SUPPLEMENTAL FIGURES AND TABLES LEGENDS

Supplemental Figure 1. Controls for cellular senescence and exogenous modifications. (**A**) Cells were considered presenescent (PRE) if, when subconfluent in 10% serum, the labeling index (L.I.) of >70% of cells incorporated BrdU over a 1 d interval and <5% stained for SAβgal. Cells were considered senescent (SEN) if <10% of cells incorporated BrdU over a 1 d interval and >80% were SA-βgal positive (1). Average and standard deviation around the mean are shown. Average % SA-βgal and BrdU incorporation were calculated from values across all cell types per indicated conditions (see **Supplemental Table 1** for cell list). (**B**) Detection of p53 by immunofluorescence. Inactivation of p53 was achieved by stable over expression of genetic suppressor element GSE22 as previously described (2). Intracellular accumulation of p53 due to GSE22 expression indicates p53 inactivation. Loss of p53 was induced either before or after senescence (XRA or REP). Quantified p53-intensities per nuclei are provided (bar graphs on the right; averages). Normal fibroblasts can senescence without a functional p53 (3). (**C**) Immunofluorescence detection of $p16^{INK4a}$ and IL6 in WI-38 and IMR-90 cells expressing an shRNA against $p16^{INK4a}$ previously validated in (4). IL6 expression serves as control of SASP development. Normal fibroblasts can senescence without a functional p16 (5). (**D**) $p16^{INK4a}$ mRNA levels measured in presenescent and senescent cells (TaqMan assay). (**E**) 53BP1 foci were detected by immunofluorescence and scored as the number of cells with ≥3 foci, indicative of persistent DNA damage (6, 7) (ave ± s.e.). (**F**) ATM mRNA levels measured in shATM-expressing cells. The shRNA against ATM was previously validated in (6). Normal fibroblasts can senescence without a functional ATM. (**G**) $p21^{WAF1}$ mRNA levels measured in presenescent and senescent cells (TaqMan assay). (**H**) Immunofluorescence detection of $p16^{INK4a}$ in WI-38 and IMR-90 cells expressing ectopic $p16^{INK4a}$.

Supplemental Figure 2. NKG2D-ligands expression in senescent fibroblasts and epithelial cells. (**A**) Time course of cell surface expression of NKG2D-Ls (top immunofluorescence panels) and intracellular IL7 (bottom immunofluorescence panels) in WI-38 cells after XRA (10 Gy). Bottom graphs show levels of secreted factors at specific times after senescence induction, measured by antibody array. Secreted factors are color-coded as shown on the left. X indicates p53 deficient status; O indicates oncogenic RAS (HRAS(G12V)) expression; Δ indicates p53-deficient status and subsequent oncogenic RAS expression. We provide SASP factors known to impact innate immune responses (ILs, CXCLs, CCLs, CSFs).

(**B**) NKG2D-Ls expression in immortalized breast epithelial cells (MCF10A) and prostate cancer cells (PC-3, BPH1, DU145) senesced with XRA (10 Gy), MIT/mitoxantrone, or ETO/etoposide.

Supplemental Figure 3. Senescent cells are preferentially targeted by activated innate immune cytolytic cells via NKG2D/NKG2D-Ls detection and GRZB/PRF killing. (**A**) Cytolysis of WI-38 cells measured by LDH release after co-culture with individual leukocyte populations that were either unactivated or pre-activated. (**B**) Percent survival (1-%LDH release) of fibroblasts incubated with a recombinant NKG2D coupled to an Fc fragment (rNKG2D/Fc) prior to initiation of 12h co-cultures with IL2 pre-activated primary NK cells or PBMCs. The increase in SnCs killing in rec.NKG2D/Fc treated cultures, indicates that SnCs display NKG2D-Ls at their cell surface, and that their selective expression and presentation of NKG2D-Ls at their cell surface promotes their killing. This can be used to mediate antibody-dependent cell-mediated cytotoxicity (ADCC; (8)) and enhance NK cells' cytotoxicity against SnCs. (**C**) Percent survival of human prostate cancer cells and immortalized mammary epithelial cells induced to senesce by radiation (10 Gy (XRA)), or chemotherapy (mitoxantrone (MIT), etoposide (ETO)), and co-cultured with IL2 pre-activated primary NK or PBMCs. LDH release was measured at 14-20h after co-culture. (**D**) Blocking NKG2D receptor increases survival of senescent cancer cells, i.e. NKG2D-Ls recognition limits their survival/persistence. (**E**) Survival of SnCs co-cultured with IL2 pre-activated PBMCs in presence of a blocking antibody against DNAM1. LDH release was measured at 16-18h after co-culture. IgG/M served as a control. Blocking DNAM1-mediated cytolysis only partially prevents killing of SnCs. (**F**) Survival assessed by cell number in direct co-cultures, and indirect co-cultures (using trans-wells culture plates), after 10days using unactivated, freshly isolated PBMCs. A 1:10 ratio of target fibroblasts/effector PBMCs was used. (**G**) Granzyme B (GRZB) levels measured by ELISA in media after 9days of co-culture of PBMCs with SnCs. (**H**) Levels of immune cytokines TNFα and IFNγ were measured by ELISA in conditioned media collected after 7-9days of mono and co-cultures with PBMCs. Data from different fibroblasts (Wi-38, IMR-90, HCA2) were pooled. (**I**) Granzyme B (GRZB) levels after 10days in direct co-cultures with PBMCs and complemented with rec.NKG2D/Fc every 2days (dil. 1:100). Higher levels of GRZB release in the media of SEN(XRA) + PBMC cultures treated with rec.NKG2D/Fc indicates that expression of NKG2D-Ls mediate and promote the immune killing of SnCs. (**J**) Survival measured by LDH release to assess effects of perforin (PRF) inhibition by concanamycin A (ConA). Fibroblasts (WI-38, IMR-90, HCA2) and epithelial cancer cells (PC3) were co-cultured with activated ConA/mock pre-treated PBMCs and NKs.

Supplemental Figure 4. Balance between MMPs and NKG2D-Ls determine the fate of SnCs. (**A**) NKG2D-Ls (i) and MMPs (ii) mRNA levels 10 d after initiating control monocultures (naïve) or PBMC co-cultures (persistent). Data were calculated as percent normalized to GUS, then normalized to the fibroblast marker CD90. Expression of CD90, NKG2D-Ls and MMPs was either not detected or barely detectable in leukocytes (**Supplemental Table 2D**). (**B**) sMICA in media from fibroblasts (top; WI-38, IMR-90, HCA2) and epithelial cells (bottom; BPH1, RWPE1, PC3, DU145), detected by ELISA. n.d. = not detectable. (**C**) Survival assessed by LDH release in senescent prostate cancer cells treated with GM6001 prior to 10 h co-culture with IL2 pre-activated NK cells (Effector:Target ratio 1:4). Protease inhibitor treatment decreases the levels of immune persistence of SnCs.

Supplemental Figure 5. RAS-induced senescence entails an exaggerated immune-evasive phenotype. (**A**) Immunofluorescence of $p16^{INK4a}$ and IL6 in WI-38 (left) and IMR-90 (right) fibroblasts induced to senesce by oncogenic HRAS(G12V) expression. (**B**) Senescence-associated β-galactosidase activity (blue bars) and labeling index (red bars) in RAS SnCs. (**C**) Quantification of 53BP1 foci as in **Supplemental Figure 1E**. (**D**) Granzyme B (GRZB) levels measured by ELISA in media collected after 9 d co-culture of RAS SnCs with PBMCs. (**E**) MMP1, MMP3 and IL8 detected by immunofluorescence in p53-wild type, or p53-deficient, RAS WI-38 cells. (**F**) Secreted MMPs levels in media were determined by antibody arrays. Values were normalized for cell number per volume of medium, compared to PRE cells. (**G**-**H**) MICA and MICB levels were measured by ELISA in media from RAS-induced senescence in fibroblasts (WI-38, IMR-90, HCA2; panel (**G**)) and prostate cancer cells (BPH1, RWPE1; panel (**H**)). (**I**) Percent survival of SEN (RAS) and SEN (p53-deficient RAS) cells treated with GM6001 prior to 10-12 h co-culture with IL2 pre-activated NK cells (Effector:Target ratio 1:10). GM6001 treatment increases the immune clearance of RAS SnCs by up to 20% compared to baseline.

Supplemental Figure 6. Gene expression profiles of prostate tumors from patients treated or not with genotoxic therapy (mitoxantrone). (**A**) Expression of senescence-associated markers in tumors after mitoxantrone therapy. Schematic of patient treatment and collection of residual tumor after chemotherapy regimen is shown (left). Heatmap of unsupervised hierarchical clustering of expression profiles show mRNA levels of senescence-associated cell cycle arrest markers ($p16^{INK4a}$, $p21^{WAF1}$, PCNA, MCM3, cyclin A, Ki-67) and SASP factors (IL6, IL7, IL8/CXCL8, GROa/CXCL1, MCP2/CCL8, GM-CSF/CSF2, IL1β) measured in the individual patients' paired prostate cancer tissue before and after mitoxantrone

chemotherapy. (**B**) mRNA levels of NK/T-cells markers CD94 and CD8. Expression was measured by qRT-PCR in paired prostate cancer tissues before and after chemotherapy. (**C**) Quantification of CD57 (+) cells (NK cell marker) by immunohistochemistry on untreated and mitoxantrone-treated cancer tissues Tissue microarray (TMA) of unpaired tissue cases were used (same sets as in main **Figure 7B**). (**D**) Expression profiles for each patient (#1-10) paired tumor set. Expression profiles for NKG2D-Ls, MMPs and NKG2D are shown in the 3 bar graphs. Expression was normalized to pretreatment values for each gene and patient.

