Supplementary Information

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1. Materials and methods

Multiple myeloma cell culture

Mononuclear cells were isolated from fresh BM aspirates by density gradient centrifugation using Ficoll-Paque™ PLUS (GE Healthcare Bio-Sciences AB, Sweden). $CD138^+$ MM cells were selected by magnetic beads and columns according to the manufacturer's instructions (Miltenyi Biotec, Auburn, CA). All MM cell lines and primary MM cells were cultured in RPMI 1640 medium (Gibco, UK) supplemented with 10% heat-inactivated FBS (Corning, Manassas, VA), 2 mM L-Glutamine (Gibco, Grand Island, NY), 100 units/ml penicillin and 100 µg/ml streptomycin (Gibco, Grand Island, NY).

Generation of CRISPR modified cells

To generate $CD38^{KO}$ and $CD16^{KO}$ NK cells, we used crisprRNA (crRNA) (5-CTGAACTCGCAGTTGGCCAT) targeting the exon 1 of the CD38 gene[33] and crRNA (5-AAAGAGACTTGGTACCCAGG) targeting the exon 5 of CD16A gene. Generating Cas9/RNP complex has been described previously[27, 34]. In brief, pre-transcribed Alt-R® CRISPR-Cas9 crRNAs and Alt-R® CRISPR-Cas9 tracrRNA (Catalog# 1072532) were purchased from IDT (Integrated DNA Technologies, Inc., Coralville, Iowa). Guide RNA (gRNA) was prepared by incubating 200 µM each of crRNA and tracrRNA together in a total volume of 10 µl in Nuclease-Free IDTE, pH 7.5 (1X TE solution, Catalog # 11-01-02-02) at 95 ℃ for 5 minutes. The Cas9/RNP complex was formed by incubating 2 µl of Alt-R® S.p. HiFi Cas9 Nuclease V3 protein (122 pmol) (Catalog# 1081060), 2 µl of gRNA (400 pmol), and 1 µl of PBS in a total volume of 5 µl for 15-20 minutes at room temperature. Day 7-expanded NK cells were resuspended in 20 µl of P3 Primary Cell 4D-Nucleofector™ X Solution and 5 µl of Cas9/RNP complex and 1 µl of 100µM of Alt-R®

Cas9 Electroporation Enhancer (Catalog# 1075915), and electroporated using Lonza 4D-Nucleofector system with pulse EN-138. Wild type NK ($CD38^{WT}$ NK) cells were electroporated without Cas9/RNP complex. After electroporation, the NK cells were rested for 2 days in AIM-V/ICSR growth medium supplemented with 50 IU of rIL-2 before assessing the efficiency of CRISPR modification using flow cytometry. The cells were then expanded with CSTX002. Residual $CD38^{+}$ NK cells were removed by labeling with biotinylated anti-CD38 antibody (BioLegend) followed by anti-biotin microbeads (Miltenyi Biotec, Auburn, CA) and depletion on an LD column (Miltenyi Biotec, Auburn, CA).

Identifying off-target effects of CD38-targeted Cas9/RNP

Whole genome sequencing was used to identify the off-target effects of Cas9/RNP targeted to CD38. Genomic DNA (gDNA) was isolated form $CD38^{WT}$ and $CD38^{KO}$ NK cells using DNeasy Blood and Tissue Kit (Qiagen, Cat No./ID: 69504). DNA libraries were constructed using NEBNext Ultra II-FS DNA Library Prep Kit (New England Biolabs, Ipswhich MA). Samples were enzymatically fragmented, 5´ phosphorylated, dA-Tailed, and ligated with a unique, dual indexed adapter approach to prevent sample mis-assignment and resolve index hopping (Integrated DNA Technologies, Iowa). The adaptor-ligated DNA was amplified by limit-cycle PCR and purified using a magnetic-bead based approach. Library quality was analyzed on Tapestation High-Sensitivity D1000 ScreenTape (Agilent Biotechonologies) and quantified by KAPA qPCR (KAPA BioSystems). Libraries were sequenced at 2 x 150 bp read lengths to a depth of ~30X coverage on the Illumina HiSeq4000 platform.

