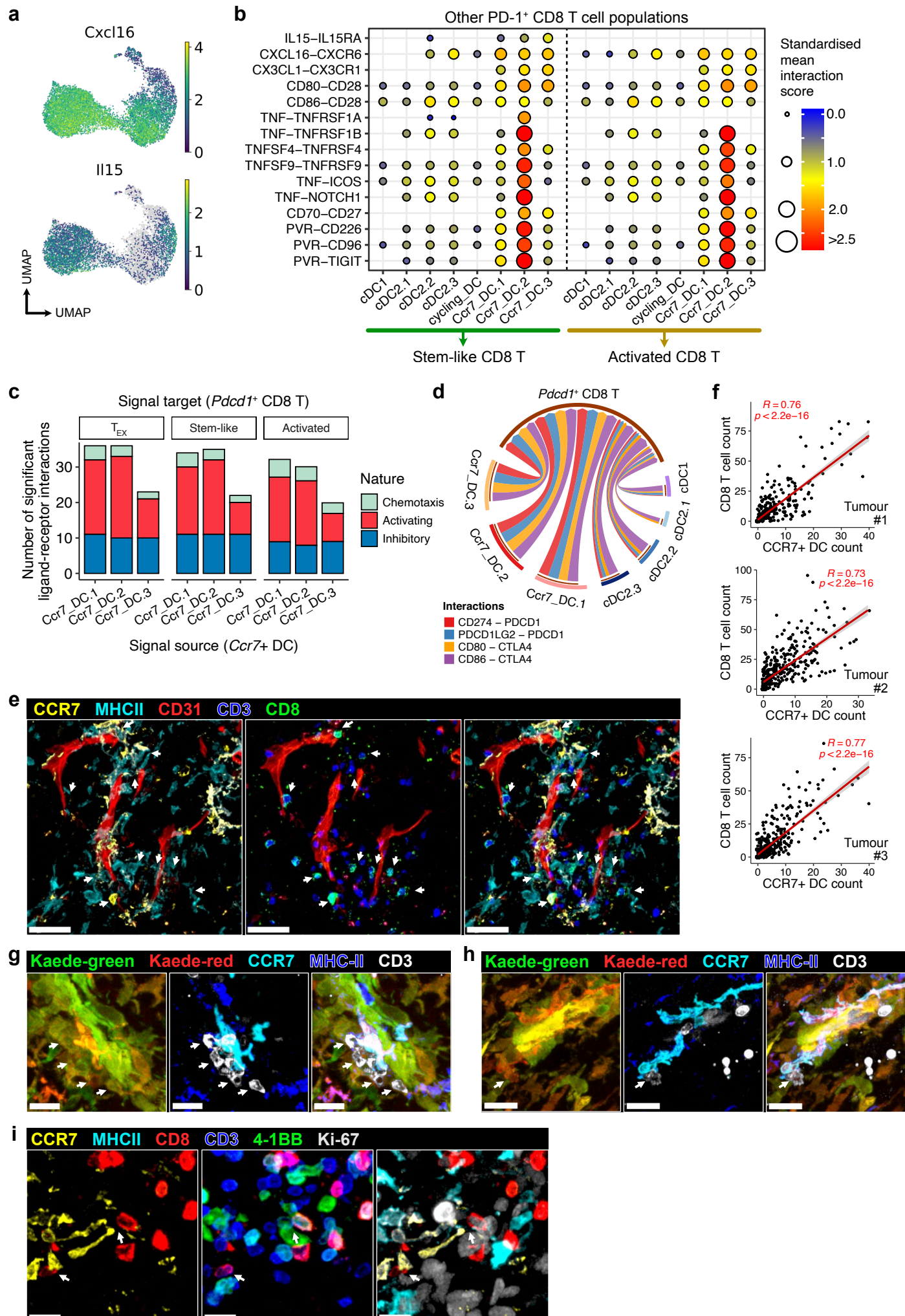
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Supplementary Figure 8 | Single cell profiling of tumour CD8⁺ T cells following anti-PD-L1 treatment.