Supplemental Figure 7. Gene expression profiles of breast tumors from patients treated or not with either genotoxic/DNA-damaging therapy or targeted/endocrine therapy. (**A**) Analysis of microarray data from studies GSE23988, GSE20194 and E-TABM-43 (9-11), combined without batch effects. The comparison of 339 untreated patients (No Tx) to 37 patients treated with epirubicin/cyclophosphamide (EPR/CTX) is shown as a differential gene expression heatmap (higher in EPR/CTX than in untreated in red; lower in EPR/CTX than in untreated in green). FDR corrected p-values are displayed on a grey-scale. Probe sets for Affymetrix platforms and Entrez IDs are provided in **Supplemental Table 3B**. Change in expression levels for MMPs, NKG2D-Ls, NKG2D, NK/T cell markers (CD8/94/57/56), and senescence-associated growth arrest markers (p16, p21, cyclin A, PCNA, MCM3, Ki-67) are shown. (**B-C**) Analysis of microarray data from studies GSE16391, GSE13787, GSE22035 and GSE1446 (12-15), combined without batch effects. The comparison of 186 untreated patients to 55 patients treated with targeted therapy tamoxifen/letrozole (TMX/LET) is shown as a heatmap in (**B**) and box plots in (**C**). Affymetrix U133 plus 2.0 was used (probe sets for ULBP-3 and ULBP-4 were available and are included). Each box plot displays: the median (horizontal red line), first to third quartile range (Q1 to Q3, or interquartile range (IQR); blue box), minimum to maximum (dashed lines), outliers (red dots).

Supplemental Figure 8. Gene expression profile of human nevi and normal skin. (**A**) Box plots showing expression of 24 genes in normal skin and nevi. In each panel, the left box displays the distribution of expression across the 7 normal skin samples, while the right box displays the distribution of the expression across 18 nevi samples. Each box plot displays: the median (horizontal red line), first to third quartile range (Q1 to Q3, or interquartile range (IQR); blue box), minimum to maximum (dashed lines), outliers (red dots). (**B**) Heatmap of median level of expression of indicated genes in normal skin (baseline) and nevi (green-to-white-to-red color scale indicates expression levels lower-to-equal-to-higher compared to normal skin

expression levels). FDR p-values are displayed above each box plot in panel (**A**) or as a grey-scale on the right of the differential expression color-coded heatmap in panel (**B**). To ease comparisons of data between **Supplemental Figures 7 and 8**, we show results from the same probe set across the different platforms and studies.

Supplemental Table 1. Fibroblasts and epithelial cells used in this study. Normal human fibroblasts (WI-38, IMR-90, HCA2), and human epithelial cells of prostate (PC-3, DU145, BPH1, RWPE1) and mammary gland (MCF10A) origins, were cultured in 3% oxygen as described (16). Cells were induced to senesce by ionizing radiation (10 Gy X-ray), or DNA damaging agent treatment (mitoxantrone, or etoposide), or expression of oncogenic HRAS(G12V), $p16^{INK4a}$ or $p21^{CIP1/WAF1}$, and were fully senescent 10 to 12 d later (see **Supplemental Figure 1A**). Alternatively, normal fibroblasts were repeatedly sub-cultured until proliferation ceased (telomere shortening leading to replicative senescence, REP). To express $p16^{INK4a}$, $p21^{CIP1/WAF1}$, HRAS(G12V), shp16, shATM or GSE22 (peptide suppressor of p53 activity), cells were transduced with lentiviruses carrying no insert or GFP (control), or the indicated cDNAs or shRNA control (non-functional shRNA), as described (4, 6, 16). PC-3 and DU145 are prostate carcinoma cells, BPH1 is a benign, immortalized (SV40 large T) prostatic hyperplastic epithelial line (17), and RWPE1 are non-malignant, immortalized (HPV-18 E7) cells from normal prostate tissue (18). MCF10A are non-transformed mammary epithelial cells. Symbols: $p53^{-}$ (lenti-GSE overexpression (GSE22)(2)); $p16^{INK4a+}$ or $p21^{+}$ (lenti-p16 or p21 overexpression); * = total number of independent cell cultures used in the study; ** = subset of representative, independent cell culture experiments (annotated in parenthesis) used to compile senescence-associated characteristics (LI, SA-βGAL); *** = lenti- GSE, shp16, shATM, p16, p21, and HRAS(G12V) were defined and used as described previously (4, 6, 16); GSE expression induces p53 dysfunction; **** = not exogenously damaged; nt = not tested; na = not applicable (e.g. HCA2 are fibroblasts that require a functional p53 in order to senesce). Other notes: HCA2 are fibroblasts that senesce without significantly elevating p16 expression (4).

Supplemental Table 2. Gene expression profiles in presenescent and senescent cells. Average mRNA levels measured by TaqMan and normalized to GUS expression. Cell types, genetic modifications and treatments, culture conditions (3% or 20% O_2), and number of independent biological extracts/samples (n) tested in at least quadruplicate quantitative RT-PCR runs per individual sample and gene probe set, are indicated on the left. Gene names are listed at the top. Genes other than NKG2D-Ls and MMPs serve as control and/or additional reference

for comparison, to highlight the specificity of the functions of NKG2D-Ls and MMPs across different senescence modalities. (**A**) Cell surface ligands: NKG2D-Ls (MICA/B, ULBP-1/2/3), DNAM1-Ls (CD112, CD155), HLA-C/E. (**B**) Senescence-associated growth arrest and tumor suppressors p16, p21, p53 and additional control genes (GAPDH, H1A). (**C**) Extracellular matrix metallo-protease genes MMP1, MMP3, MMP10, MMP12, MMP8, MMP13, and other proteases: Erp5/PDIA6, ADAM17, MT1-MMP/MMP14, PSEN1. Collectively, results from panels (**A,B,C**) represent data from 1,600 different biological samples gene expression data points derived from >6,500 TaqMan assay readouts. (**D**) Expression of fibroblast-specific marker CD90 and key genes (MICA/B, ULBP-1/2/3, MMP-1/3), were compared between monocultures of fibroblasts or PBMCs. nt = not tested; nd = not detectable. Results in (D) show that the values observed when measuring expression of NKG2D-Ls in experiments were both cell types (fibroblasts and PBMCs) are present, come essentially from the fibroblast population, and that PBMCs do not contribute to those values.

Supplemental Table 3. Gene expression probes. (**A**) Probes used in TaqMan assays (prostate tumor data; see main **Figures 1A, 7A-D** and **Supplemental Figure 6**). (**B**) Probes used for data mining expression arrays (breast cancer and nevi data; see main **Figures 1B-C, 7E-I** and **Supplemental Figures 7-8**).

Supplemental Table 4. NIH/GEO accession numbers of the patient studies considered for analysis for breast cancer (A) and nevi (B). Enumeration of studies selected for bias analysis and then remaining (highlighted) for gene expression analysis, and whose results are presented in **Figures 1B-C, 7E-I** and **Supplemental Figures 7-8**. Gene Expression Omnibus: http://www.ncbi.nlm.nih.gov/geo/. ArrayExpress: http://www.ebi.ac.uk/arrayexpress/

