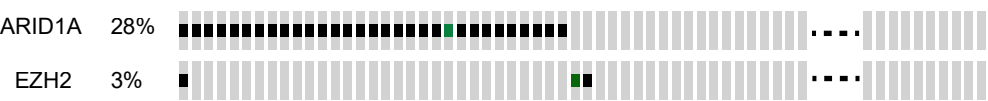
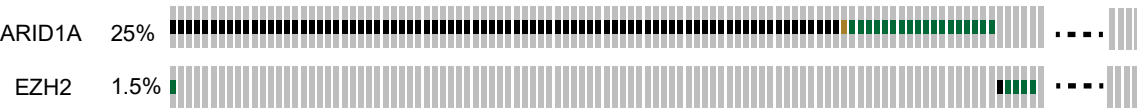


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Muscle Invasive Bladder Cancer (MSKCC, Eur Urol 2014)



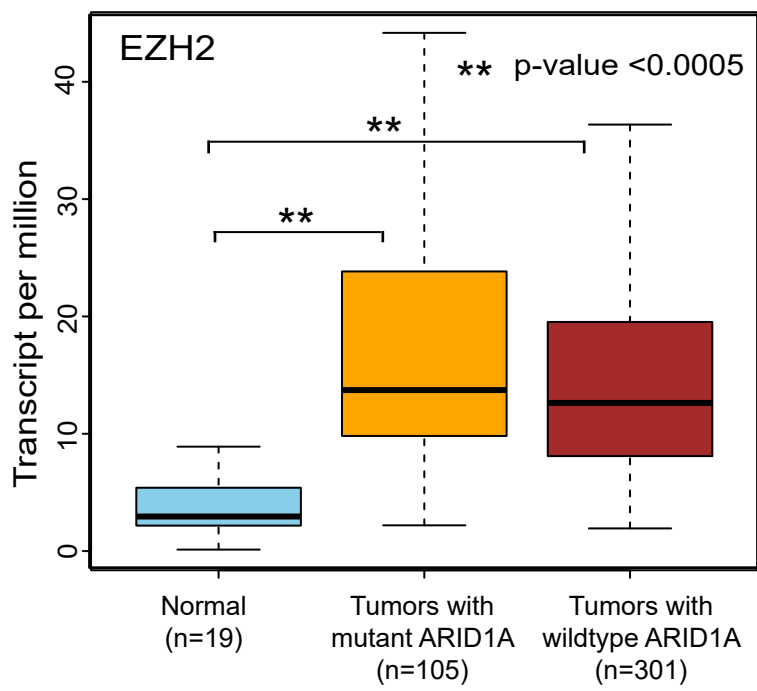
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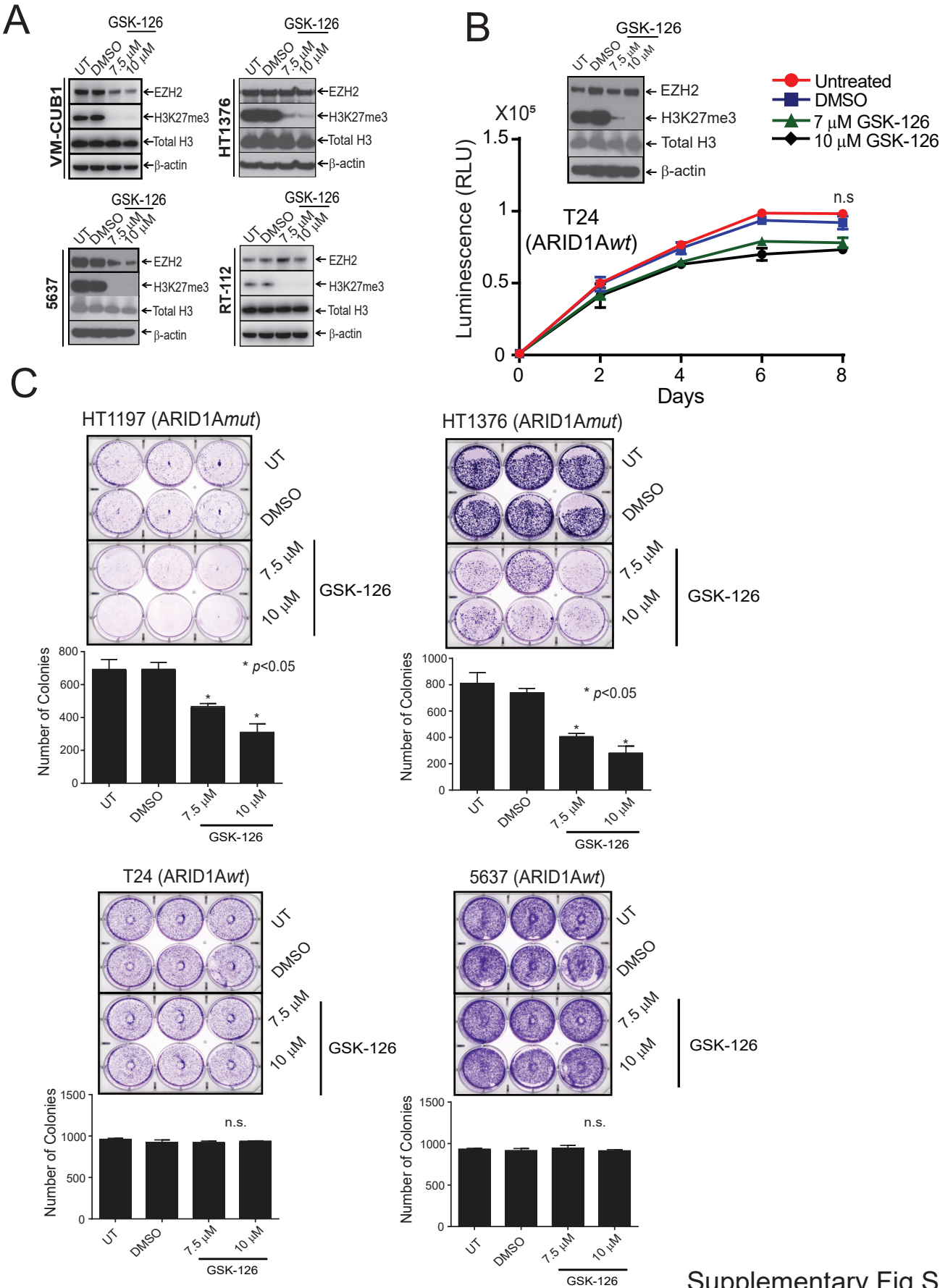