(a) UMAP of TILs from scRNA-seq of FACS-sorted CD45⁺TER119⁻ Kaede-green⁺/Kaede-red⁺ cells 48h after photoconversion of subcutaneous MC38-Ova tumours, and canonical marker gene expression (b). (c) Canonical marker gene expression of CD8⁺ T cell clusters from Fig. 5a. (d) Gene signature enrichment of published T cell exhaustion transcriptomic signatures in CD8⁺ T cell clusters. (e) Proportion of CD8⁺ T cells by Kaede fluorescence and treatment group. (f) Regression of cell cycle genes from the cycling_CD8T cluster followed by re-integration, and label transfer. Majority of the cycling cluster embedded with the CD8⁺ T_{EX} cell cluster. (g) Proportion of cycling_CD8T cells belonging to each CD8⁺ T cell cluster, from (f). (h) Representative flow cytometry of Ki-67, GzmB and IFN γ expression in CD3⁺CD8⁺Kaede-red⁺PD-1⁺ T cells; isotype control and anti-PD-L1 treatment; MC38-Ova tumours (h-l). (i) Flow cytometry of Kaede-red⁺PD-1⁺CD8⁺ T cells, showing increased frequency of cycling cells (Ki-67⁺) following anti-PD-L1. (j) GSEA for T_{EX} CD8⁺ T cells from anti-PD-L1 versus isotype control-treated tumours. P-adj, Benjamini-Hochberg-adjusted p values. (k) Flow cytometry of Kaede-red⁺PD-1⁺CD8⁺ T cells, showing increased frequency of GzmB⁺IFN γ ⁺ cells following anti-PD-L1. (l) Flow cytometry of Kaede-red⁺PD-1⁺ CD8⁺ T cells, showing increased expression (MFI) of GzmB following anti-PD-L1. Chi-squared test for over-representation of T_{EX} CD8⁺ T cells (g), or two-sided student's t-test (i, k, l) were used. Points represent independent mice and data are shown as means $\pm$ s.d. (i, k, l). The results shown in (h-i, k-l) are from one experiment ($n = 8$ animals), representative of two independent experiments.