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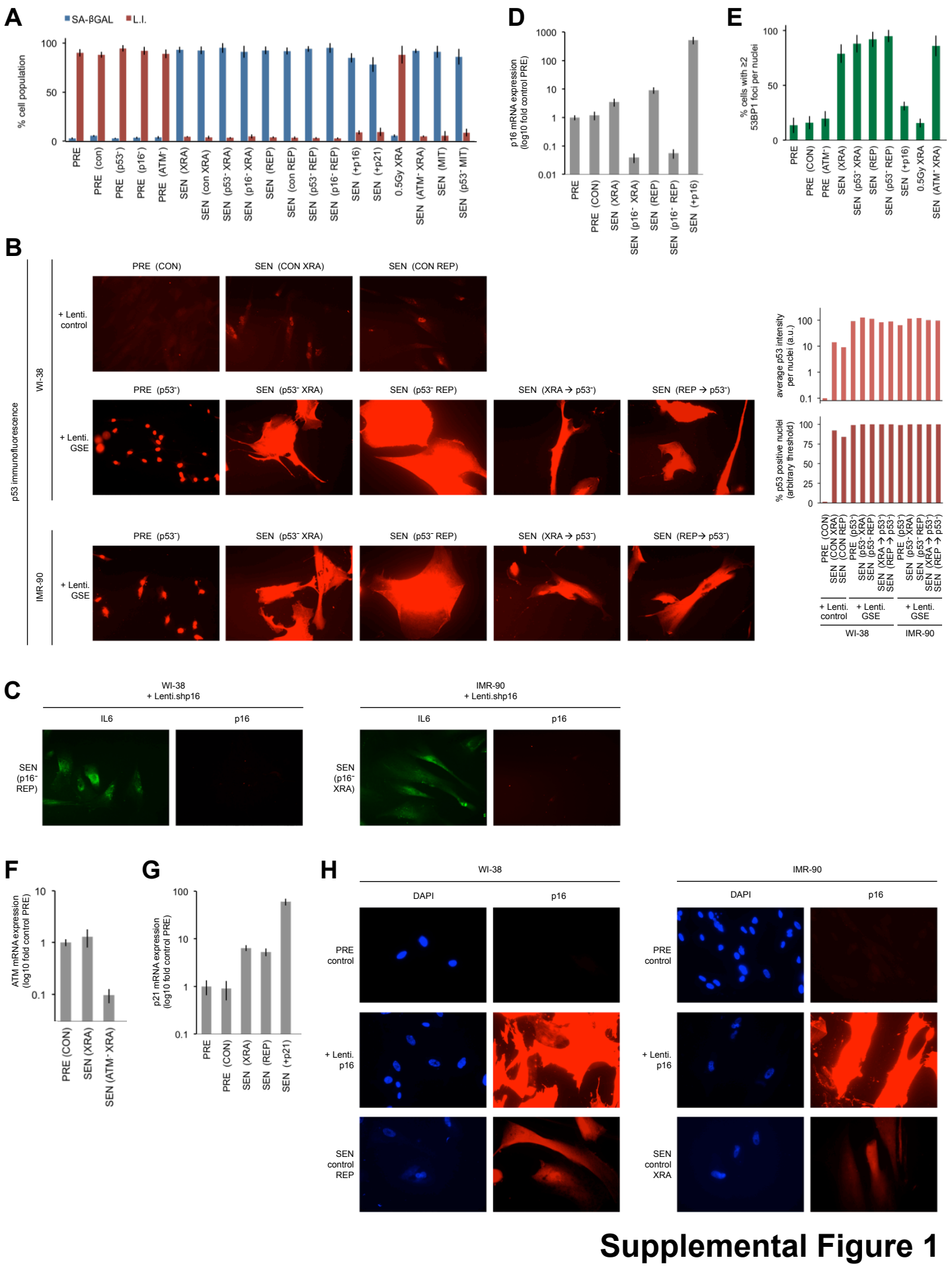
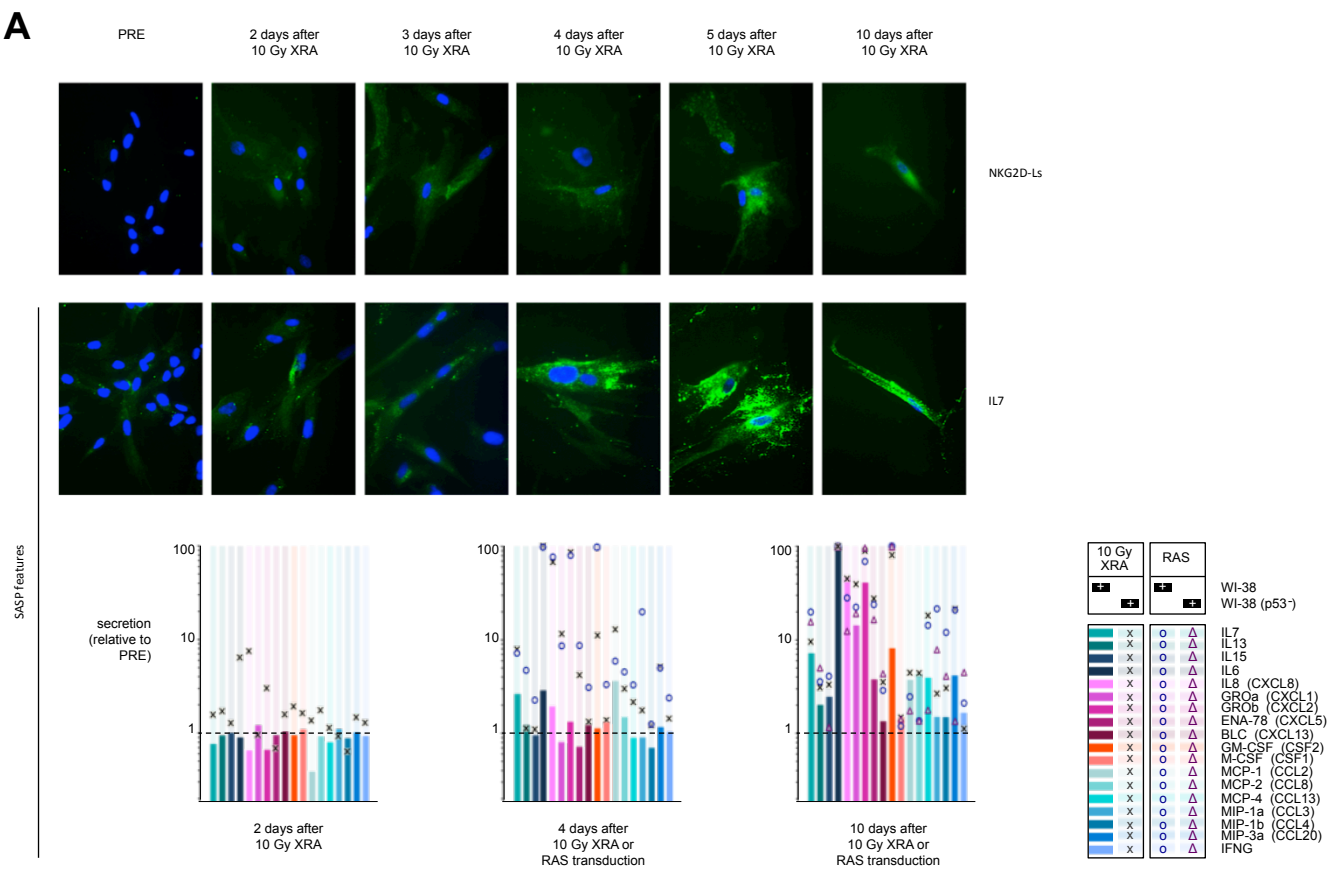


Supplemental Figure 1

A

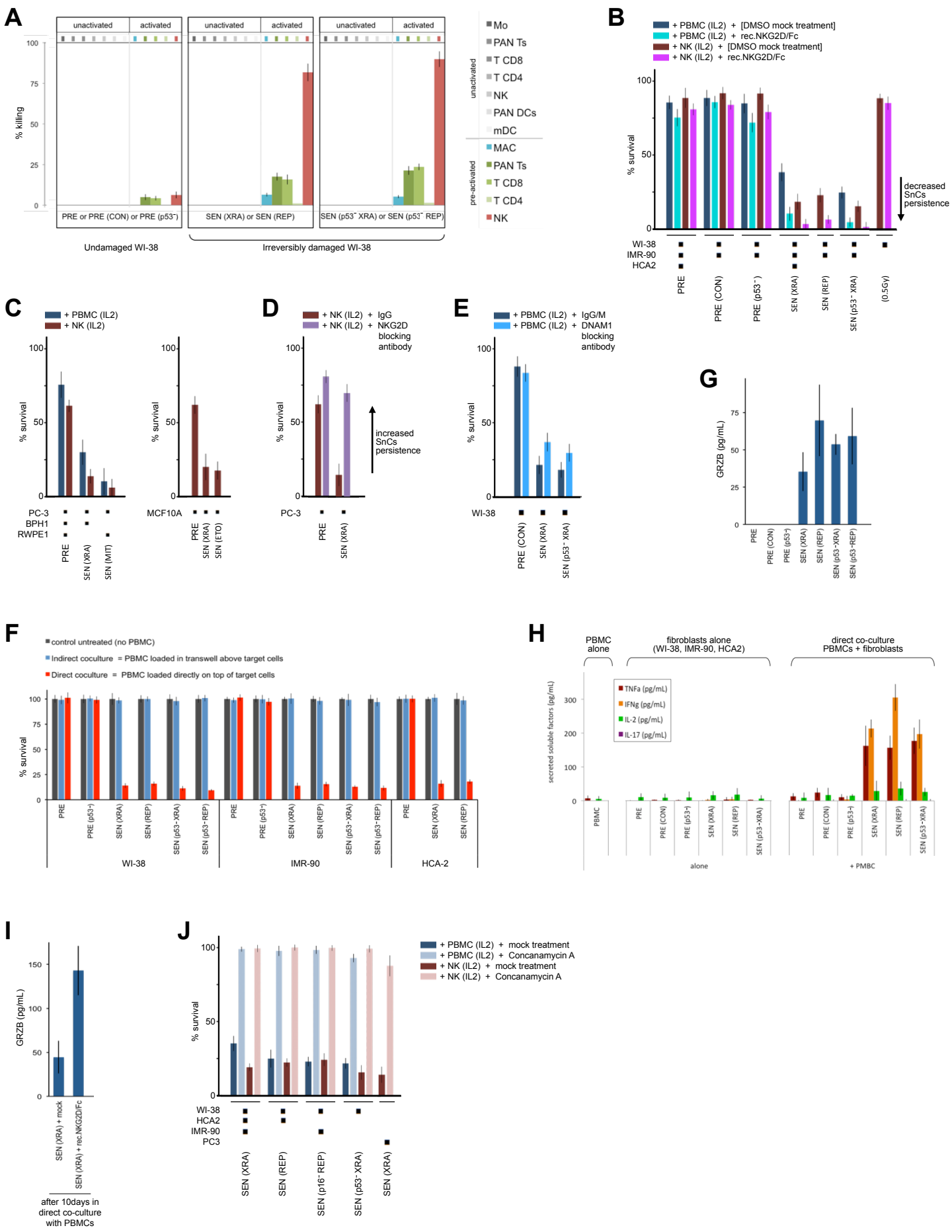


B

Fold change vs. PRE		MICA	MICB	ULBP1	ULBP2	ULBP3
MCF10A	**PRE**	1.0	1.0	n.d.	1.0	1.0
	SEN (XRA)	4.9	2.5	n.d.	3.0	0.8
	SEN (ETO)	3.4	1.6	n.d.	2.7	0.2
PC-3	**PRE**	1.0	1.0	1.0	1.0	1.0
	SEN (XRA)	3.2	2.8	1.2	3.1	n.d.
	SEN (MIT)	5.7	2.3	n.d.	3.5	2.1
BPH1	**PRE**	1.0	1.0	nt	1.0	1.0
	SEN (XRA)	9.5	8.3	nt	1.8	n.d.
	SEN (MIT)	11.3	6.7	nt	3.5	1.4
DU145	**PRE**	1.0	1.0	nt	1.0	nt
	SEN (XRA)	2.8	1.7	nt	2.4	nt