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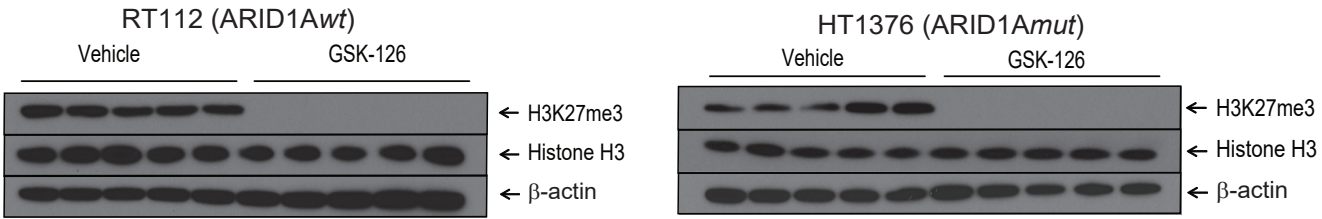
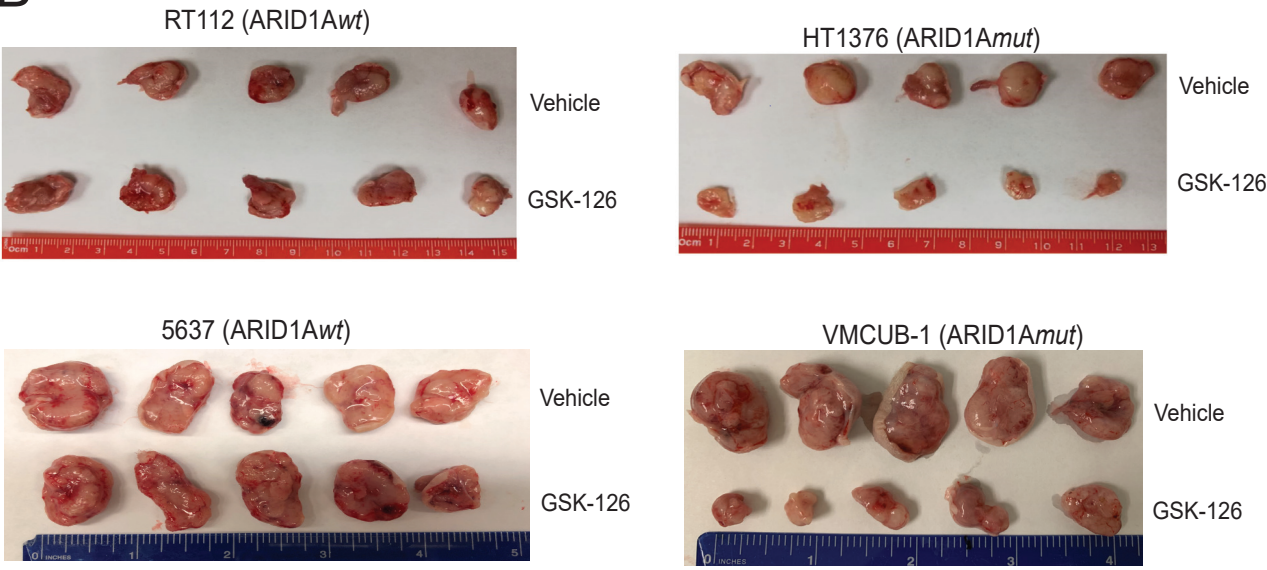


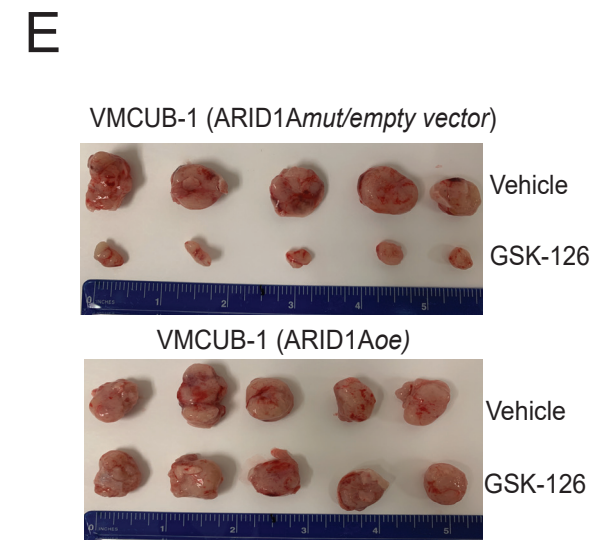
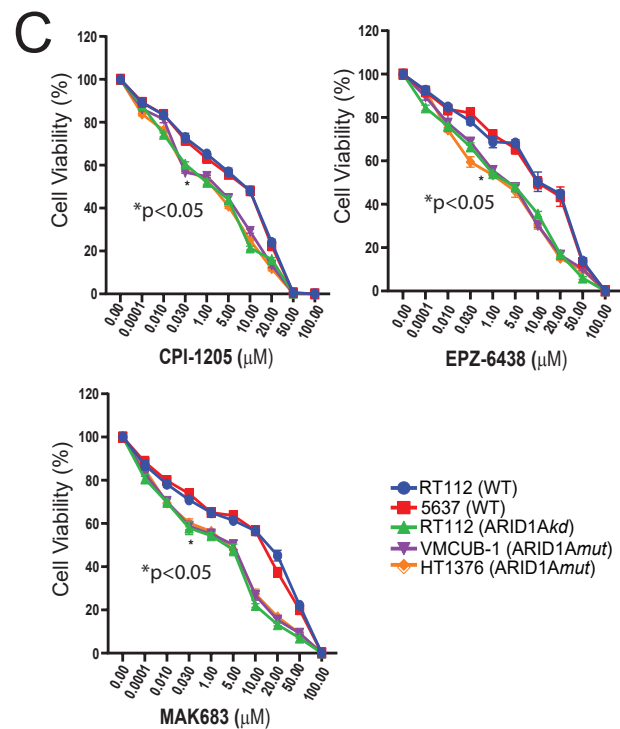
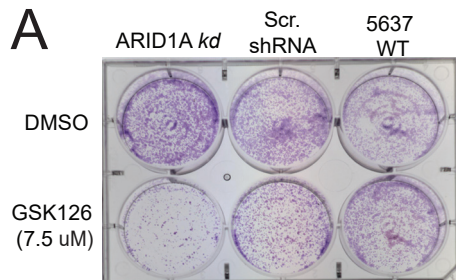
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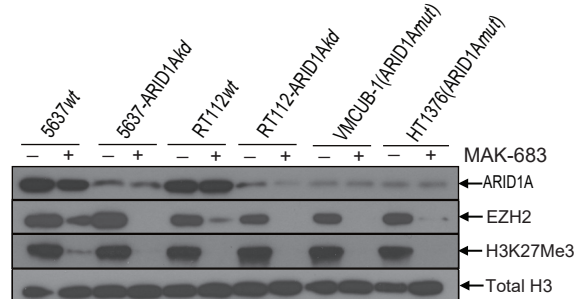
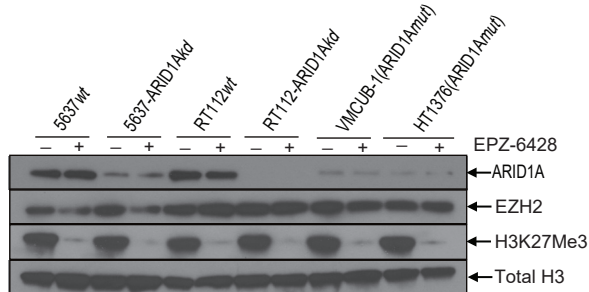
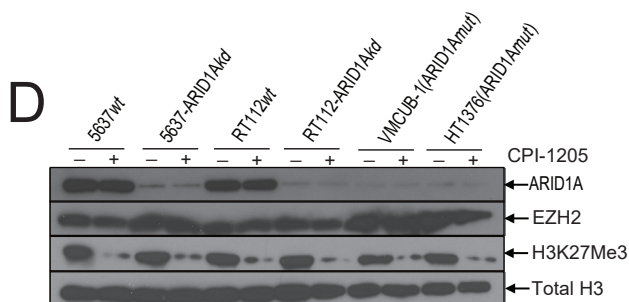
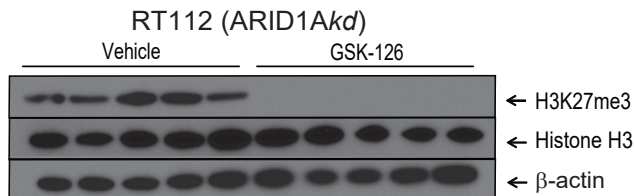
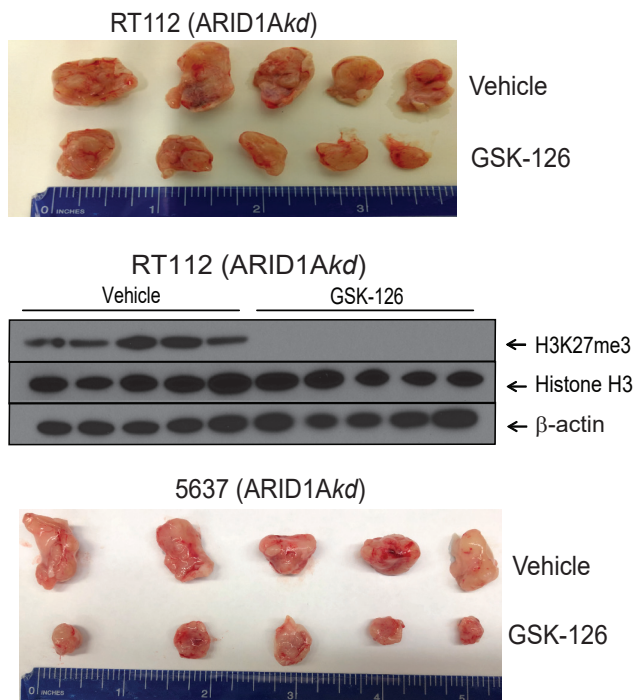