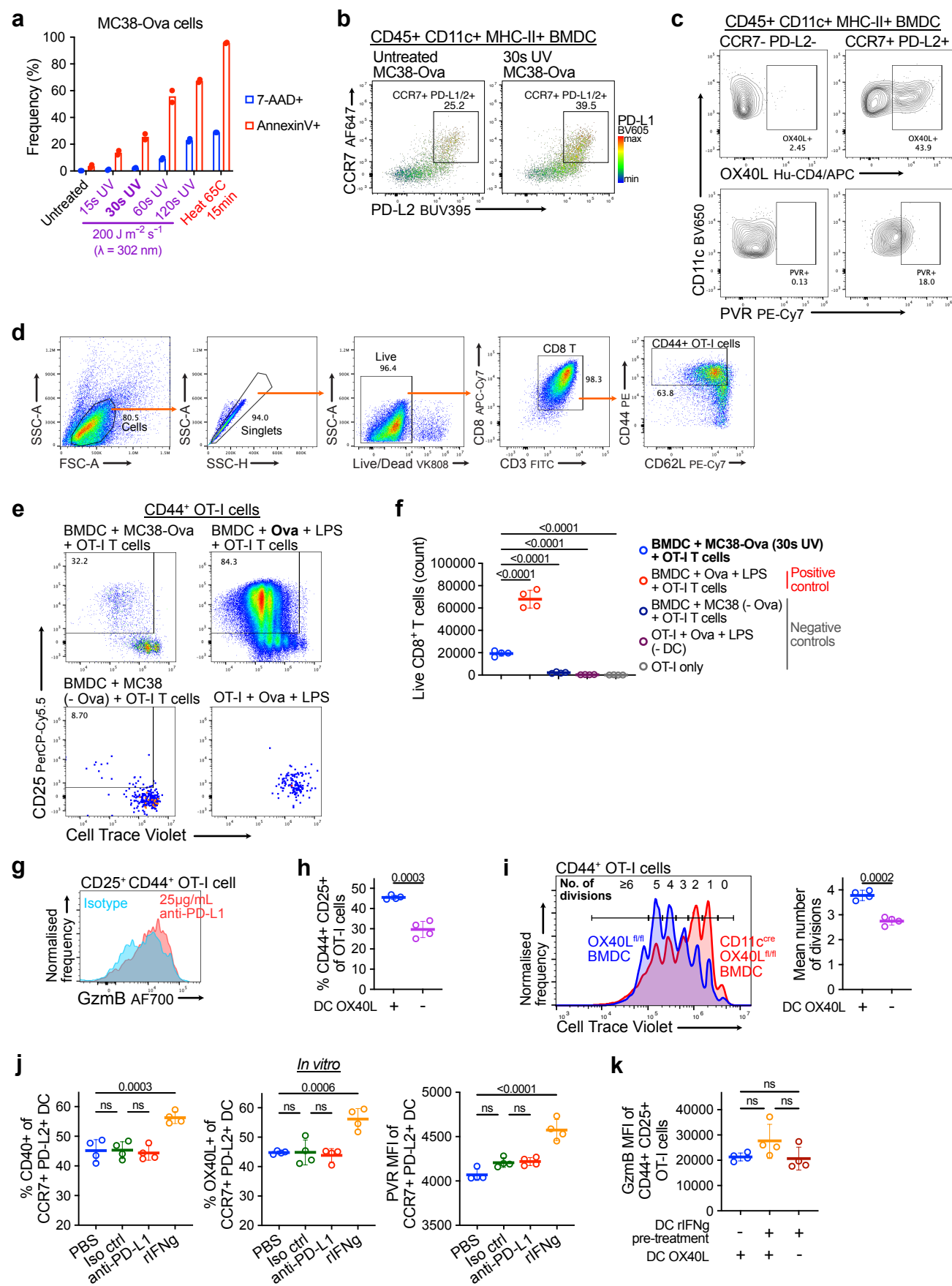
Supplementary Figure 9



Supplementary Figure 9 | CCR7⁺ DC-CD8⁺ T cell interaction in murine tumours.

(a) Expression of *Cxcl16* and *Il15*. (b-d) CellPhoneDB cell-cell communication analysis between DCs and *Pdcd1*⁺ CD8⁺ T cells in scRNA-seq of MC38-Ova tumours. (b) Ligand-receptor predicted interactions between tumour DCs and activated or stem-like CD8⁺ cells. (c) Interactions that are well-described in existing literature to influence CD8⁺ T cells function were identified and classified based on whether engagement of the cognate T cell receptor was activating (increase in effector function, proliferation, or survival) or inhibitory in nature. (d) *PDCD1* or *CTLA4*-mediated inhibitory signals; edge width scaled to standardised interaction scores. Only significant interactions ($p < 0.05$) shown (b-d). (e) Representative confocal microscopy images of MC38 tumours, showing co-localisation of CCR7⁺MHC-II⁺ DCs and CD3⁺CD8⁺ T cells (arrows). Scale bar, 80 μ m. (f) Quantification of (e); Pearson correlation between CCR7⁺ DC and CD8⁺ T cell counts in 200 x 200 μ m fields of MC38 tumour sections. 3 independent tumours were analysed. (g-h) Representative microscopy of independent MC38 tumours 48h after tumour photoconversion, showing interactions between Kaede-red CCR7⁺MHC-II⁺ DCs and Kaede-red CD3⁺ T cells (arrows). Scale bar, 15 μ m (g), 20 μ m (h). (i) Representative microscopy of independent MC38 tumour, showing co-localisation of CCR7⁺MHC-II⁺ DCs and CD3⁺CD8⁺ 4-1BB⁺/Ki-67⁺ T cells (arrows). Scale bar, 15 μ m. Two-sided Pearson correlation was used (f). Data are shown as linear regression with 95% confidence interval (f). The results shown in (e) are representative of two independent experiments ($n = 3$ animals); and (g-i) are representative of two independent experiments ($n = 5$ animals).