n.t. = not tested
n.d. = not detected

Supplemental Figure 2



Supplemental Figure 3

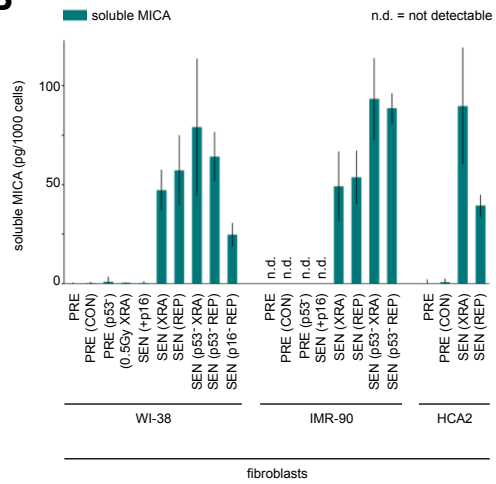
A

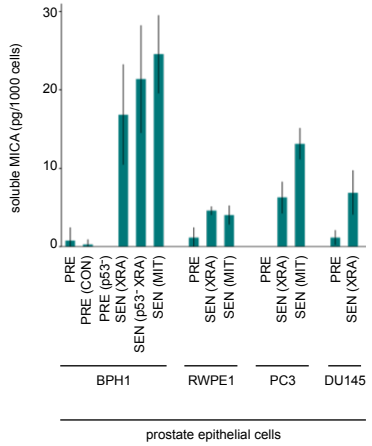
gene expression normalized to CD90 expression	SEN (XRA) naïve (mock treated) @ +10d	SEN (XRA) after PBMC @ +10d	SEN (p16i3- XRA) naïve (mock treated) @ +10d	SEN (p16i3- XRA) after PBMC @ +10d	SEN (REP) naïve (mock treated) @ +10d	SEN (REP) after PBMC @ +10d	SEN (XRA) after 1 round of PBMC @ +10d	SEN (XRA) after 2 rounds of PBMC @ +20d
MICA	1.00	3.37	1.00	2.55	1.00	2.04	1.00	1.91
MICB	1.00	24.54	1.00	20.53	1.00	52.21	1.00	2.54
ULBP1	1.00	0.83	1.00	2.37	1.00	0.67	1.00	0.98
ULBP2	1.00	2.12	1.00	2.17	1.00	2.66	1.00	1.46
ULBP3	1.00	3.25	1.00	1.98	1.00	2.37	1.00	1.08

C

MMP1	1.00	4.25	1.00	7.06	1.00	1.80	1.00	5.60
MMP3	1.00	14.17	1.00	>10,000	1.00	>10,000	1.00	14.59
MMP10	1.00	187.19	1.00	105.77	1.00	37.80	1.00	65.18
MMP12	1.00	>10,000	1.00	8092.96	1.00	3181.20	1.00	11.27

B





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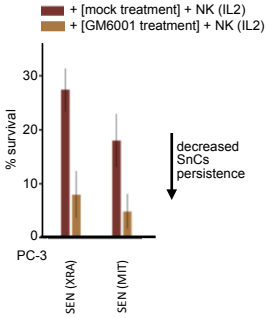
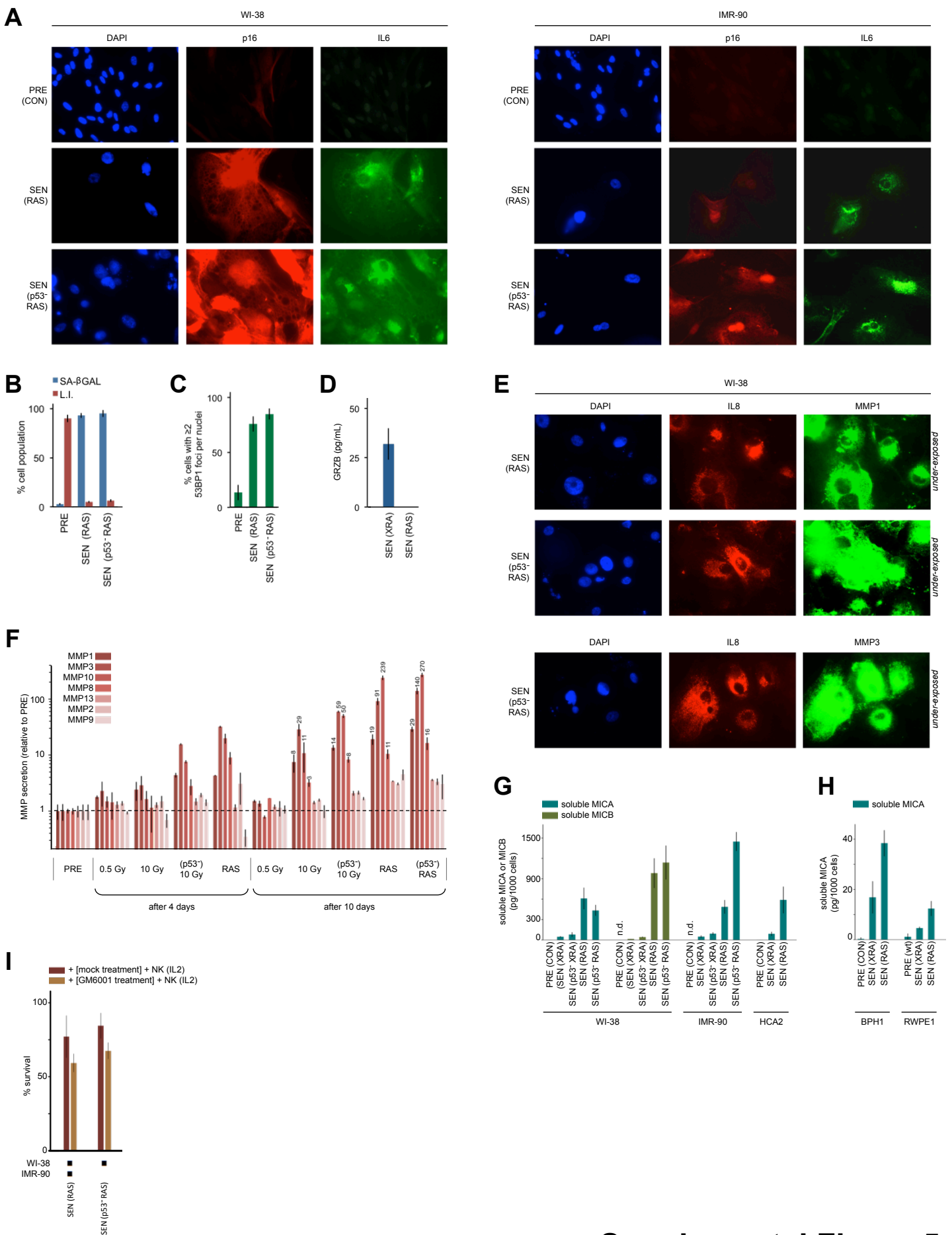
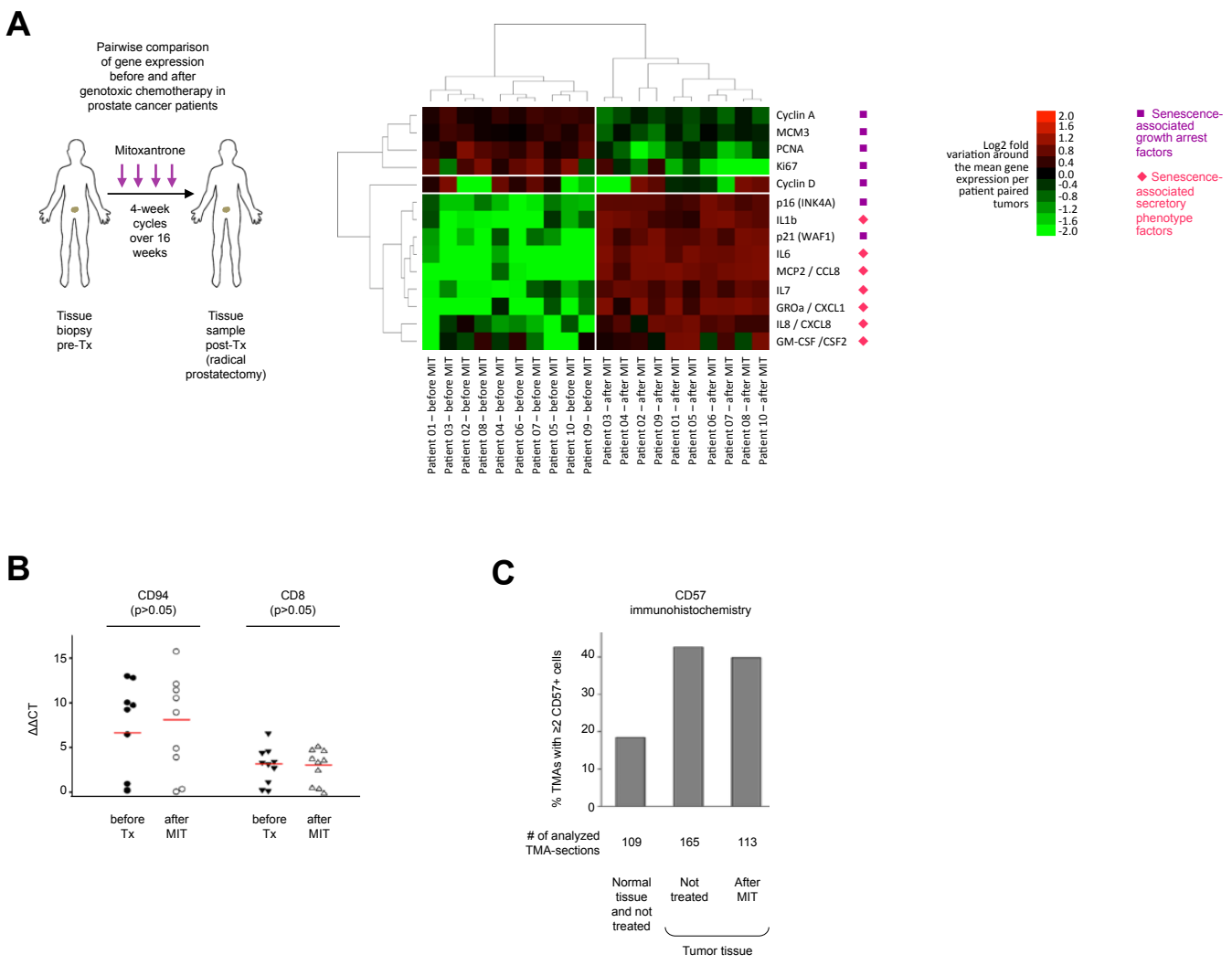