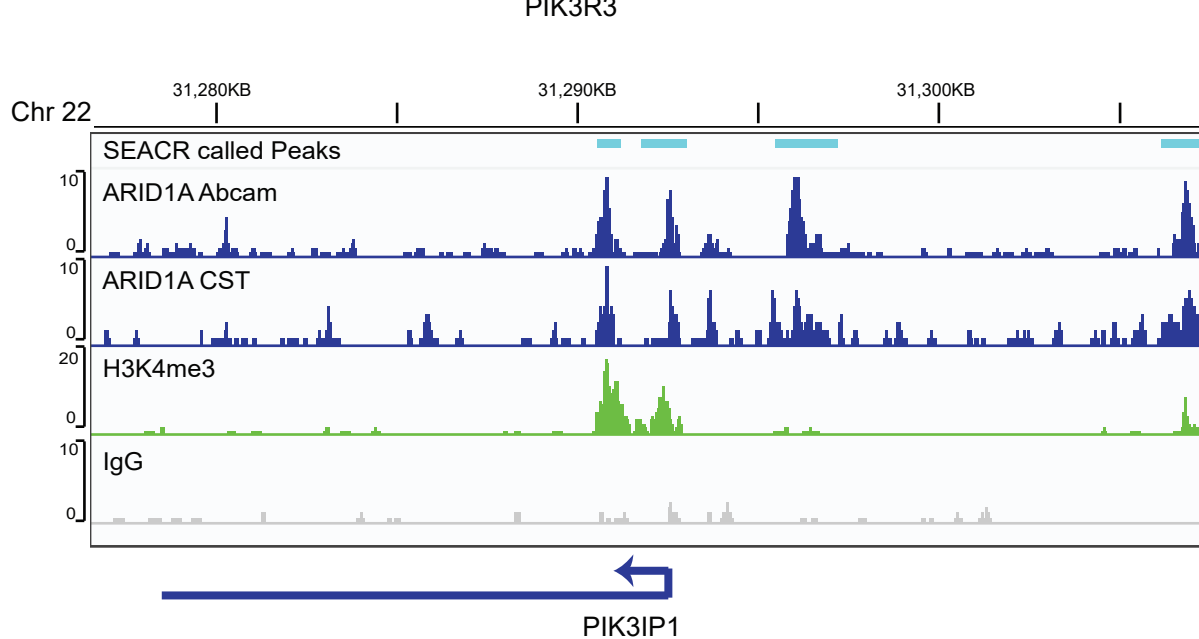
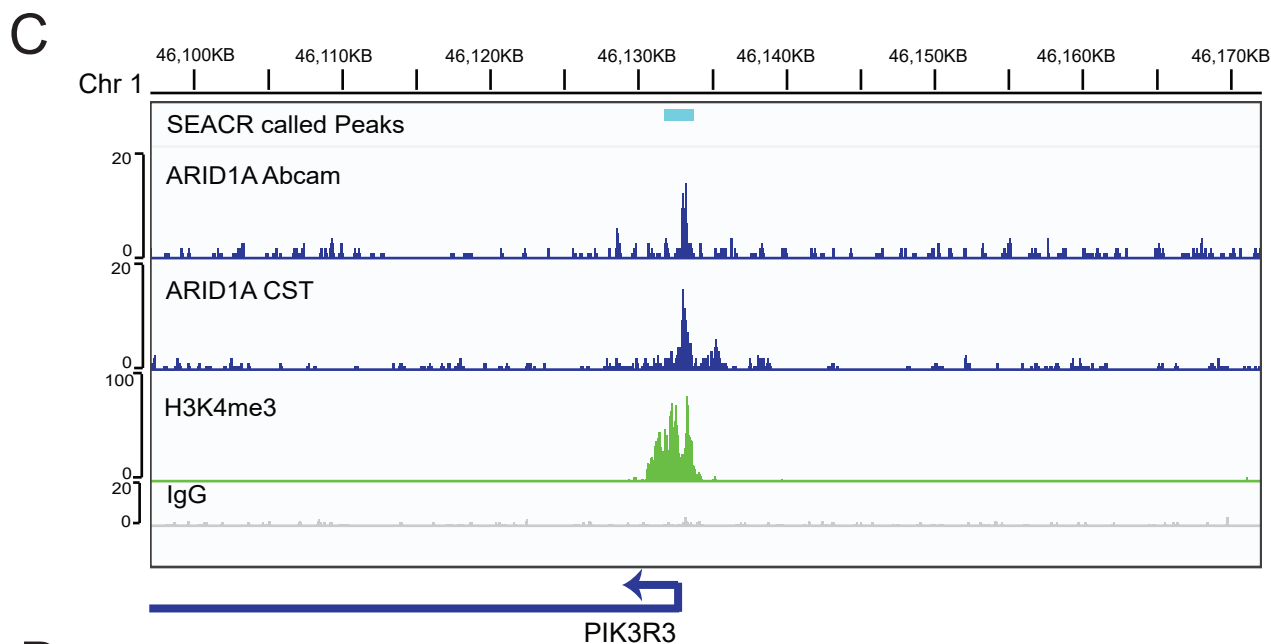
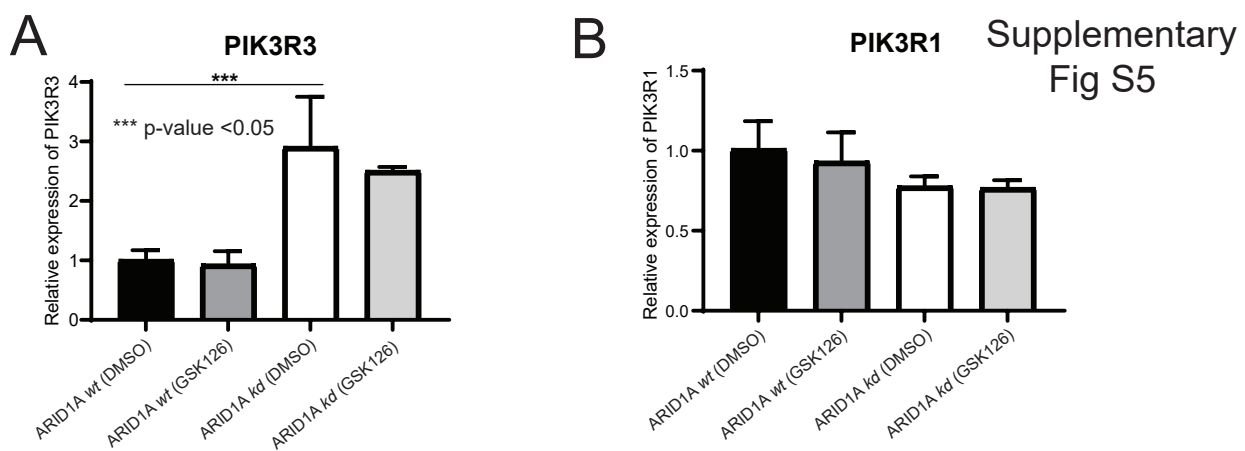
Supplementary Fig S2