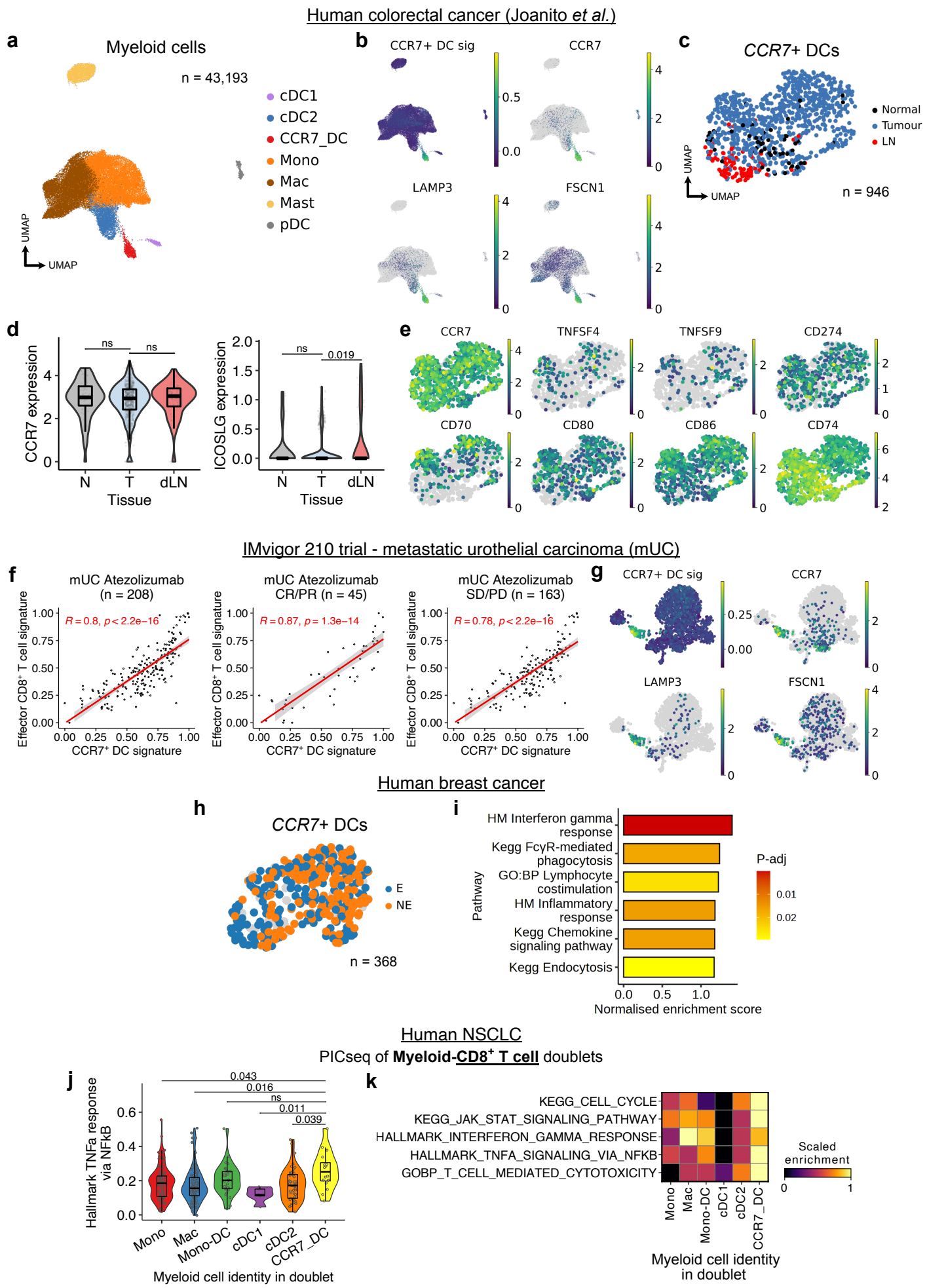
Supplementary Figure 10



Supplementary Figure 10 | *In vitro* DC and OT-I CD8⁺ T cell cultures.

(a) Apoptosis and cell death following UV irradiation of MC38-Ova cell monolayer *in vitro*; for optimisation of tumour cell-line apoptosis. 30s UV exposure was used for subsequent experiments. (b) Representative flow cytometry and FACS isolation strategy for activated BMDC following 8h culture with UV-irradiated MC38-Ova cells (Fig. 5f-g). (c) Representative flow cytometry of OX40L and PVR expression on BMDCs. (d) Flow cytometry gating strategy for CD44⁺ OT-I T cells (Fig. 5g-k). (e) Representative flow cytometry of OT-I activation and proliferation in DC co-cultures. Culture with apoptotic MC38-Ova experienced FACS-sorted CCR7⁺PD-L2⁺ BMDC, top left; positive control (BMDC + Ova + LPS), top right; negative controls, bottom left (no Ova antigen) and right (no DC). (f) Number of live CD8⁺ T cells in CCR7⁺PD-L2⁺ BMDC + OT-I co-culture set-up versus controls. (g) Representative flow cytometry histograms of GzmB expression in OT-I cells; +/- anti-PD-L1 antibodies. (h) Flow cytometry of OT-I activation, and (i) CTV proliferation in OT-I cells and mean number of cell divisions, following culture with OX40L-expressing (+, OX40L^{fl/fl}) or OX40L-deficient (-, CD11c^{cre}OX40L^{fl/fl}) CCR7⁺PD-L2⁺ BMDC. (j) Flow cytometry of CCR7⁺PD-L2⁺ BMDCs following 8h treatment with antibodies or recombinant IFN γ (rIFN γ), which were used for OT-I co-cultures in subsequent independent experiments. (k) Flow cytometry of OT-I cells following co-culture with CCR7⁺PD-L2⁺ BMDC; DCs pre-treated with rIFN γ (+) or PBS (-), OX40L-expressing (+) or OX40L-deficient (-) BMDCs. One-way analysis of variance (ANOVA) and Šidák's multiple comparisons test (f, j-k), or two-sided student's t-test (h-i) was used. Points represent independent biological samples (a, f, h-k). Data are shown as means (a), or means $\pm$ s.d. (f, h-k). The results shown in (a) is from one experiment; (b-c) are from one experiment ($n = 4$ biological samples), representative of three independent experiments; and (d-k) are from one experiment ($n = 4$ biological samples), representative of two independent experiments.