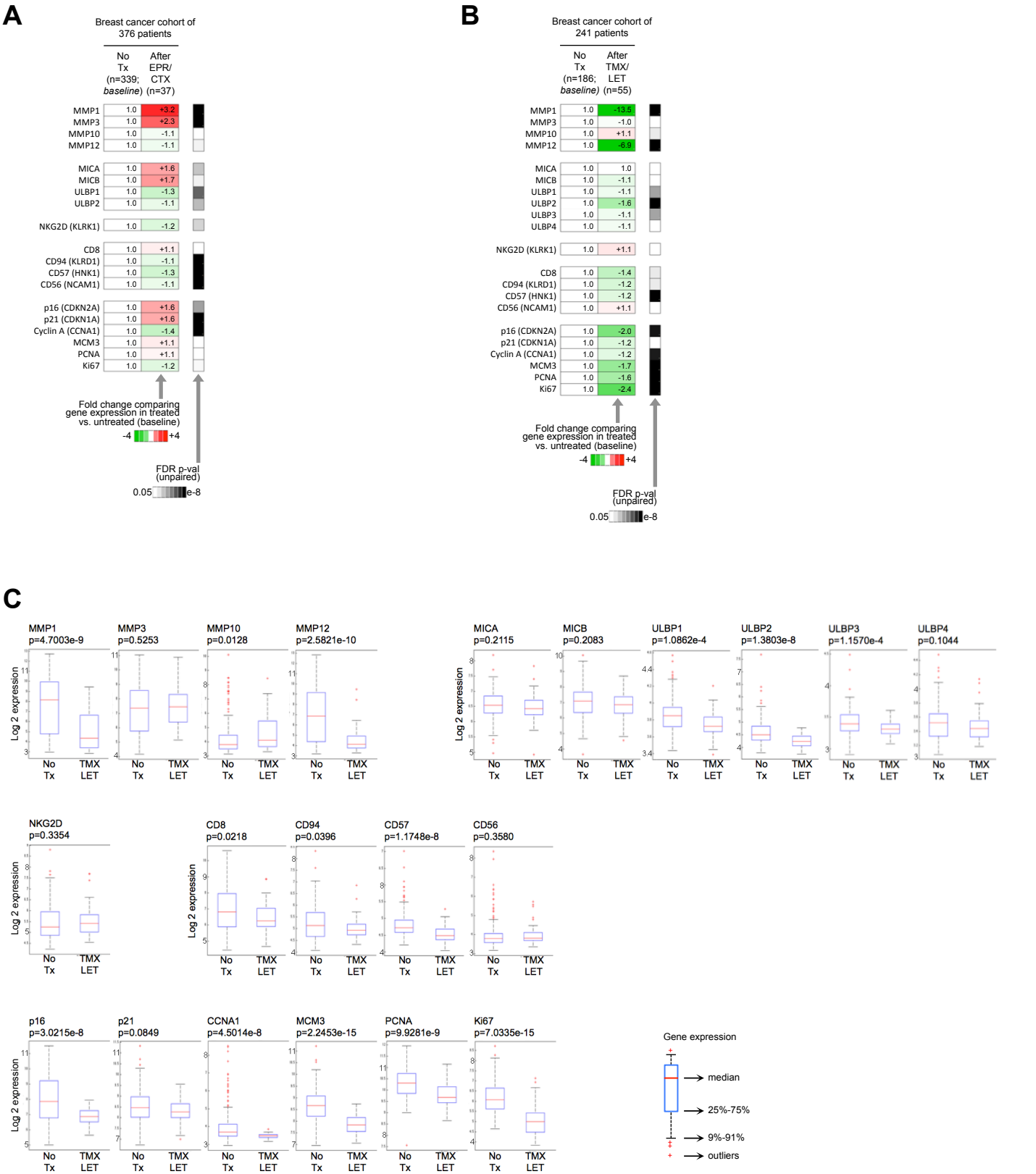
Supplemental Figure 4



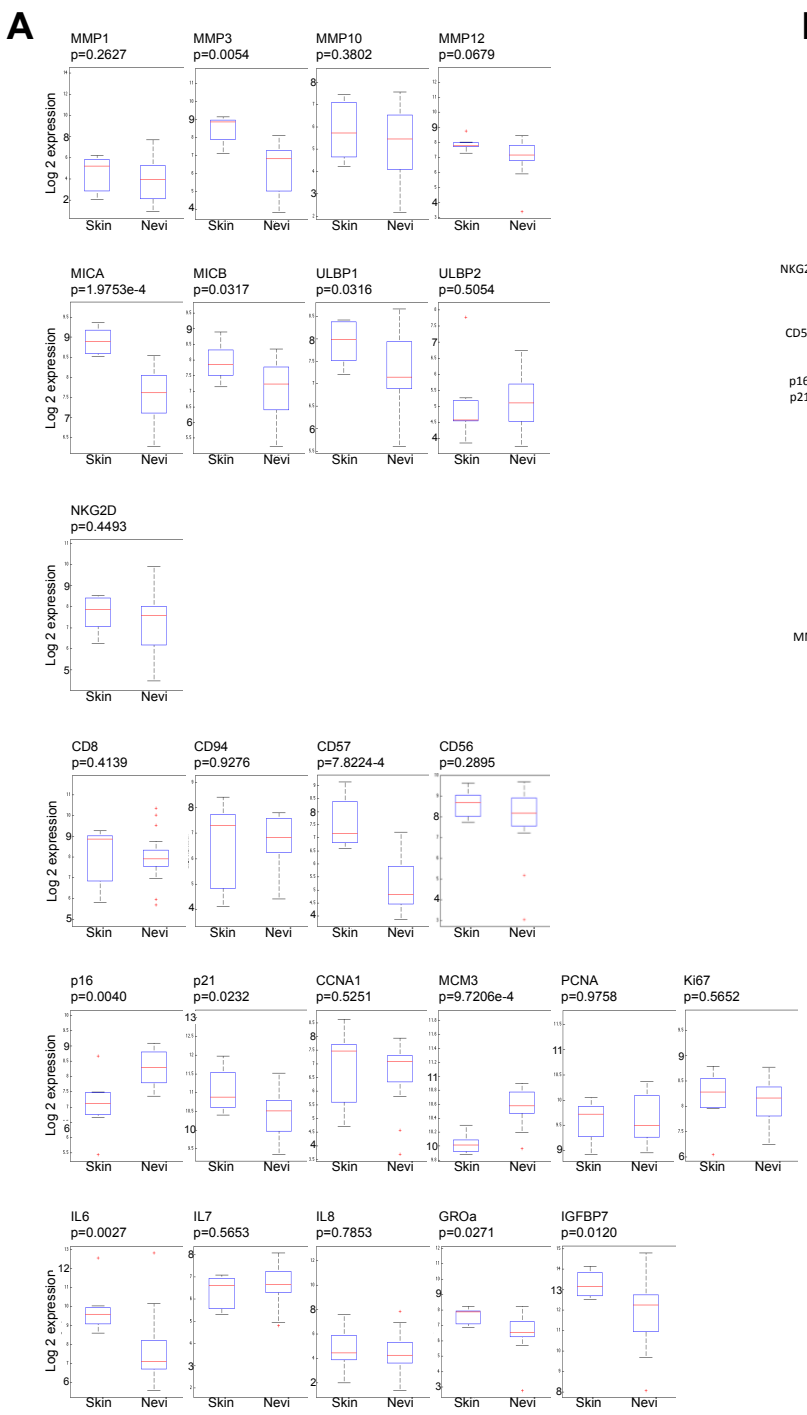
Supplemental Figure 5

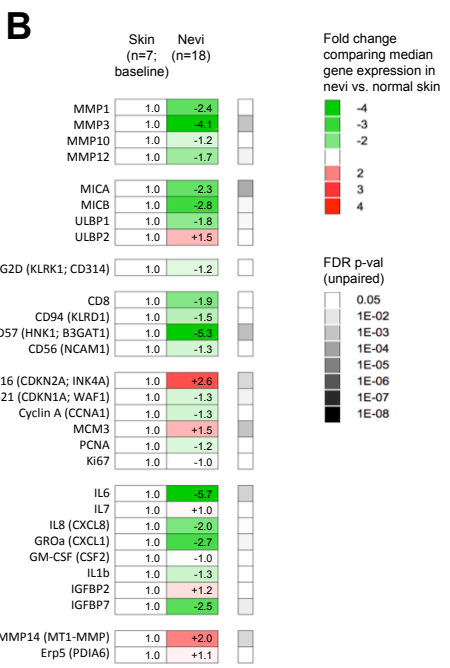


Supplemental Figure 6



Supplemental Figure 7





Supplemental Figure 8

designation name	phenotype	genetic alteration	treatment	damage type	n* (n**)		
					WI-38 (3% O2)	IMR-90 (3% O2)	HCA2 (3% O2)
PRE	presenescent	wild type	none	undamaged	15 (2)	9 (1)	8 (1)
PRE (CON)	presenescent	control (i.e. lenti-GFP, or empty lenti-vector, or lenti-shCON)	none	undamaged	11 (2)	5 (1)	4 (1)
PRE (p53-)	presenescent	lenti-GSE ***	none	undamaged	6 (1)	4 (1)	na
PRE (p16-)	presenescent	lenti-shp16 ***	none	undamaged	2 (1)	2 (1)	nt
SEN (XRA)	senescent	wild type	10 Gy XRA; radiomimetic-induced senescence	genomic damage	12 (3)	8 (1)	5 (1)
SEN (CON XRA)	senescent	control (i.e. lenti-GFP, or empty lenti-vector, or lenti-shCON)	10 Gy XRA; radiomimetic-induced senescence	genomic damage	5 (1)	1 (1)	1 (0)
SEN (p53- XRA)	senescent	lenti-GSE expression in PRE cells before X-ray-induced senescence	10 Gy XRA; radiomimetic-induced senescence	genomic damage	4 (2)	3 (1)	na
SEN (XRA > p53-)	senescent	lenti-GSE expression in SEN cells after X-ray-induced senescence	10 Gy XRA; radiomimetic-induced senescence	genomic damage	2 (1)	2 (1)	na
SEN (p16- XRA)	senescent	lenti-shp16 expression in PRE cells before X-ray-induced senescence	10 Gy XRA; radiomimetic-induced senescence	genomic damage	2 (1)	2 (1)	nt
SEN (REP)	senescent	wild type	replicative senescence	genomic damage	10 (2)	4 (2)	3 (1)
SEN (CON REP)	senescent	control (i.e. lenti-GFP, or empty lenti-vector, or lenti-shCON)	replicative senescence	genomic damage	2 (1)	2 (1)	nt
SEN (p53- REP)	senescent	lenti-GSE expression in PRE cells before replicative senescence	replicative senescence	genomic damage	6 (1)	2 (1)	na
SEN (REP > p53-)	senescent	lenti-GSE expression in SEN cells after replicative senescence	replicative senescence	genomic damage	2 (1)	2 (1)	na
SEN (p16- REP)	senescent	lenti-shp16 expression in PRE cells before replicative senescence	replicative senescence	genomic damage	2 (1)	2 (1)	nt
SEN (MIT)	senescent	mitoxantrone-induced senescence	chemotherapy-induced senescence	genomic damage	5 (1)	2 (1)	nt
SEN (p53- MIT)	senescent	lenti-GSE expression in PRE cells before Mitoxantrone-induced senescence	chemotherapy-induced senescence	genomic damage	2 (1)	nt	na
SEN (+p16)	senescent	lenti-p16 ***	tumor suppressor overexpression-induced senescence	undamaged	3 (1)	2 (1)	nt
SEN (+p21)	senescent	lenti-p21 ***	tumor suppressor overexpression-induced senescence	undamaged	1 (1)	1 (0)	nt
PRE (0.5Gy XRA)	recovered	wild type	0.5 Gy XRA-induced transient genomic damage	recovered from damage	3 (2)	nt	nt
PRE (ATM-)	presenescent	lenti-shATM ***	none	undamaged	3 (1)	nt	2 (1)
SEN (ATM- XRA)	senescent	lenti-shATM	10 Gy XRA; radiomimetic	genomic damage	3 (1)	nt	2 (1)
SEN (RAS)	senescent	lenti-HRAS(G12V) ***	oncogene-induced senescence	oncogenic damage	6 (2)	4 (0)	3 (1)
SEN (p53- RAS)	senescent	lenti-GSE expression in PRE cells before lenti-HRAS(G12V) expression-induced senescence	oncogene-induced senescence	oncogenic damage	3 (2)	2 (0)	na

					PC3 (3% O2)	LNCaP (3% O2)	RWPE1 (3% O2)	BPH1 (3% O2)