A**Supplementary Fig S3****B**



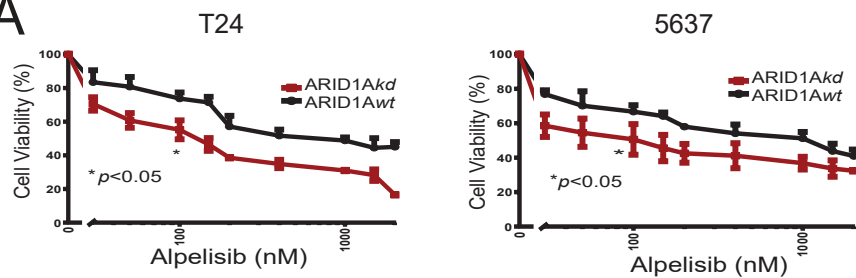
B Supplementary Fig S4



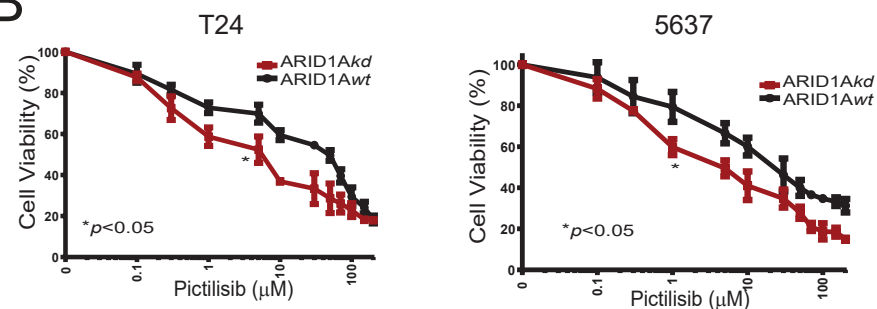


Supplementary Fig S6

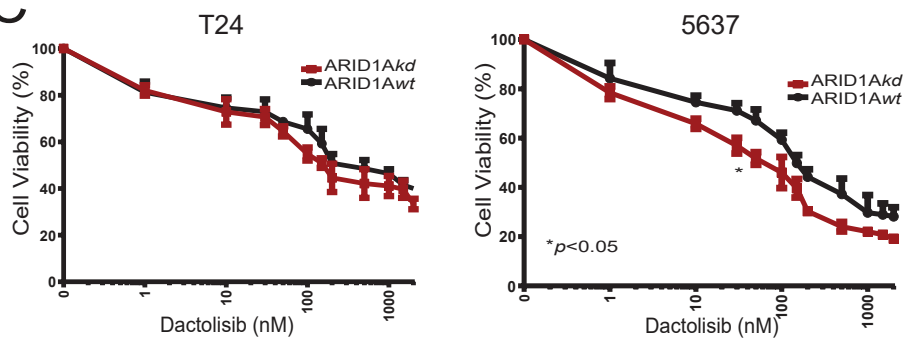
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