Conjugation assay

Each NK cells were stained with 5 μM of green dye CFDA SE (CFSE) cell tracer (Invitrogen, Eugene, Oregon) for 15 minutes at 37℃ or 5 μM red dye PKH26 (Sigma-Aldrich, St. Louis, MO) for 5 minutes at room temperature. Staining was stopped by adding complete medium to the cell suspension. The cells were washed twice with complete medium. The green and red labeled NK cells were mixed at the ratio of 1:1 in 200 μl of total volume supplemented with 10 μg/ml of DARA or solvent control (saline) and co-cultured at 37℃ in a 5% CO_2 incubator for 4 hours. Then the cells were gently collected and fixed with 200 μl of 4% formaldehyde and 20,000 cells were analyzed for conjugation using flow cytometry.

Flow-based killing assay

To assess DARA-mediated ADCC, MM cell lines or purified primary $CD138^+$ MM cells were labeled with 5 μM of CFSE and co-cultured with $CD38^{WT}$ or $CD38^{KO}$ NK cells at the indicated effector-to-target (E:T) ratios in flat bottom 96-well plates (Falcon, USA) in the presence of 10 μg/ml of DARA or solvent as control. Due to low frequency of DARA-resistant primary samples ($CD38^{negative/low}$), we did not purify these cells and MM cells were defined as $CD138^+CD45^-$ cells. The viability of target cells was analyzed after 4 hours for MM cell lines, and 24 hours for primary samples. In some experiments, myeloma cell lines were pretreated with 50 nM of ATRA for 2 days prior to 4-hour cytotoxicity assay. To study the effect of ATRA on overall DARA-mediated NK cell cytotoxicity, MM cells and NK cells were co-cultured for 48 hours in the presence of DARA and 50 nM of ATRA. Viable target cells were assessed from the percent or absolute number of 7-AAD negative/CFSE positive cells among total CFSE positive cells or using beads. Background was determined from the target cells incubated in the absence of effector cells and DARA. The percentage of DARA-mediated ADCC (%) was calculated according

to the formula: (1- the percent or absolute number of viable target cells in the presence of effector cells with DARA / that of the corresponding sample with solvent control) × 100. All assays were performed in triplicate with 2 or 3 independent donors.

RNA sequencing and Ingenuity Pathway Analysis

Strand-specific RNA-seq libraries were prepared using NEBNext Ultra II Directional RNA Library Prep Kit, following the manufacturer's recommendations (New England Biolabs, Ipswhich MA). In summary, total RNA isolated from same donors (n = 6) $CD38^{WT}$ and $CD38^{KO}$ NK cells, (total of 12 samples) quality was assessed using RNA 6000 Nano kit on Agilent 2100 Bioanalyzer (Agilent Biotechnologies) and concentration measured using Qubit RNA HS assay kit (Life Technologies). A 40-500 ng aliquot of total RNA was rRNA depleted using NEB's Human/Mouse/Rat RNAse-H based Depletion kit (New England BioLabs). Following rRNA removal, mRNA was fragmented and then used for first- and second-strand cDNA synthesis with random hexamer primers. Double stranded cDNA fragments underwent end-repair and a-tailing and ligation of dual-unique adapters (Integrated DNA Technologies). Adaptor-ligated cDNA was amplified by limit-cycle PCR and purified using a magnetic-bead based approach. Library quality was analyzed on Tapestation High-Sensitivity D1000 ScreenTape (Agilent Biotechonologies) and quantified by KAPA qPCR (KAPA BioSystems). Libraries were pooled and sequenced at 2 x 150 bp read lengths on the Illumina HiSeq 4000 platform to generate approximately 60-80 million paired-end reads per sample. We next used the normalized RNA-seq data and filtered before input to Ingenuity Pathway Analysis (IPA) by eliminating genes that were not expressed at greater than or equal to 10 FPM in at least one sample. Differentially-expressed genes (DEGs) were identified as those in which a paired two-

sided t-test of gene expression levels between $CD38^{WT}$ and $CD38^{KO}$ NK cells yielded a p-value of less than 0.05. Adjusting the p-value cutoff for DEGs to 0.01 or 0.1, or adjusting the minimum gene expression cutoff to 5 FPM, does not qualitatively affect conclusions. The mean fold changes of each gene across $CD38^{WT}$ and $CD38^{KO}$ NK cells are approximately equal to the fold changes of the means in the reported pathways, so inter-individual effects ($CD38^{WT}$ and $CD38^{KO}$ NK cells from the same donors) can be considered negligible for these conclusions. All default settings for a core analysis in IPA were implemented. We were not able to study the transcriptomic profile of $CD38^{WT}$ and $CD38^{KO}$ NK cells in presence of DARA, as the $CD38^{WT}$ NK cells are killed by DARA-induced fratricide.