PRE	presenescent	wild type	none	undamaged ****	4 (2)	2 (1)	2 (1)	2 (1)
SEN (XRA)	senescent	wild type	10 Gy XRA; radiomimetic-induced senescence	genomic damage	4 (2)	2 (1)	2 (1)	2 (1)
SEN (MIT)	senescent	mitoxantrone-induced senescence	chemotherapy-induced senescence	genomic damage	3 (1)	1 (1)	2 (1)	1 (0)
SEN (RAS)	senescent	lenti-HRAS(G12V) ***	oncogene-induced senescence	oncogenic damage	2 (1)	nt	2 (1)	nt

					MCF10A (3% O2)
PRE	presenescent	wild type	none	undamaged ****	3 (2)
SEN (XRA)	senescent	wild type	10 Gy XRA; radiomimetic-induced senescence	genomic damage	2 (1)
SEN (ETO)	senescent	etoposide-induced senescence	chemotherapy-induced senescence	genomic damage	3 (1)

Supplemental Table 1

A

				activating ligands (i.e. promoting cytolysis)							inhibitory ligands	
				NKG2D-ligands					DNAM1-ligands			
				MICA	MICB	ULBP-1	ULBP-2	ULBP-3	CD112	CD155	HLA-C	HLA-E
	mRNA expression in WI-38 PRE (n=11)			*44.8*	*41.2*	*4.6*	*7.7*	*4.1*	*451*	*164*	*3098*	*391*
WI-38	PRE	3 & 20 % O2	n=5	0.6	0.5	0.3	0.6	0.6	0.8	0.4	1.1	1.0
	PRE (CON)	3% O2	n=4	1.1	1.2	1.0	1.1	0.9	1.1	1.3	0.9	0.9
	PRE (p53-)	3% O2	n=2	1.7	1.5	2.4	1.5	2.3	1.2	1.5	0.9	1.4
	SEN (XRA)	3 & 20 % O2	n=5	29.9	24.0	11.6	9.9	3.8	5.2	24.5	3.4	3.0
	SEN (REP)	3 & 20 % O2	n=4	24.9	40.0	11.3	23.1	4.7	5.0	23.4	2.6	2.9
	SEN (p53- XRA)	3% O2	n=2	22.8	11.5	23.2	14.8	3.2	3.5	16.5	3.1	3.2
	SEN (p53- REP)	3% O2	n=2	37.7	16.7	40.7	32.5	5.4	5.5	35.0	3.8	4.7
	SEN (XRA> p53-)	3% O2	n=1	43.3	16.1	22.8	31.9	5.9	5.8	35.6	2.4	3.0
	SEN (REP> p53-)	3% O2	n=2	70.4	25.3	34.9	54.9	7.0	6.4	45.8	2.4	3.4
	SEN (p16- XRA)	3% O2	n=1	20.9	16.0	10.9	11.7	4.2	4.0	18.3	n.t.	n.t.
	SEN (p16- REP)	20% O2	n=2	20.6	18.9	11.9	14.4	3.5	2.2	8.6	1.4	1.4
	SEN (+p16)	3% O2	n=2	3.8	4.2	2.5	5.1	1.4	2.0	6.7	1.6	3.4
	SEN (ATM- XRA)	3% O2	n=2	4.7	3.0	6.6	3.0	1.7	1.9	3.5	n.t.	1.2
	SEN (RAS)	3% O2	n=1	134.7	59.5	206.1	105.3	18.1	11.8	97.0	2.7	7.7
	SEN (p53- RAS)	3% O2	n=1	109.4	49.6	241.6	75.9	30.1	7.3	54.0	2.1	5.0
	mRNA expression in IMR-90 PRE (n=6)			*43.2*	*29.9*	*2.4*	*3.0*	*6.2*	*562*	*175*	*1254*	*334*
IMR-90	PRE	3 & 20 % O2	n=3	1.0	1.0	0.8	0.9	0.6	1.3	1.3	1.5	0.7
	PRE (CON)	3% O2	n=1	0.8	1.0	0.9	0.8	1.3	0.6	0.8	0.5	1.1
	PRE (p53-)	3% O2	n=2	1.2	1.1	1.4	1.4	1.5	0.7	0.7	0.5	1.6
	SEN (XRA)	3 & 20 % O2	n=3	16.2	8.9	3.5	18.9	9.3	3.3	7.4	4.7	5.0
	SEN (REP)	3% O2	n=2	25.4	13.7	6.5	54.5	9.2	1.7	7.4	2.1	6.2
	SEN (p53- XRA)	3% O2	n=2	20.0	12.5	11.0	24.0	7.4	2.4	6.3	1.9	4.1
	SEN (p53- REP)	3% O2	n=1	22.7	13.1	8.8	39.3	8.3	2.0	6.8	2.0	5.2
	SEN (+p16)	3% O2	n=2	2.1	3.1	2.5	3.0	1.6	1.2	2.0	1.2	3.0
	SEN (+p21)	3% O2	n=2	3.9	3.8	2.2	5.9	3.2	0.8	1.5	1.4	1.8
	SEN (RAS)	3% O2	n=1	60.6	33.8	48.0	78.8	28.7	2.9	14.9	1.1	5.9
	SEN (p53- RAS)	3% O2	n=1	41.4	20.2	43.1	78.7	14.3	2.0	9.6	1.0	4.7
	mRNA expression in HCA2 PRE (n=5)			*65.6*	*52.8*	*0.9*	*5.4*	*2.8*	*386*	*104*	*3677*	*457*
HCA2	PRE	3% O2	n=3	1.0	1.0	0.9	1.1	0.8	1.2	1.0	1.2	0.9
	PRE (p53-)	3% O2	n=2	1.1	1.0	1.1	0.7	1.3	0.6	0.9	0.6	1.1
	SEN (XRA)	3% O2	n=2	8.8	5.7	1.7	3.2	1.2	1.5	2.1	1.9	1.6
	SEN (REP)	3% O2	n=1	9.7	8.3	1.7	3.5	1.5	1.4	5.5	1.1	1.8
	SEN (RAS)	3% O2	n=2	39.2	17.8	28.4	31.1	8.4	3.7	30.2	0.9	3.9
	SEN (p53- RAS)	3% O2	n=1	44.2	17.5	70.9	35.8	13.1	3.6	33.3	1.1	4.4