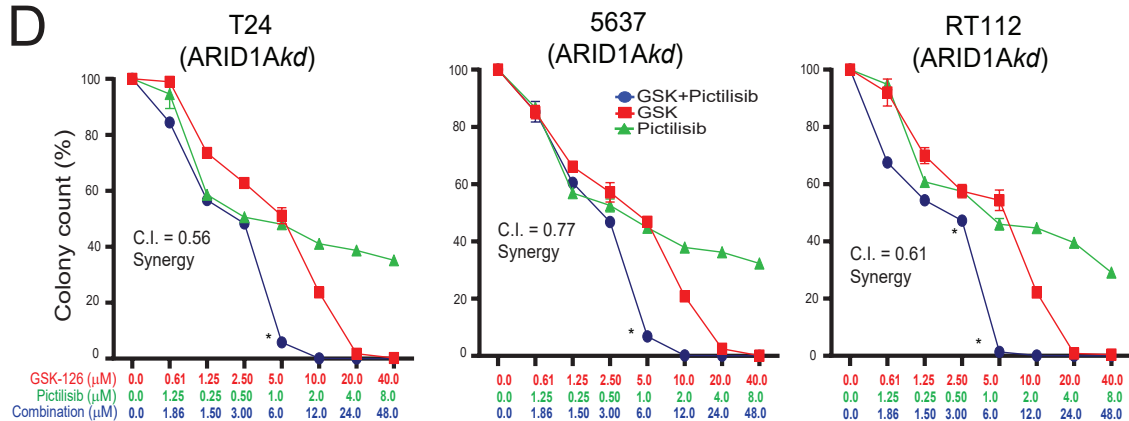
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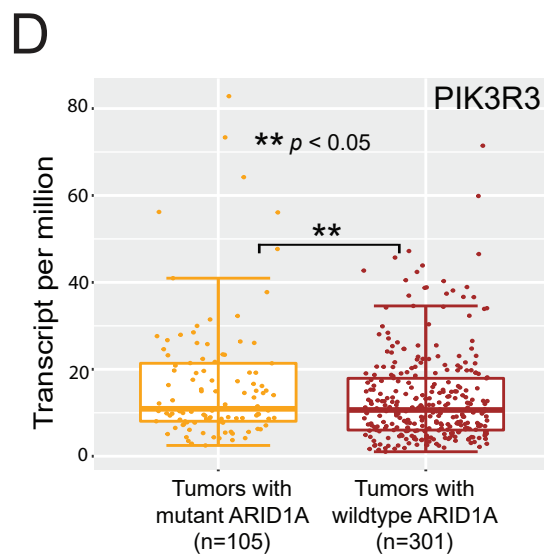
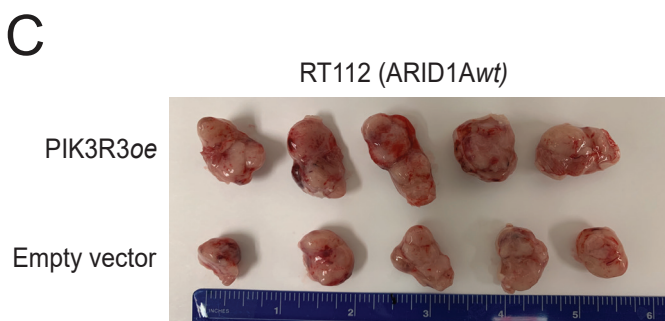
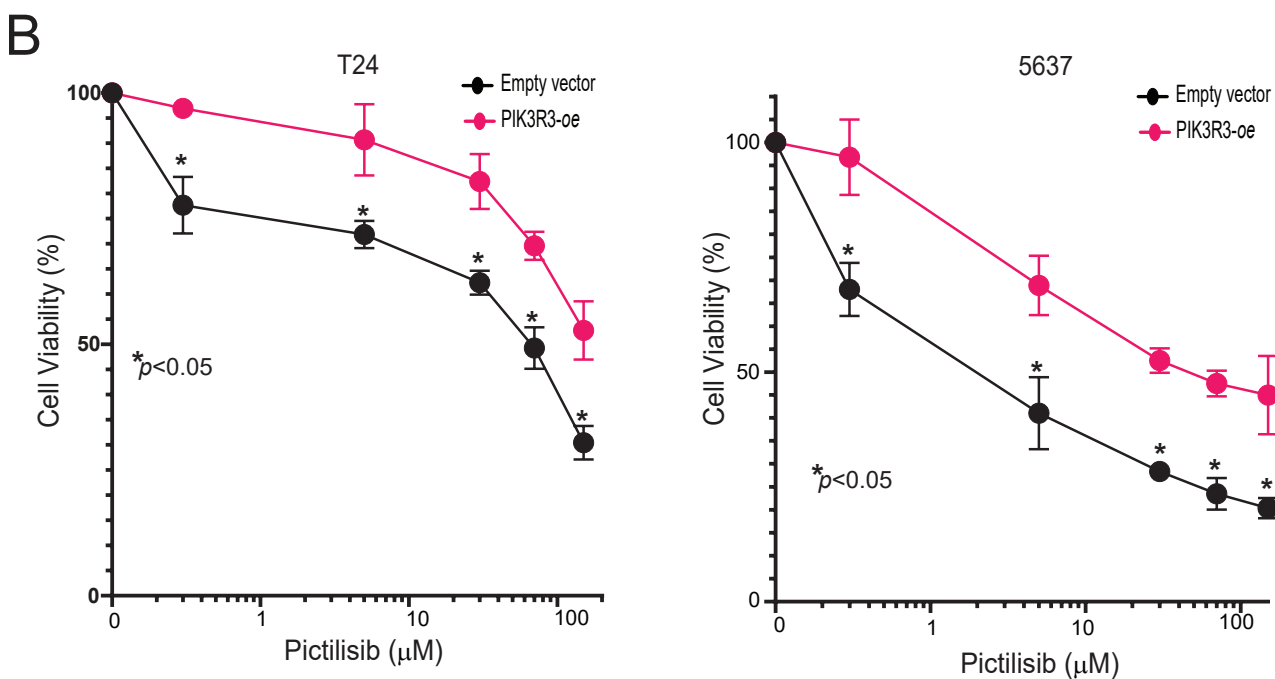
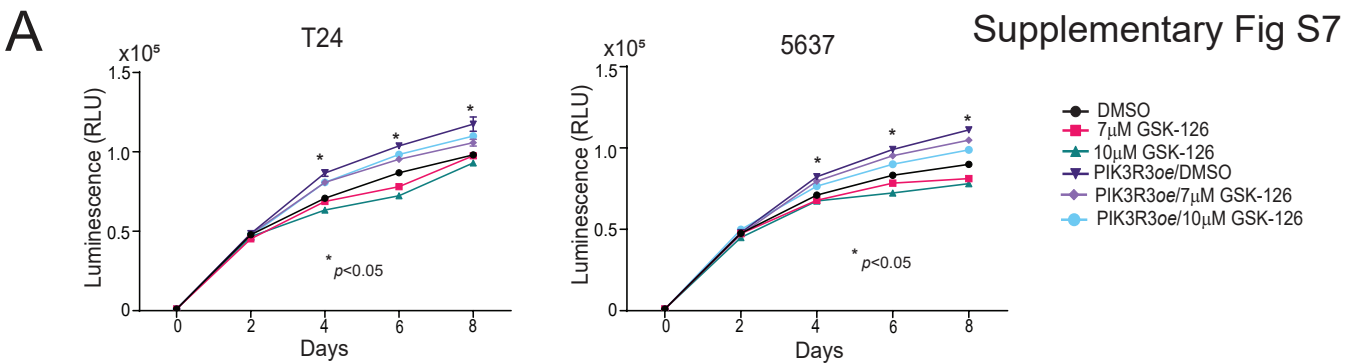