2. Supplementary figure legends

Figure S1. Immunophenotype of ex vivo expanded NK cells.

(A) The purity of NK cells 14 days after stimulation is shown. (B) Representative FACS analyses of CD16 expression on $CD38^{WT}$ and $CD38^{KO}$ NK cells of the same donor are shown. Each figure indicates the percentage of CD16 expressing NK cells. Isotype controls are depicted with filled histogram.

Figure S2. Expression of genes affected by Cas9/RNP.

Relative mRNA expression by RNA-seq of highly-affected genes from Table S3.

Figure S3. Biological features of $CD16^{KO}$ NK cells.

(A) FACS analyses of $CD16^{WT}$ and $CD16^{KO}$ NK cells. (B) The viability of each $CD16^{WT}$ and $CD16^{KO}$ NK cells treated with DARA (10μg/ml) for 4 or 24 hours compared to that of control samples is shown. $CD16^{KO}$ NK cells show no evidence of fratricide. C-D) H929 cell line was incubated with $CD16^{WT}$ or $CD16^{KO}$ NK cells in the presence or absence of DARA (10μg/ml) for 4 hours. The viability compared to control sample and ADCC activity of paired $CD16^{WT}$ and $CD16^{KO}$ NK cells are shown.

Figure S4. Dependence of DARA-mediated lysis of MM cells on effector NK cells.

Each MM cell line was incubated with DARA (10μg/ml) with or without $CD38^{KO}$ NK effector cells (E/T = 5) for 4 hours. The viability compared to each control sample are shown. The experiment was performed in the presence of heat inactivated serum and thus in the absence of complement.

Figure S5. CD38 expression of MM cells from a relapsed case during DARA treatment. BM mononuclear cells from a DARA-resistant patient and healthy donor-derived $CD38^{WT}$ and $CD38^{KO}$ NK cells were stained with multi-epitope anti-CD38 antibodies. MM cells were defined by expression of CD138. MFI (CD38) of stained samples and fluorescence minus one (FMO) (without multi-epitope anti-CD38 antibodies) controls are shown.

Figure S6. Relative advantage of $CD38^{KO}$ NK cells compared to $CD38^{WT}$ NK cells as effectors for DARA-ADCC compared to expression level of CD38 on the MM cell targets.

X axis indicates the relative ratio of MFI (CD38) of target cells to $CD38^{WT}$ NK cells. Y axis indicates the relative increase in ADCC of $CD38^{KO}$ NK cells compared to that of $CD38^{WT}$ NK cells. The values of ADCC (E/T = 5, in 4 hour-assay) are used for MM.1S, H929, and OPM-2, and those (E/T = 0.1, in 24 hour-assay) are used for RPMI 8226 and patient samples. Spearman's rank-correlation coefficients (*r*) and *p* value are presented.

Figure S7. CD38 expression on MM cell lines 48 hours after incubation with ATRA. Control and ATRA treated samples are shown with black and gray line respectively. Unstained controls are depicted with filled histogram.

Figure S8. Volcano plot of normalized RNA-seq data of 6 different pairs of $CD38^{WT}$ and $CD38^{KO}$ NK cells.

The most significantly changed genes in $CD38^{KO}$ NK cells compared to $CD38^{WT}$ NK cells are shown.

3. Supplementary tables

Table S1. Character of clinical samples.

	Age	Subtype	Frequency of plasma cells*	ISS	Chromosomes	Treatment history	Last DARA infusion
Patient-1	75	IgG lambda myeloma	50%	NA	nuc ish(D3Z1,CCND1-XT)x3 (D9Z1,D15Z4)x3−4	naïve	none
Patient-2	67	IgG lambda myeloma	70-80%	1	t(11;14) and 11q-	PI, IMiD, autologous SCT, DARA XPO1 inhibitor, BCL2 inhibitor, anti-BCMA A	22 months before
Patient-3	47	IgG kappa plasmacytoma	<5%	1	unkown	naïve	none
Patient-4	73	IgA lambda myeloma	10-15%	1	1q+, 1p-, 6+, 9+, 15+ IgH translocation without usual partne	naïve	none
Patient-5	57	IgA kappa myeloma	40-50%	3	1q+, t(4;14), -13 myc disruption	naïve	none
DARA-resistant patient	42	IgG lambda myeloma	<1%	3	+1q, monosomy 13, del 14	PI, autologous SCT, IMiD, DARA	10 days before

NA indicates not applicable. *Frequncy of plasma cells are aseessed in biopsy samples.