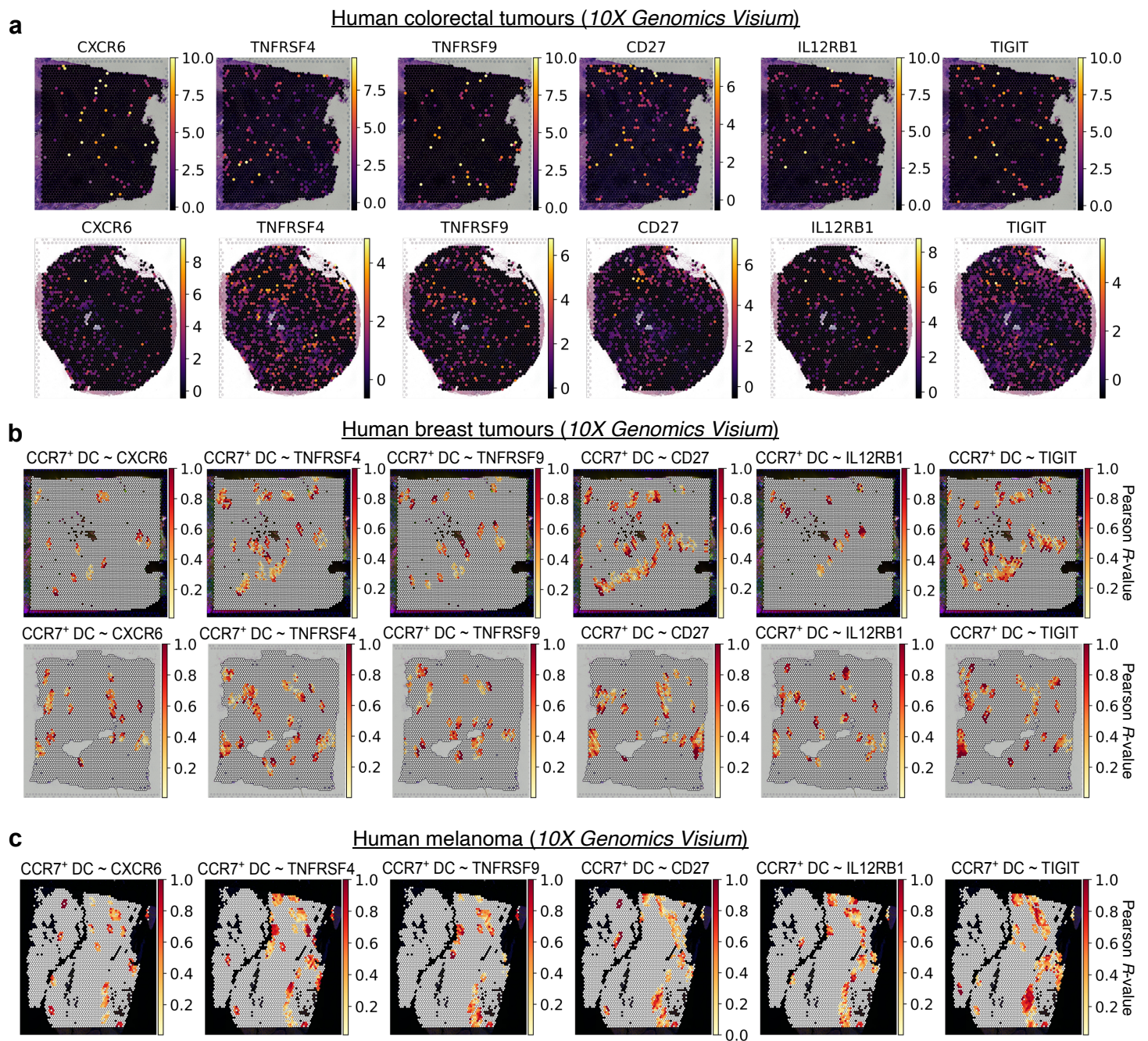
Supplementary Figure 11



Supplementary Figure 11 | CCR7⁺ DC heterogeneity and ligand-receptor interactions with CD8⁺ T cells in human tumours.