B

				p16	p21	p53	GAPDH	H1A
	mRNA expression in WI-38 PRE (n=11)			*150*	*456*	*224*	*16295*	*51.4*
WI-38	PRE	3 & 20 % O2	n=5	0.1	1.2	0.8	0.9	1.1
	PRE (CON)	3% O2	n=4	2.3	0.9	0.9	1.0	0.6
	PRE (p53-)	3% O2	n=2	0.4	0.4	2.2	1.1	1.3
	SEN (XRA)	3 & 20 % O2	n=5	3.0	6.8	2.9	1.9	0.2
	SEN (REP)	3 & 20 % O2	n=4	9.8	5.2	2.9	2.3	0.2
	SEN (p53- XRA)	3% O2	n=2	27.3	2.9	4.6	2.3	1.0
	SEN (p53- REP)	3% O2	n=2	47.7	0.9	6.6	3.1	1.7
	SEN (XRA> p53-)	3% O2	n=1	n.t.	n.t.	n.t.	n.t.	n.t.
	SEN (REP> p53-)	3% O2	n=2	105.9	1.1	5.9	2.7	1.1
	SEN (p16- XRA)	3% O2	n=1	n.t.	n.t.	n.t.	n.t.	n.t.
	SEN (p16- REP)	20% O2	n=2	0.1	3.3	1.2	2.6	0.6
	SEN (+p16)	3% O2	n=2	2922	4.8	4.6	1.4	0.5
	SEN (ATM- XRA)	3% O2	n=2	151.5	n.t.	n.t.	n.t.	n.t.
	SEN (RAS)	3% O2	n=1	181.0	55.3	5.7	7.5	0.2
	SEN (p53- RAS)	3% O2	n=1	131.4	14.8	7.2	5.4	0.3
	mRNA expression in IMR-90 PRE (n=6)			*30.6*	*581*	*454*	*15734*	*55.1*
IMR-90	PRE	3 & 20 % O2	n=3	0.8	0.9	0.8	1.0	1.0
	PRE (CON)	3% O2	n=1	0.9	0.8	0.9	0.9	0.7
	PRE (p53-)	3% O2	n=2	1.4	0.8	1.1	1.1	1.5
	SEN (XRA)	3 & 20 % O2	n=3	2.2	3.3	1.0	2.4	0.7
	SEN (REP)	3% O2	n=2	8.0	3.1	1.6	5.1	1.0
	SEN (p53- XRA)	3% O2	n=2	6.5	2.3	1.6	3.1	1.6
	SEN (p53- REP)	3% O2	n=1	n.t.	n.t.	n.t.	4.1	1.3
	SEN (+p16)	3% O2	n=2	500.6	2.5	0.7	1.5	0.6
	SEN (+p21)	3% O2	n=2	1.3	15.0	0.6	1.8	1.0
	SEN (RAS)	3% O2	n=1	32.3	13.9	1.1	6.7	0.8
	SEN (p53- RAS)	3% O2	n=1	20.9	2.5	0.8	4.5	1.5
	mRNA expression in HCA2 PRE (n=5)			*2.1*	*188*	*288*	*14985*	*45.4*
HCA2	PRE	3% O2	n=3	1.0	1.3	0.9	1.0	0.9
	PRE (p53-)	3% O2	n=2	1.0	0.4	1.3	1.0	1.1
	SEN (XRA)	3% O2	n=2	2.5	4.7	1.0	1.1	1.8
	SEN (REP)	3% O2	n=1	0.7	6.0	2.0	1.2	5.2
	SEN (RAS)	3% O2	n=2	43.7	16.8	1.9	5.8	1.4
	SEN (p53- RAS)	3% O2	n=1	208.2	6.4	3.4	4.8	2.1

C

				matrix metallo-proteases (MMP) genomic cluster								other extracellular proteases			
				MMP1	MMP3	MMP10	MMP12	MMP8	MMP13	MMP27	MMP7	PDIA6	ADAM17	MT1-MMP	PSEN1
	mRNA expression in WI-38 PRE (n=11)			*205*	*24.7*	*5.1*	*0.4*	*0.1*	*0.1*	*0.1*	*0.2*	*82.5*	*79.0*	*2140*	*54.2*
WI-38	PRE	3 & 20 % O2	n=5	1.3	0.8	1.6	0.3	0.6	1.3	1.3	1.2	0.6	0.6	n.t.	1.0
	PRE (CON)	3% O2	n=4	0.8	1.6	0.6	1.5	1.5	0.6	0.5	0.4	1.2	0.9	1.0	0.8
	PRE (p53-)	3% O2	n=2	1.2	0.3	0.5	0.7	0.7	0.6	0.6	0.8	0.8	1.7	n.t.	1.6
	SEN (XRA)	3 & 20 % O2	n=5	59.6	244.0	21.7	195.1	12.5	2.7	2.3	1.6	3.8	2.6	1.5	1.9
	SEN (REP)	3 & 20 % O2	n=4	41.1	169.0	11.0	1148	3.0	1.6	0.7	1.5	3.7	4.5	n.t.	2.8
	SEN (p53- XRA)	3% O2	n=2	394.0	1776	16.1	176.5	12.0	4.5	1.8	7.4	5.0	6.1	n.t.	3.1
	SEN (p53- REP)	3% O2	n=2	489.5	3131	109.7	673.1	5.9	4.4	2.4	6.7	4.1	3.9	n.t.	3.2
	SEN (XRA> p53-)	3% O2	n=1	8.9	172.1	2.3	66.7	5.1	7.6	3.0	n.d.	n.t.	n.t.	n.t.	n.t.
	SEN (REP> p53-)	3% O2	n=2	2.3	65.8	0.8	24.8	3.2	6.1	0.7	n.d.	4.1	n.t.	n.t.	n.t.
	SEN (p16- XRA)	3% O2	n=1	6.3	6.0	3.0	529.2	5.5	1.6	2.3	1.1	n.t.	n.t.	n.t.	n.t.
	SEN (p16- REP)	20% O2	n=2	7.3	4.9	0.2	270.9	4.5	1.8	0.7	0.7	2.7	n.t.	n.t.	n.t.
	SEN (+p16)	3% O2	n=2	17.0	14.0	2.4	10.9	1.7	0.6	1.0	1.3	n.t.	n.t.	n.t.	n.t.
	SEN (ATM- XRA)	3% O2	n=2	21.8	84.8	47.7	49.1	2.2	n.d.	n.t.	n.t.	1.8	1.9	1.6	0.9
	SEN (RAS)	3% O2	n=1	2540	47623	2203	11991	109.5	27.7	7.9	7.8	8.8	9.0	n.t.	11.5
	SEN (p53- RAS)	3% O2	n=1	5471	49490	1209	12433	153.7	93.6	3.3	n.t.	7.5	10.1	n.t.	10.7
	mRNA expression in IMR-90 PRE (n=6)			*22.3*	*1.7*	*2.7*	*0.3*	*0.2*	*0.1*	*0.1*	*0.2*	*52.3*	*61.3*	*n.t.*	*54.7*
IMR-90	PRE	3 & 20 % O2	n=3	0.8	1.3	1.3	1.6	2.2	1.1	1.3	1.7	0.7	0.8	n.t.	1.1
	PRE (CON)	3% O2	n=1	0.7	0.4	0.5	0.4	0.2	n.d.	0.7	0.3	1.5	0.2	n.t.	0.2
	PRE (p53-)	3% O2	n=2	1.8	1.1	0.6	0.5	0.7	0.8	0.7	n.t.	1.5	2.4	n.t.	1.7
	SEN (XRA)	3 & 20 % O2	n=3	23.3	22.3	4.0	8.4	0.6	3.5	0.7	0.3	3.5	2.6	n.t.	1.9
	SEN (REP)	3% O2	n=2	26.2	318.2	13.5	91.9	1.6	1.6	n.d.	4.1	8.7	5.7	n.t.	3.5
	SEN (p53- XRA)	3% O2	n=2	82.4	80.1	2.3	12.8	3.7	4.8	n.d.	1.0	7.0	5.7	n.t.	3.2
	SEN (p53- REP)	3% O2	n=1	54.3	199.2	7.9	52.3	2.6	3.2	n.d.	1.5	n.t.	n.t.	n.t.	n.t.
	SEN (+p16)	3% O2	n=2	10.4	6.5	0.2	3.1	0.5	0.5	n.d.	0.6	n.t.	n.t.	n.t.	n.t.
	SEN (+p21)	3% O2	n=2	9.1	5.7	0.2	1.7	0.8	0.4	0.4	n.d.	3.1	2.6	n.t.	1.6
	SEN (RAS)	3% O2	n=1	2662	7720	975.2	278.1	41.6	13.5	2.9	13.6	12.7	4.8	n.t.	5.6
	SEN (p53- RAS)	3% O2	n=1	3284	11188	257.3	113.8	30.5	22.1	4.0	n.t.	n.t.	n.d.	n.t.	n.d.
	mRNA expression in HCA2 PRE (n=5)			*86.8*	*77.0*	*0.6*	*0.4*	*0.0*	*0.1*	*0.1*	*0.1*	*46.9*	*54.3*	*n.t.*	*41.1*
HCA2	PRE	3% O2	n=3	0.8	0.9	1.0	1.0	1.0	0.6	1.0	1.0	0.8	0.7	n.t.	0.7
	PRE (p53-)	3% O2	n=2	1.5	1.1	0.9	1.0	n.d.	1.8	1.1	n.t.	1.4	1.6	n.t.	1.5
	SEN (XRA)	3% O2	n=2	28.7	20.7	11.9	19.0	2.0	2.1	2.7	5.8	2.4	1.5	n.t.	1.3
	SEN (REP)	3% O2	n=1	38.5	17.4	6.2	21.5	n.d.	4.0	1.3	4.5	4.6	3.4	n.t.	2.9
	SEN (RAS)	3% O2	n=2	617.4	932.1	546.4	1098	49.5	17.7	4.9	26.5	11.8	6.9	n.t.	7.7
	SEN (p53- RAS)	3% O2	n=1	1102	1023	1459	1601	156.8	43.6	27.2	n.t.	15.6	8.2	n.t.	9.6