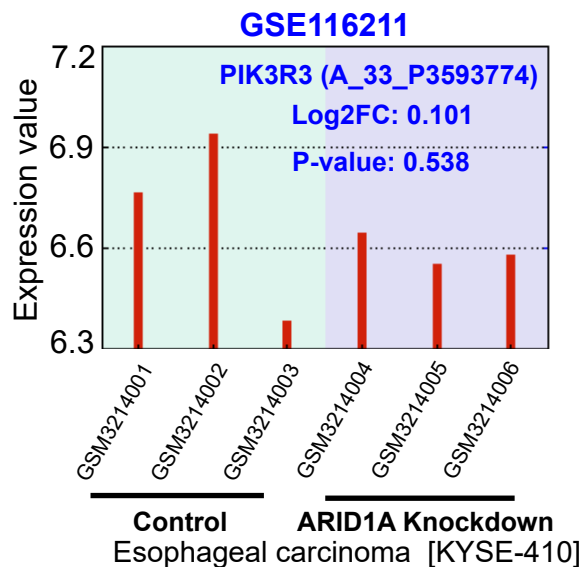
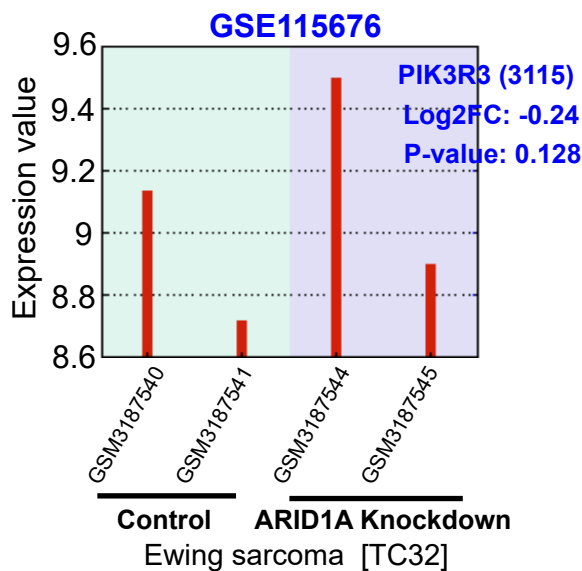
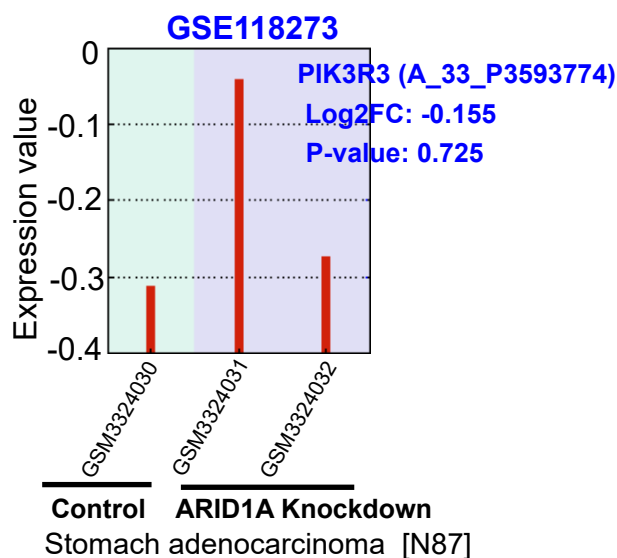
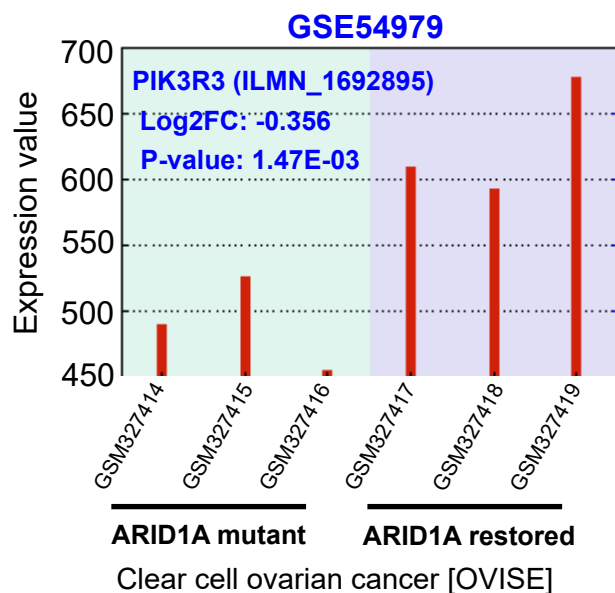
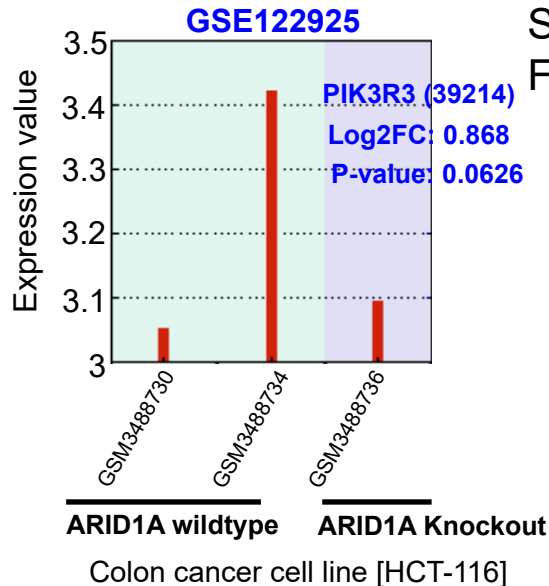
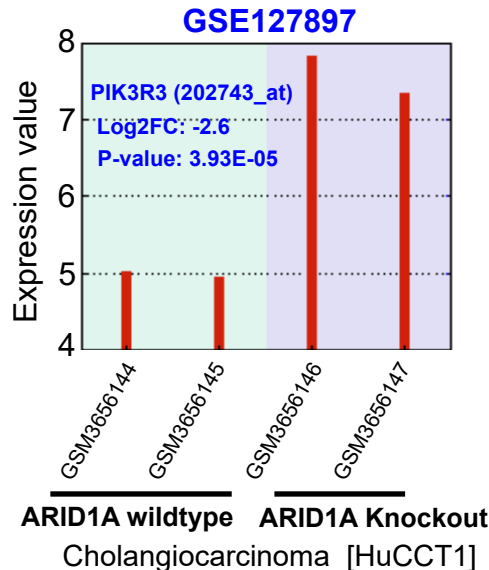
C



D







Supplementary Figure Legends

Supplementary Figure S1: Bladder cancer harbors high rates of *ARID1A* mutation, and high expression of EZH2. (A) Oncoplots showing prevalence of ARID1A and EZH2 mutations in muscle invasive and non-muscle invasive bladder carcinomas. Mutation profiles from three independent genomic sequencing data gathered from cBioPortal.org show a significant number of bladder tumors harbor truncating or missense mutations in ARID1A. (B) Box-whisker plot showing expression level of EZH2 in TCGA bladder cancers with wild-type and mutant ARID1A alleles. T-test was performed.

Supplementary Figure S2: ARID1A mutation is associated with increased GSK-126 sensitivity. (A) Immunoblot analysis indicating that GSK-126 inhibits H3K27me3 generation in all cell lines (from experiment in Figure 1C). (B) Cell viability time course with increasing concentrations of GSK-126 in T24 (ARID1A^{wt}) cell line. (C) Colony formation assay of ARID1A^{mut} or wild type bladder cancer cell lines in the presence and absence of GSK-126 (6d). Photographic images and bar plots are shown. UT = untreated. T-tests were performed.

Supplementary Figure S3: GSK-126 treatment effectively inhibits H3K27 trimethylation in xenograft tissues, and inhibits xenograft growth in ARID1A^{mut} cell lines. (A) Lysates from xenografts depicted in Figure 1D (n=5 per group) were used to perform immunoblots for H3K27me3, total H3 and β -actin in RT-112 (ARID1A^{wt}) and HT1376 (ARID1A^{mut}) cells. (B) Photographs of xenograft tumors (from Figure 1D) from mice inoculated with RT-112, 5637 (ARID1A^{wt}), or HT1376 or VMCUB-1 (ARID1A^{mut}) cells treated with and without GSK-126.

Supplementary Figure S4: ARID1A deficiency is necessary and sufficient for sensitivity to GSK-126. (A) Colony formation assay of 5637 ARID1A^{kd} or ARID1A^{wt} cells with or without GSK-126 treatment. (B) Photographs and immunoblots of xenograft tumors from mice (from Fig 2E) inoculated with RT112 (ARID1A^{kd}) or 5637 (ARID1A^{kd}). (C) Dose response cell viability experiments with various EZH2 inhibitors. Two-way ANOVA was performed comparing IC50 values of ARID1A^{wt} and ARID1A^{def} cells. (D) Immunoblots corresponding to cells in part (C) indicating effective EZH2 inhibition (via H3K27Me3) or degradation. (E) Photographs of endpoint xenograft tumors (from Fig 2H) from mice inoculated with VMCUB-1 (ARID1A^{mut}/Empty vector) and VMCUB-1 (ARID1A reconstituted/overexpression) cells with and without GSK-126 treatment.

Supplementary Figure S5: ARID1A presence and effects at PIK3R3 and PIK3IP1 loci (A) Quantitative RT-PCR was performed to determine relative expression of PIK3R3 and (B) PIK3R1 in ARID1A^{wt} and ARID1A^{kd} RT112 cells in absence and presence of GSK-126 (5 μ M) after 24h treatment. No difference was seen after GSK-126 treatment. T-test was performed. (C) CUT & RUN analysis of PIK3R3 genomic locus using two different ARID1A antibodies (Abcam and Cell Signaling Technologies (CST)), H3K4me3 as an active mark, and IgG negative control. SEACR called peaks confirmed that ARID1A is present at PIK3R3 promoter. (D) CUT & RUN analysis of PIK3IP1 genomic locus using antibodies as in (C). SEACR called peaks confirmed that ARID1A is present at the PIK3IP1 promoter.