(a) UMAP of myeloid cells from scRNA-seq of human CRC⁴² ($n = 63$ patients) with paired tumour (T), normal adjacent tissue (N) and dLN samples, and expression of CCR7⁺ DC signature genes (b). (c) UMAP of scRNA-seq of CCR7⁺ DCs from (a), coloured by tissue cells were retrieved from. (d) Expression of *CCR7* and *ICOSLG* in CCR7⁺ DCs by tissue. (e) Expression of selected genes in CCR7⁺ DCs from (c). Molecules associated with tumour-residing CCR7⁺ DCs in mice were also preferentially expressed in tumour CCR7⁺ DCs in human CRC, but not the dLN. (f) Pearson correlation between CCR7⁺ DC signature genes and effector CD8⁺ T cell signature genes in bulk RNA-seq of 208 mUC tumours treated with atezolizumab (IMvigor 210 trial⁴⁴). Left, middle and right panel show all patients, clinical responders, and non-responders respectively. Points represent individual patient samples. (g) Expression of CCR7⁺ DC signature genes in myeloid cells from scRNA-seq of human mUC⁴⁶ ($n = 11$ patients). (h) UMAP of scRNA-seq of CCR7⁺ DCs from human breast cancer²⁴ (T cell clonotype expanders (E), i.e. responders, $n = 9$ patients; non-expanders (NE), i.e. non-responders, $n = 20$ patients; total $n = 29$ patients). (i) GSEA of CCR7⁺ DCs from responders versus non-responders, in breast tumours treated with anti-PD-1 antibodies. P-adj, Benjamini-Hochberg-adjusted p values. (j) Gene signature scores for “Hallmark TNFa response via NFkB” in PICseq of NSCLC³⁸, grouped by the myeloid cell identity in each myeloid-CD8⁺ T cell doublet. (k) Gene signature scores of selected pathways in myeloid-CD8⁺ T cell physically interacting doublets, grouped by the myeloid cell identity. Two-sided Wilcoxon rank-sum test with Benjamini-Hochberg multiple-testing correction (d), two-sided Pearson correlation (f), or two-sided Wilcoxon rank-sum test (j) was used. Data are shown as box (median; box, 25th percentile and 75th percentile; whiskers, 1.5*inter-quartile range) and violin plots (d, j), or linear regression with 95% confidence interval (f).

Supplementary Figure 12



Supplementary Figure 12 | Visium spatial transcriptomics of human cancer.

(a) Expression of selected receptors expressed by CD8⁺ T cells which facilitate interactions with tumour-residing CCR7⁺ DCs in spatial transcriptomics (10X Genomics Visium) of independent human CRC tumour sections ($n = 2$). (b-c) Spatial correlation (Pearson R-value) of CCR7⁺ DC signature scores and selected CCR7⁺ DC-ligand receptors expressed by CD8⁺ T cells, in spatial transcriptomics of independent human breast tumour sections (b, $n = 2$) and a human melanoma section (c, $n = 1$).