Table S2. Antibodies list.

Brand	*Reactivity*	*Name*	*Clone*	*Format*
BD	*Human*	*CD3*	*SK7*	*FITC*
BD Pharmingen	*Human*	*CD16*	*3G8*	*PE*
BD Pharmingen	*Human*	*CD16*	*3G8*	*Alexa Fluor 647*
BD Pharmingen	*Human*	*CD19*	*SJ25C1*	*APC-Cy7*
BD Horizon	*Human*	*CD33*	*WM53*	*BV421*
BD	*Human*	*CD38*	*HB7*	*PE*
Biolegend	*Human*	*CD38*	*HB7*	*APC*
Biolegend	*Human*	*CD38*	*HIT2*	*Biotin*
Cytognos	*Human*	*CD38*	*Multi-epitope*	*FITC*
BD Pharmingen	*Human*	*CD56*	*B159*	*Alexa Fluor 647*
BD Pharmingen	*Human*	*CD138*	*MI15*	*PE*
BD Pharmingen	*Human*	*CD45*	*HI30*	*APC*
BD Horizon	*Human*	*CD45*	*HI30*	*BV421*
BD Pharmingen	*Mouse*	*CD45*	*30-F11*	*FITC*
Biolegend	*Mouse*	*FITC*	*FIT-22*	*APC*

Supplementary Table S3.

Gene	Chr	position	Variant Allele frequency in $CD38^{KO}$ NK	Variant Allele frequency in $CD38^{WT}$ NK	Ref	Alt	Filter	Loc In Gene	Effect	Impact
ANKRD36C	2	96517484	6.90%	0.00%	T	G	clustered_events,t_lod,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
APOBEC3H	22	39497425	19.05%	0.00%	T	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE
CC2D1B	1	52825875	8.00%	0.00%	T	TC	clustered_events,t_lod	coding_sequence	frameshift	HIGH
CC2D1B	1	52825879	7.69%	0.00%	C	CAG	clustered_events,t_lod	coding_sequence	frameshift	HIGH
CC2D1B	1	52825881	7.69%	0.00%	C	CCTTGGAGTCGCAGCTAGCCTCTGTGAGG	clustered_events,t_lod	coding_sequence	frameshift&stopgain	HIGH
CCDC181	1	169388367	19.05%	0.00%	C	T	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
CCDC181	1	169388373	18.18%	0.00%	C	T	clustered_events,base_quality	coding_sequence	nonsynonymous SNV	MODERATE
CD38	4	15780022	12.00%	0.00%	CCCCGCCTGGAGCC	C	clustered_events	coding_sequence	startloss&nonframeshift deletion	HIGH

					CTATGGCCAACTGCGAGTTCAGCCCGGTGTCCGGGGACAAA					
CD38	4	15780032	31.25%	0.00%	AGCCCTATG	A	clustered_events	coding_sequence	frameshift&startloss	HIGH
CD38	4	15780039	13.33%	0.00%	TG	T	clustered_events,t_lod	coding_sequence	frameshift	HIGH
CD38	4	15780040	12.50%	0.00%	GGCCAA	G	clustered_events	coding_sequence	frameshift	HIGH
CR1	1	207782852	8.00%	0.00%	T	TTCTGG	clustered_events,t_lod	coding_sequence	frameshift	HIGH