1 10 25 50 200 500 1000
color scale (saturated <1 and >1,000 fold)

n.t. = not tested
n.d. = not detected

D

	CD90	MICA	MICB	ULBP1	ULBP2	ULBP3	MMP1	MMP3
WI-38 SEN (XRA)	3325.68	1250.64	958.27	98.62	163.33	24.5618	10982.29	4420.98
WI-38 SEN (REP)	3112.55	1186.11	1447.50	90.32	209.56	27.4877	8257.81	8416.22
PBMC	0.052	4.541	3.444	0.039	0.534	0.094	0.433	n.d.

Supplemental Table 2

A

human gene name	Applied Biosystems primers ID
h- MICA	Hs 00792195 m1
h- MICB	Hs 00792952 m1
h- ULBP-1	Hs 00360941 m1
h- ULBP-2	Hs 00607609 mH
h- ULBP-3	Hs 00225909 m1
h- CD112	Hs 01071562 m1
h- CD155	Hs 00197846 m1
h- MMP-1	Hs 00233958 m1
h- MMP-10	Hs 00233987 m1
h- MMP-12	Hs 00159178 m1
h- MMP-13	Hs 00233992 m1
h- MMP-20	Hs 00191117 m1
h- MMP-27	Hs 00223193 m1
h- MMP-3	Hs 00233962 m1
h- MMP-7	Hs 00159163 m1
h- MMP-8	Hs 00223972 m1
h- H1A	Hs 00271225 s1
h- ATM	Hs 01112307_m1
h- PDIA6 (ERP5)	Hs 00194922_m1
h- ADAM17	Hs 00234221_m1
h- PSEN1	Hs 00997789_m1
h- MT1MMP (MMP-14)	Hs 00237119_m1
h- Cathepsin B (CSTB)	Hs 00157194_m1
h- NKG2D (KLRK1)	Hs 01095635_m1
h- CD56 (NCAM1)	Hs 00941833_m1
h- CD8	Hs 01555600_m1
h- CD94	Hs 00233841_m1
h- CD14	Hs 00169122_m1
h- CD1a	Hs 00233332_m1
h- CD45	Hs 00236304_m1
h- CD10	Hs 00153510_m1
h- CD90 (THY-1)	Hs 00174816_m1

human gene name	forward & reverse sequences (5' - 3')
h- p16	CCAACGCACCGAATAGTTACG
	GGGCGCTGCCCATCA
h- p21	TGGAGACTCTCAGGGTCGAAA
	AGGACTGCAGGCTTCCTGTG
h- p53	CCCAGCCAAAGAAGAAACCA
	CTCGGA ACATCTCGAAGCG
h- GUS	CTCATTTGGAATTTTGCCGATT
	CCGAGTGAAGATCCCCTTTTTA
h- GAPDH	ATTCCACCCATGGCAAATTC
	TGGGATTTCCATTGATGACAAG

B

Gene	Entrez gene ID	microarray probe set ID	platform U133A	platform U133plus2
MICA	4276	205904_at	available	available
MICA	4276	205905_s_at		available*
MICB	4277	205905_s_at		available*
MICB	4277	206247_at	available	available
ULBP1	80329	221323_at	available	available
ULBP2	80328	221291_at	available	available
ULBP2	80328	238542_at		available
ULBP3	79465	231748_at		available
ULBP4 (RAET1E)	135250	1552777_a_at		available
ICAM1 (CD54)	3383	202637_s_at	available	available
ICAM1 (CD54)	3383	202638_s_at	available	available
ICAM1 (CD54)	3383	215485_s_at	available	available
MMP1	4312	204475_at	available	available
MMP3	4314	205828_at	available	available
MMP10	4319	205680_at	available	available
MMP12	4321	204580_at	available	available
MMP13	4322	205959_at	available	available
MMP8	4317	207329_at	available	available
MMP8	4317	231688_at		available
NKG2D (KLRK1; CD314)	22914	1555691_a_at		available
NKG2D (KLRK1; CD314)	22914	1555692_at		available
NKG2D (KLRK1; CD314)	22914	205821_at	available	available
CD8 (CD8A)	925	205758_at	available	available
CD8 (CD8A)	925	205759_s_at		available
CD94 (KLRD1)	3824	207795_s_at	available	available
CD94 (KLRD1)	3824	207796_x_at	available	available
CD94 (KLRD1)	3824	210606_x_at	available	available
CD57 (HNK1; B3GAT1)	27087	219521_at	available	available
CD56 (NCAM1)	4684	209968_s_at	available	available
CD56 (NCAM1)	4684	209969_s_at		available
CD56 (NCAM1)	4684	212843_at	available	available
CD56 (NCAM1)	4684	214952_at	available	available
CD56 (NCAM1)	4684	217359_s_at	available	available
CD56 (NCAM1)	4684	227394_at		available
CD56 (NCAM1)	4684	227395_at		available
CD56 (NCAM1)	4684	229799_s_at		available
CD56 (NCAM1)	4684	231532_at		available
p16 (CDKN2A; INK4A)	1029	207039_at	available	
p16 (CDKN2A; INK4A)	1029	209644_x_at	available	available
p16 (CDKN2A; INK4A)	1029	211156_at	available	available
p21 (CDKN1A; WAF1)	1026	1555186_at		available
p21 (CDKN1A; WAF1)	1026	1555187_at		available
p21 (CDKN1A; WAF1)	1026	202284_s_at	available	available
IL6	3569	205207_at	available	available
IL7	3574	206693_at	available	available
IL8 (CXCL8)	3576	202859_x_at	available	available
IL8 (CXCL8)	3576	211506_s_at	available	available
GROa (CXCL1)	2919	204470_at	available	available
GM-CSF (CSF2)	1437	210228_at	available	available
GM-CSF (CSF2)	1437	210229_s_at	available	available
GM-CSF (CSF2)	1437	210230_at		available
IGFBP2	3485	202718_at	available	available
IL1B	3553	205067_at	available	available
IL1B	3553	39402_at	available	available
CyclinA (CCNA1)	8900	205899_at	available	available
MCM3	4172	201555_at	available	available
PCNA	5111	201202_at	available	available
IL2	3558	207849_at	available	available
Ki67 (MKI67)	4288	212020_s_at	available	available
Ki67 (MKI67)	4288	212021_s_at	available	available
Ki67 (MKI67)	4288	212022_s_at	available	available
Ki67 (MKI67)	4288	212023_s_at	available	available
MMP14 (MT1-MMP)	4323	160020_at	available	available
MMP14 (MT1-MMP)	4323	202827_s_at	available	available
MMP14 (MT1-MMP)	4323	202828_s_at	available	available
MMP14 (MT1-MMP)	4323	217279_x_at	available	available
Erp5 (PDIA6)	10130	207668_x_at	available	available
Erp5 (PDIA6)	10130	207669_at		available
Erp5 (PDIA6)	10130	207670_at		available
Erp5 (PDIA6)	10130	208638_at	available	available
Erp5 (PDIA6)	10130	208639_x_at	available	available
Erp5 (PDIA6)	10130	216640_s_at	available	available

* = probe overlap over two genes & data excluded

Supplemental Table 3

A

Study ID	included after bias analysis
E-TABM-43	yes; to study effects of chemotherapy
GSE10281	no
GSE11264	no
GSE12787	no
GSE1378 / GSE1379	no
GSE13787	yes; to study effects of endocrine therapy
GSE1456	no
GSE16391	yes; to study effects of endocrine therapy
GSE16446	yes; to study effects of endocrine therapy
GSE16680	no
GSE17705	no
GSE17705	no
GSE18728	no
GSE18864	no
GSE20194	yes; to study effects of chemotherapy
GSE20194	no
GSE20271	no
GSE2034	no
GSE21653	no
GSE22035	yes; to study effects of endocrine therapy
GSE22093	no
GSE23399	no
GSE23988	yes; to study effects of chemotherapy
GSE25066	no
GSE3494	no
GSE4056	no
GSE61 / E-SMDB-4	no
GSE6434 / GSE349 / GSE350	no
GSE6577	no
GSE6861	no
GSE7515	no

B

Study ID	included after bias analysis
GSE1133	no
GSE19234	no
GSE22138	no
GSE2361	no
GSE2503	no
GSE3189	yes
GSE4587	no
GSE7553	no

Supplemental Table 4