Supplementary table 1: Antibodies and reagents

Target	Fluorophore	Clone	Supplier	Catalogue no.	Dilution	Application
CCR7	AF647	EPR23192-57	Abcam	ab275165	1/100	Microscopy
MHC Class II I-A/I-E	Pacific Blue	M5/114.15.2	BioLegend	107620	1/50	Microscopy
CD3	AF488 / AF647	17A2	BioLegend	100209 / 100210	1/100	Microscopy
CD8a	PE	53-6.7	BioLegend	100707	1/50	Microscopy
CD31	AF594	MEC13.3	BioLegend	102520	1/100	Microscopy
4-1BB	-	AF6-120.1	R&D Systems	AF937	1/200	Microscopy
Ki-67	PE	SolA15	ThermoFisher	12-5698-82	1/50	Microscopy
Donkey anti-goat	AF647	Polyclonal	ThermoFisher	A32849	1/200	Microscopy
CD16/32	-	2.4G2	BioLegend	101302	1/100	Flow
LIVE/DEAD™ Viability dye	APC-Cy7 / NIR	-	ThermoFisher	L10119 / L34981	1/500	Flow
Viability dye	VK808	-	Beckman Coulter	C36628	1/250	Flow
CellTrace Violet	-	-	ThermoFisher	C34571	1/1000	Flow
CD45	BV785 / BUV395	30-F11	BioLegend / BD	103149 / 564279	1/200	Flow
CD11c	AF647 / AF700 / BV650	N418	Invitrogen	56-0114-82 / 416-0114-82	1/200	Flow
CD11b	PE-594 / BV785 / FITC	M1/70	BioLegend	101255 / 101243 / 101205	1/200	Flow
XCR1	AF647 / BV510	ZET	BioLegend	148213 / 148218	1/200	Flow
Ly6C	BV421 / BV711	HK1.4	BioLegend / BD	128031 / 755195	1/250	Flow
MHC Class II I-A/I-E	Pacific Blue / BV510	M5/114.15.2	BioLegend	107620 / 107636	1/200	Flow
CCR7	PE-Cy7 / PE	4B12	Invitrogen	12-1971-82	1/100	Flow
PD-L1	BV711 / BV605	10F.9G2	BioLegend	124319 / 124321	1/100	Flow
PD-L2/CD273	BUV395	TY25	BD Horizon	565102	1/200	Flow
CD40	FITC	HM40-3	ThermoFisher	11-0402-82	1/100	Flow
CD155 (PVR)	PE-Cy7	TX56	BioLegend	131511	1/100	Flow
F4/80	BV605	BM8	BioLegend	123133	1/100	Flow
Ly6G	BV650	1A8	BioLegend	127641	1/200	Flow
CD3	BV650 / FITC	17A2	BioLegend	100229 / 100203	1/100	Flow
CD3e	FITC / BV605	145-2C11	BD / BioLegend	100305 / 100351	1/100	Flow
CD8a	BV510 / BV711	53-6.7	BioLegend	100751 / 100747	1/200	Flow
CD8b	APC-Cy7	YTS156.7.7	BioLegend	126619	1/200	Flow
B220	BV650	RA3-6B2	BioLegend	103241	1/200	Flow
NK1.1	BV650	PK136	BD	564143	1/200	Flow
PD-1	BV421 / APC / BV605	29F.1A12	BioLegend	135217 / 135209 / 135219	1/250	Flow
Ki-67	PE-Cy7	SolA15	eBioscience	25-5698-82	1/200	Flow
Granzyme B	AF700	QA16A02	BioLegend	372222	1/200	Flow
IFNγ	BUV737	XMG1.2	BD	612769	1/200	Flow
CD62L	PE-Cy7	MEL-14	BioLegend	104418	1/200	Flow
CD44	PE	IM7	ThermoFisher	12-0441-82	1/400	Flow
CD25	PerCP-Cy5.5	PC61.5	Invitrogen	45-0251-82	1/200	Flow
Human CD4	APC	RPA-T4	BioLegend	300514	1/100	Flow
Human CD4	BV711	OKT4	BioLegend	317439	1/100	Flow
CD45	BV785 / BUV395	30-F11	BioLegend	103149 / 564279	1/200	FACS
CD11b	eFluor 450 / BV785	M1/70	Invitrogen / BioLegend	48-0112-82 / 101243	1/200	FACS
Ter119	PE-Cy7	TER-119	Invitrogen	25-5921-82	1/250	FACS
LIVE/DEAD™ Viability dye	APC-Cy7	-	ThermoFisher	L10119	1/500	FACS
NK1.1	BV650	PK136	BD	564143	1/200	FACS
B220	BV421	RA3-6B2	BioLegend	103239	1/200	FACS
CD11c	AF700 / PE	N418	Invitrogen	56-0114-82 / 12-0114-82	1/200	FACS
CD3e	FITC	145-2C11	BioLegend	100305	1/100	FACS
CD8b	APC-Cy7	YTS156.7.7	BioLegend	126619	1/200	FACS
CD62L	PE-Cy7	MEL-14	BioLegend	104418	1/200	FACS
CD44	PE	IM7	ThermoFisher	12-0441-82	1/400	FACS
MHC Class II I-A/I-E	Pacific Blue / BV510	M5/114.15.2	BioLegend	107620 / 107636	1/200	FACS
PD-L2/CD273	BUV395	TY25	BD Horizon	565102	1/200	FACS