Supplementary Figure S6: ARID1A deficiency sensitizes bladder cancer cells to PI3K inhibitors. (A-C) Cell viability dose-response analysis in 5637 and T24 cells with ARID1A^{Kd} or empty vector (ARID1A^{wt}) using alpelisib (a PI3K alpha-selective inhibitor), pictilisib (a PI3K class I selective inhibitor), and dactolisib (a dual PI3K/mTOR inhibitor) (all 48h). T-tests were performed on IC50 values. (D) Colony formation dose-response assay using increasing doses of GSK-126, pictilisib, or combination showed that the combination was synergistic. Combination Indices (CI) were calculated by the Chou-Talalay method.

Supplementary Figure S7: PIK3R3 overexpression increases growth kinetics of ARID1A^{wt} cells. (A) ARID1A^{wt} cells T24 and 5637 with PIK3R3 overexpression (oe) show increased proliferation at baseline (compared with empty vector), but no increased sensitivity to GSK-126. Two-way ANOVA was performed. (B) Dose-response cell viability assay using T24 and 5637 ARID1A^{wt} cells with PIK3R3 overexpression (oe) or empty vector showing that PIK3R3 overexpression causes resistance to the PI3K inhibitor, pictilisib (48 h). T-tests were performed. (C) Photographs of end-point xenograft tumors (from Fig 7G) from mice inoculated with RT112 (ARID1A^{wt}) over-expressing PIK3R3 compared to empty vector. (D) Box-plot analysis of TCGA bladder cancer data comparing PIK3R3 mRNA levels between ARID1A^{mut} and ARID1A^{wt} tumors. T-test was performed.

Supplementary Figure S8: PIK3R3 expression pattern in ARID1A mutated/altered cancer cell lines. GEO2R profile graphs showing expression level of PIK3R3 in ARID1A altered ovarian cancer (OVISE), colon cancer (HCT-116), cholangiocarcinoma (HuCCT1), stomach adenocarcinoma (N87), Ewing sarcoma (TC32) and esophageal carcinoma (KYSE-410) cell lines. Expression value from each sample is shown in addition to probe identifiers, log 2 fold change and t-test p-value. A 2 fold change was considered significant.

Supplementary Methodology

In silico data analysis

Using cBioPortal, the mutation profiles of ARID1A and EZH2 in muscle-invasive and non-muscle invasive bladder cancer were obtained (1). cBioPortal provides user-friendly graphical interface to analyze whole exome sequencing datasets from The Cancer Genome Atlas Project and other published studies (2-4). To study EZH2 gene expression profile in invasive bladder carcinoma patients, TCGA level 3 RNA-seq data (including “raw_read_count” and “scaled_estimate” for each sample) was downloaded for all primary tumor and normal samples using TCGA-Assembler (5). Transcript per million values for each gene was obtained by multiplying scaled estimate by 1,000,000. Using patient ID from cBioPortal, primary tumors were categorized based on ARID1A mutation status. Boxplot was generated using R (<https://cran.r-project.org/>).

We studied the expression pattern of PIK3R3 in ARID1A altered/mutated cancer cell lines. Microarray datasets related to OVISE [GSE54979], HCT-116 [GSE122925], N87 [GSE118273], HuCCT1 [GSE127897], TC32 [GSE115676] and KYSE410 [GSE116211] cell lines were analyzed using GEO2R tool (<https://www.ncbi.nlm.nih.gov/geo/geo2r/>)(6-11). Differential expression analysis between ARID1A mutated/alterd and control samples was performed for each study separately. Profile graphs, log2 fold change and p-value was obtained for probes related to PIK3R3.

Colony formation assay

Bladder cancer cells were seeded at 800 cells per well of 6-well plates (triplicate) and incubated at 37 °C with 5% CO₂ for 7–10 days while treating with GSK126 or pictilisib at the doses indicated every two days. Here both untreated and DMSO treated cells served as controls. Colonies were fixed with 10% (v/v) ethanol for 30 min and stained with crystal violet (Sigma-Aldrich, St Louis, MO, USA) for 20 min. Then, the photographs of the colonies were taken using Amersham Imager 600RGB (GE Healthcare Life Sciences, Pittsburgh, PA, USA). Colony quantification was carried out using ImageQuant TL Colony v.8.1 software (GE Healthcare Life Sciences).

qRT-PCR analysis

RNA from cultured cells was extracted with Direct-zol RNA miniprep kit (Zymo Research). For qRT-PCR, cDNA was generated using a High-Capacity cDNA Reverse Transcription kit (Applied Biosystems). qRT-PCR analysis was performed using the Taqman Gene Expression Master reagent mixed with Taqman primers and analyzed with the QuantStudio™ 6K Flex Real-Time PCR System (Applied Biosystems). mRNA expression levels were normalized to human TATA-binding protein (TBP) or GAPDH, and the normalized cycle threshold (Ct) values were quantified using the double delta Ct analysis. qRT-PCR data represent relative expression. In general, controls in experimental data are normalized to a value of 1. Indicated Taqman primers were predesigned from Applied Biosystems as follows: *PIK3R1* (Hs00933163_m1), *PIK3R3* (Hs01103591_m1), *GAPDH* (Hs02786624_g1), *TBP* (Hs00427620_m1).

Protein extraction and western blotting of Xenograft tissues

The mice were euthanized, and the xenograft tumors were excised and weighed. Next, fragments of xenograft tumors were lysed in RIPA lysis buffer (Thermo Scientific, IL, USA) and boiled for 10 min at 90 °C. Protein concentrations were measured using the BCA assay. Approximately, 50 µg of protein extract from fresh surgical xenograft tissues were separated by 10% SDS-polyacrylamide gel electrophoresis (SDS-PAGE). The gels were then electrotransferred onto polyvinylidene difluoride (PVDF) membranes (EMD Millipore, Billerica, MA, USA).

CUT&RUN library preparation

RT112 cells were harvested using Accutase (Gibco) and slow frozen in aliquots containing 500,000 cells in serum-containing DMEM+10% DMSO. For CUT&RUN, cell aliquots were rapidly thawed at 37°C and washed as directed. Samples were processed in a single batch using the CUTANA CUT&RUN Kit (Epiccypher) according to the manufacturer's instructions and using 1 ng e. coli spike-in DNA per sample. After DNA purification, DNA libraries were prepared using the NEBNext Ultra II DNA Library Prep Kit for Illumina (NEB) and NEBNext Multiplex Oligos for Illumina (96 Unique Dual Index Primer Pairs, NEB) with samples randomized into two library preparation batches. Library quality was assessed using Qubit (Thermo Fisher) and 4150 TapeStation System (Agilent). All libraries were pooled and sequenced on a NextSeq 550 (Illumina) using a NextSeq 500/550 v2.5 Mid Output Kit (150 cycles, paired-end, Illumina).