CR1	1	207782853	8.00%	0.00%	A	AGCTGTGC	clustered_events,t_lod	coding_sequence	frameshift	HIGH
CR1	1	207782855	8.33%	0.00%	T	TTTATTAGTAG	clustered_events,t_lod	coding_sequence	frameshift	HIGH
CR1	1	207782860	7.69%	0.00%	G	A	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
DENND4B	1	153903191	8.70%	0.00%	TG	T	clustered_events,t_lod	coding_sequence	frameshift	HIGH
DENND4B	1	153903197	8.33%	0.00%	CCCTAGAGTT	C	clustered_events,t_lod	coding_sequence	nonframeshift deletion	MODERATE
DENND4B	1	153903210	10.53%	0.00%	A	T	clustered_events,base_quality,read_position	coding_sequence	nonsynonymous SNV	MODERATE
DENND4B	1	153903213	9.52%	0.00%	A	T	clustered_events,base_quality,read_position	coding_sequence	nonsynonymous SNV	MODERATE
DENND4B	1	153903216	11.11%	0.00%	A	AAACTCTAGG	clustered_events,base_quality,read_position	coding_sequence	stopgain&nonframeshift insertion	HIGH
FGF7	15	49776539	12.50%	0.00%	A	T	PASS	coding_sequence	nonsynonymous SNV	MODERATE
FGFR2	10	123244918	10.53%	0.00%	G	GGGCACCGGCAGGAAAGACAAC	PASS	coding_sequence	nonframeshift insertion	MODERATE
FGFR2	10	123244922	10.53%	0.00%	A	ACCAACGAACTGTAAGGGCT	PASS	coding_sequence	frameshift	HIGH
FHOD1	16	67270885	10.53%	0.00%	G	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE

FOXP3	X	49107814	9.09%	0.00%	T	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE
KMT2C	7	151962134	9.76%	0.00%	G	T	PASS	coding_sequence	stopgain	HIGH
MT-ATP6	MT	9181	1.33%	0.00%	A	G	PASS	coding_sequence	nonsynonymous SNV	MODERATE
MYH4	17	10352331	10.00%	0.00%	T	G	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
MYH4	17	10352336	8.70%	0.00%	C	CAGG	clustered_events,t_lod,read_position	coding_sequence	nonframeshift insertion	MODERATE
MYH4	17	10352338	8.70%	0.00%	T	TGCAGAAGAA	clustered_events,t_lod,read_position	coding_sequence	nonframeshift insertion	MODERATE
MYH4	17	10352340	9.09%	0.00%	C	A	clustered_events,base_quality,t_lod,read_position	coding_sequence	nonsynonymous SNV	MODERATE
NBPF14	1	148252777	15.38%	0.00%	T	TCTC	PASS	coding_sequence	nonframeshift insertion	MODERATE
OR2T4	1	248525100	12.50%	0.00%	G	A	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
OR2T4	1	248525135	11.11%	0.00%	G	A	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
OR2T4	1	248525138	13.79%	0.00%	C	T	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101718932	7.41%	0.00%	C	G	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101718968	7.14%	0.00%	C	T	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101721705	8.33%	0.00%	G	T	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101721709	8.33%	0.00%	T	A	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE

PABPC1	8	101721812	11.54%	0.00%	G	A	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101721817	10.71%	0.00%	T	C	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC1	8	101721839	10.34%	0.00%	C	A	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
FGFR2	10	123244918	10.53%	0.00%	G	GGGCACCGGCAGGAAAGACAAC	PASS	coding_sequence	nonframeshift insertion	MODERATE
FGFR2	10	123244922	10.53%	0.00%	A	ACCAACGAACTGTAAGGGCT	PASS	coding_sequence	frameshift	HIGH
PABPC3	13	25670851	9.38%	0.00%	A	G	PASS	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671545	6.67%	0.00%	C	A	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671549	9.38%	0.00%	A	G	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671555	9.68%	0.00%	G	A	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671558	9.38%	0.00%	C	T	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671564	10.34%	0.00%	A	T	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671573	15.63%	0.00%	CA	TG	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671585	18.75%	0.00%	TA	CC	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671699	12.50%	0.00%	GT	CC	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
PABPC3	13	25671742	7.14%	0.00%	G	A	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE

PABPC3	13	25671759	7.14%	0.00%	C	T	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
WDR89	14	64066367	10.00%	0.00%	A	T	clustered_events,t_lod	coding_sequence	nonsynonymous SNV	MODERATE
WDR89	14	64066453	14.29%	0.00%	G	A	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
WDR89	14	64066471	12.00%	0.00%	GT	AG	clustered_events,mapping_quality	coding_sequence	stopgain	HIGH
FGF7	15	49776539	12.50%	0.00%	A	T	PASS	coding_sequence	nonsynonymous SNV	MODERATE
FHOD1	16	67270885	10.53%	0.00%	G	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE
MYH4	17	10352331	10.00%	0.00%	T	G	clustered_events	coding_sequence	nonsynonymous SNV	MODERATE
MYH4	17	10352336	8.70%	0.00%	C	CAGG	clustered_events,t_lod,read_position	coding_sequence	nonframeshift insertion	MODERATE
MYH4	17	10352338	8.70%	0.00%	T	TGCAGAAGAA	clustered_events,t_lod,read_position	coding_sequence	nonframeshift insertion	MODERATE
MYH4	17	10352340	9.09%	0.00%	C	A	clustered_events,base_quality,t_lod,read_position	coding_sequence	nonsynonymous SNV	MODERATE
STXBP4	17	53237239	12.50%	0.00%	T	G	clustered_events,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
STXBP4	17	53237241	12.50%	0.00%	G	T	clustered_events,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
STXBP4	17	53237250	13.04%	0.00%	A	C	clustered_events,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
SIGLEC10	19	51919263	11.11%	0.00%	G	T	clustered_events,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
SIGLEC10	19	51919279	12.50%	0.00%	A	G	clustered_events,mapping_quality	coding_sequence	startloss	LOW

SIGLEC10	19	51919302	9.52%	0.00%	T	C	clustered_events,mapping_quality	coding_sequence	nonsynonymous SNV	MODERATE
APOBEC3H	22	39497425	19.05%	0.00%	T	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE
FOXP3	X	49107814	9.09%	0.00%	T	A	PASS	coding_sequence	nonsynonymous SNV	MODERATE
MT-ATP6	MT	9181	1.33%	0.00%	A	G	PASS	coding_sequence	nonsynonymous SNV	MODERATE

Figure S1.

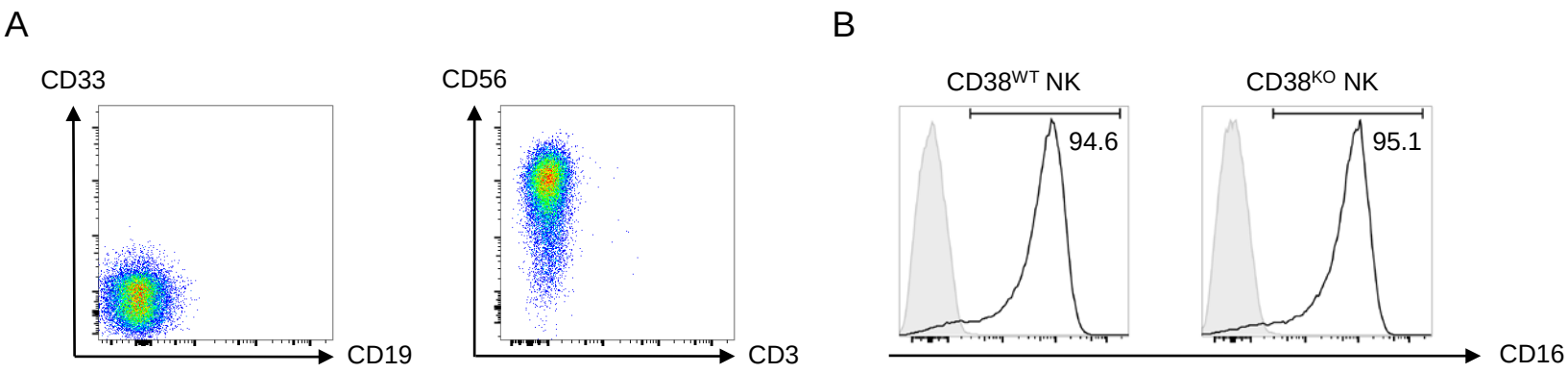


Figure S2.

Figure S3.

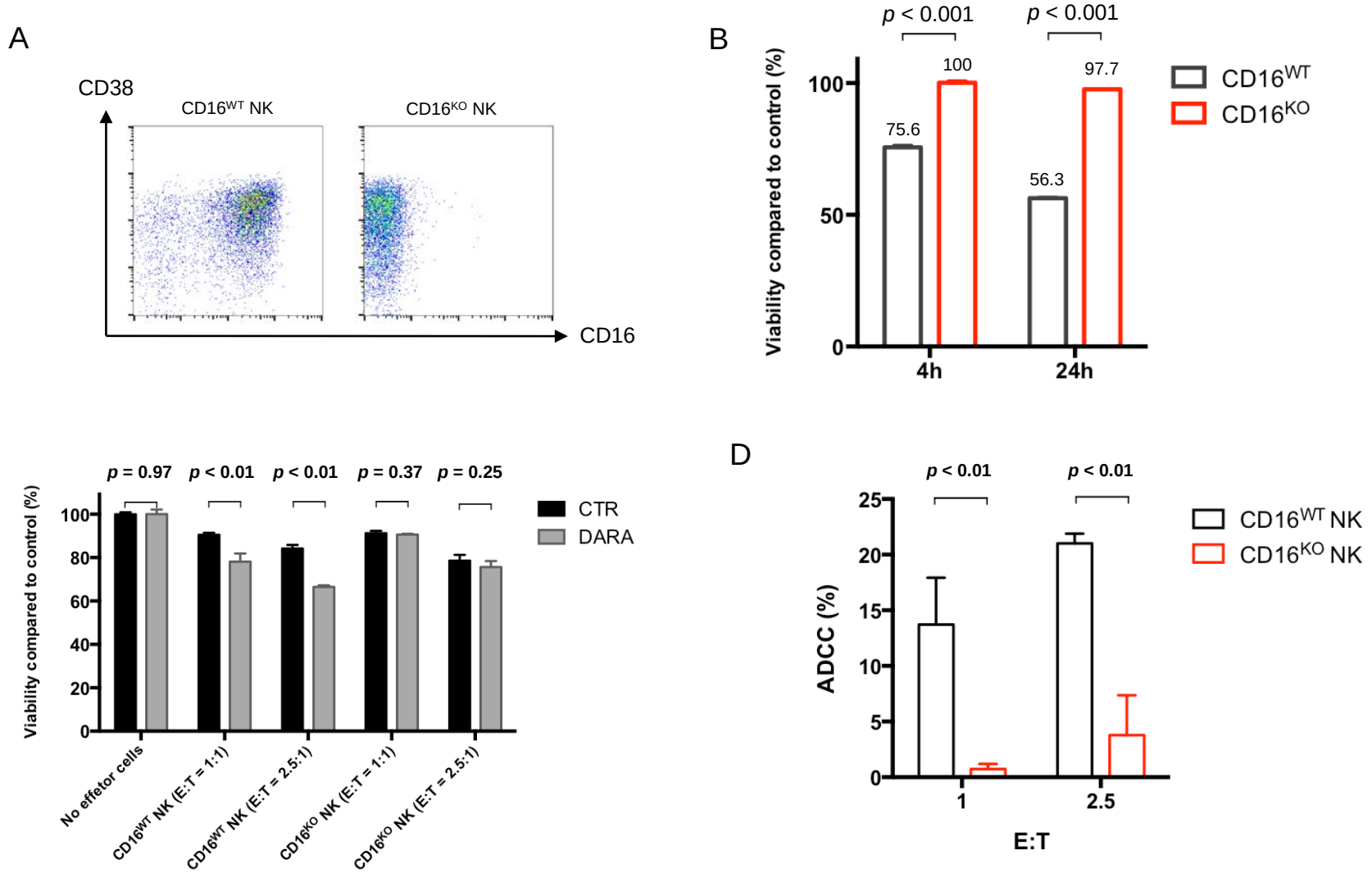
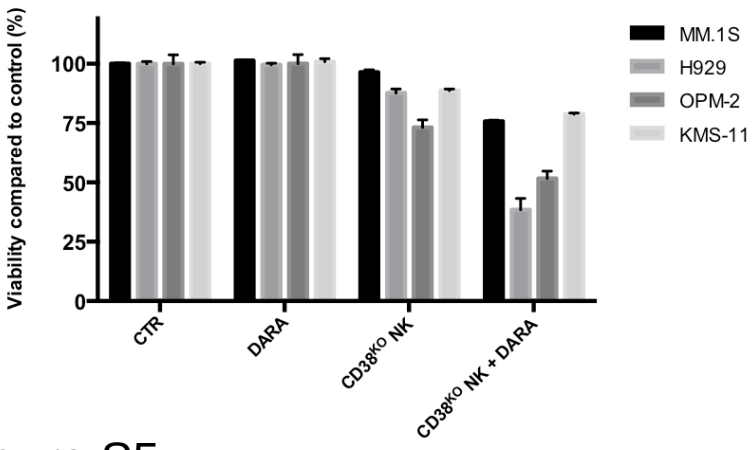


Figure S4.



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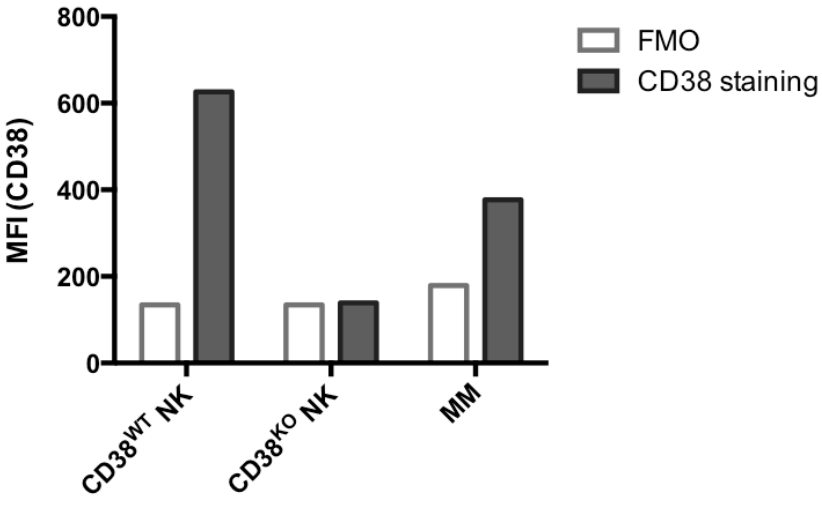

Figure S6.

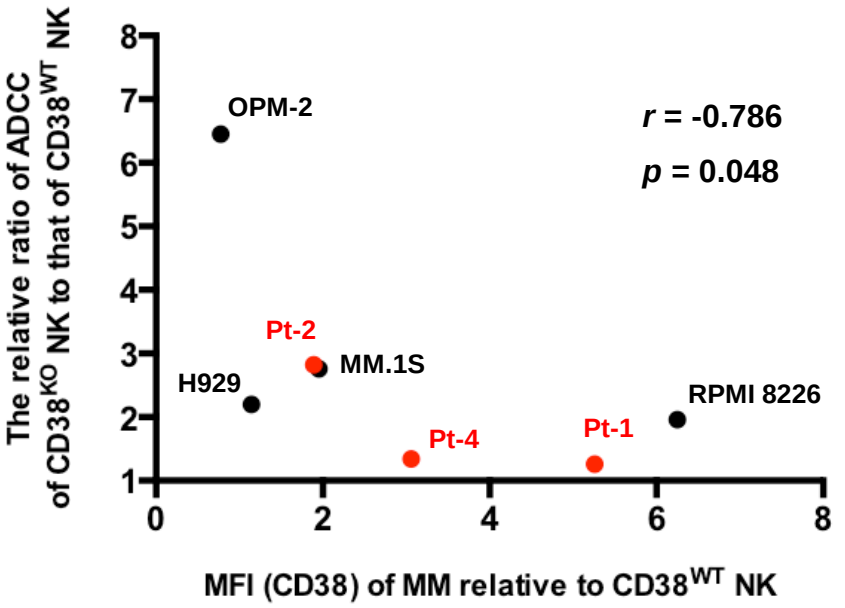

Figure S7.

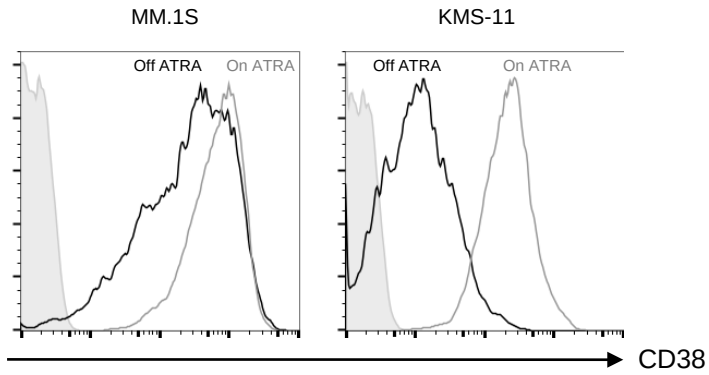

Figure S8.