CUT&RUN data processing and analysis

Sequencing reads were demultiplexed and converted to FASTQ format using bcl2fastq2 (Illumina). Adapter sequences were trimmed (cutadapt) and reads were aligned to both the human (GRCh38) and E. Coli K-12 MG1655 genomes (bwa-mem2). PCR duplicates were removed (Picard) and non-uniquely mapping reads were filtered out (samtools). Replicates were scaled to each other using the ratio of E. coli reads to total reads and scaled BigWig files were generated for data visualization (bedtools bamcoverage). Alignment files were converted to paired-end BED files and then full-fragment BED files (bedtools bamtobed, awk, cut) and scaled BedGraph files were generated using the same scaling strategy as for the BigWig files (deeptools genomecoverage). Peaks were called from the scaled BedGraph files using SEACR (12) in relaxed mode to facilitate broad discovery of potential peaks; the top 0.001% of peaks were retained for analysis. Replicate peaks were merged (bedtools merge), annotated (ChIPpeakAnno) for the closest TSS, and visualized in IGV. The CUT&RUN data generated in this study are publicly available in Gene Expression Omnibus (GEO) at GSE203033.

Table 1

Antibody	Dilution	Supplier	Cat. No.	RRID
ARID1A	1:1000 (IB), 1:10 (CUT&RUN, CNR)	Cell Signaling Technologies, Denver, MA	12354S	AB_2637010

ARID1A	1:100 (CNR)	Abcam, Waltham, MA	ab182560	AB_2889973
BRG1	1:1000	Cell Signaling Technologies, Denver, MA	49360S	AB_2728743
BRM	1:1000	Cell Signaling Technologies, Denver, MA	11966S	AB_2797783
BAF47	1:1000	Cell Signaling Technologies, Denver, MA	91735S	AB_2800172
EZH2	1:1000	Cell Signaling Technologies, Denver, MA	5246S	AB_10694683
IgG	1:50 (CNR)	EpiCypher, Durham, NC	13-0042	
H3K4me3	1:50 (CNR)	EpiCypher, Durham, NC	13-0041	
H3K27me3	1:2000	Cell Signaling Technologies, Denver, MA	9733S	AB_2616029
p-AKT (T308)	1:1000	Cell Signaling Technologies, Denver, MA	13038	AB_2629447
p-AKT (S473)	1:1000	Cell Signaling Technologies, Denver, MA	9271	AB_329825
AKT	1:1000	Cell Signaling Technologies, Denver, MA	9272	AB_329827
Phospho-mTOR	1:1000	Cell Signaling Technologies, Denver, MA	2971	AB_330970
Phospho-p70 S6 Kinase	1:1000	Cell Signaling Technologies, Denver, MA	9205	AB_330944
p70 S6 Kinase	1:1000	Cell Signaling Technologies, Denver, MA	9202	AB_331676
Phospho-4E-BP1 (Thr37/46)	1:1000	Cell Signaling Technologies, Denver, MA	2855	AB_560835
4E-BP1	1:1000	Cell Signaling Technologies, Denver, MA	9452	AB_331692
PI3 Kinase p110 α	1:1000	Cell Signaling Technologies, Denver, MA	4255	AB_659888
PI 3-kinase p110 β	1:1000	Santa Cruz Biotechnology, CA	376641	AB_11150840
p38 MAPK	1:1000	Cell Signaling Technologies, Denver, MA	9212	AB_330713
Phospho-p38 MAPK (Thr180/Tyr182)	1:1000	Cell Signaling Technologies, Denver, MA	4631	AB_331765
p44/42 MAPK (Erk1/2)	1:1000	Cell Signaling Technologies, Denver, MA	4695	AB_390779
Phospho-p44/42 MAPK (Erk1/2) (Thr202/Tyr204)	1:1000	Cell Signaling Technologies, Denver, MA	4376	AB_331772
Phospho-SAPK/JNK (Thr183/Tyr185)	1:1000	Cell Signaling Technologies, Denver, MA	4668	AB_823588
SAPK/JNK	1:1000	Cell Signaling Technologies, Denver, MA	9252	AB_2250373
Cleaved Caspase 3	1:1000	Cell Signaling Technologies, Denver, MA	9661	AB_2341188
LC3B (D11) XP	1:1000	Cell Signaling Technologies, Denver, MA	3868	AB_2137707
PTEN	1:1000	Cell Signaling Technologies, Denver, MA	9559	AB_390810
PIK3IP1	1:500	Proteintech Group, Inc, Rosemont, IL	16826-1-AP	AB_2163333

PIK3R1 (p85 α /p55 α /p50 α)	1:1000	Abcam, Waltham, MA	ab191606	AB_2891324
PIK3R2 (p85 β)	1:1000	Abcam, Waltham, MA	ab28356	AB_777259
PIK3R3 (p55 γ)	1:1000	Santa Cruz Biotechnology	sc-376615	AB_11150683
Histone H3	1:2000	Cell Signaling Technologies, Denver, MA	4499	AB_10544537
Ubiquitin	1:1000	Life Sensors	VU-101	AB_2716558
β -actin	1:20000	Proteintech Group, Inc, Rosemont, IL	60008	AB_2883409
GAPDH	1:20000	Proteintech Group, Inc, Rosemont, IL	60004	AB_2107436
Anti-rabbit	1:5000	Proteintech Group, Inc, Rosemont, IL	SA00001-2	AB_2722564
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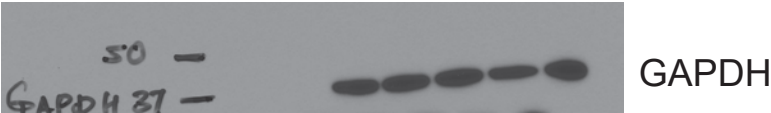
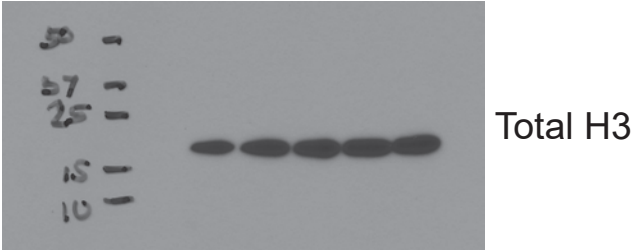
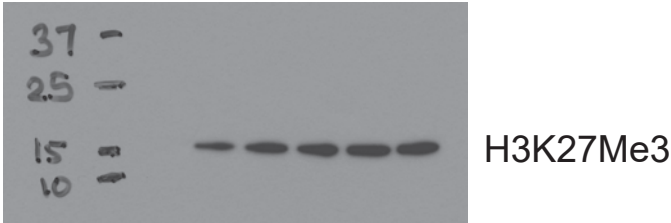
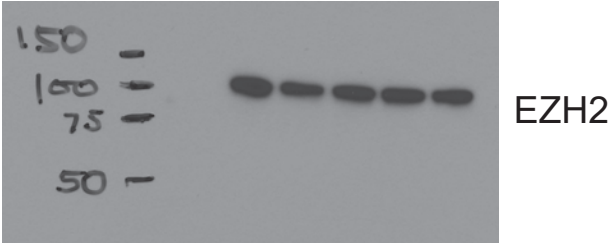
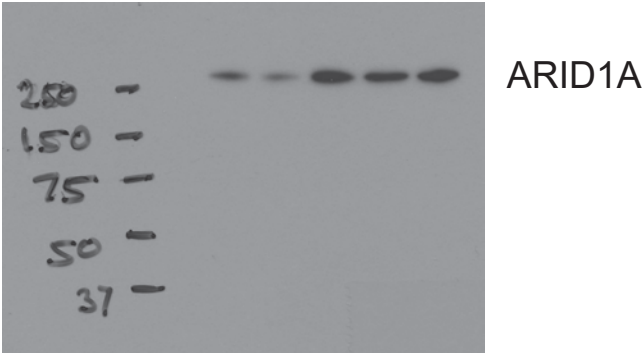
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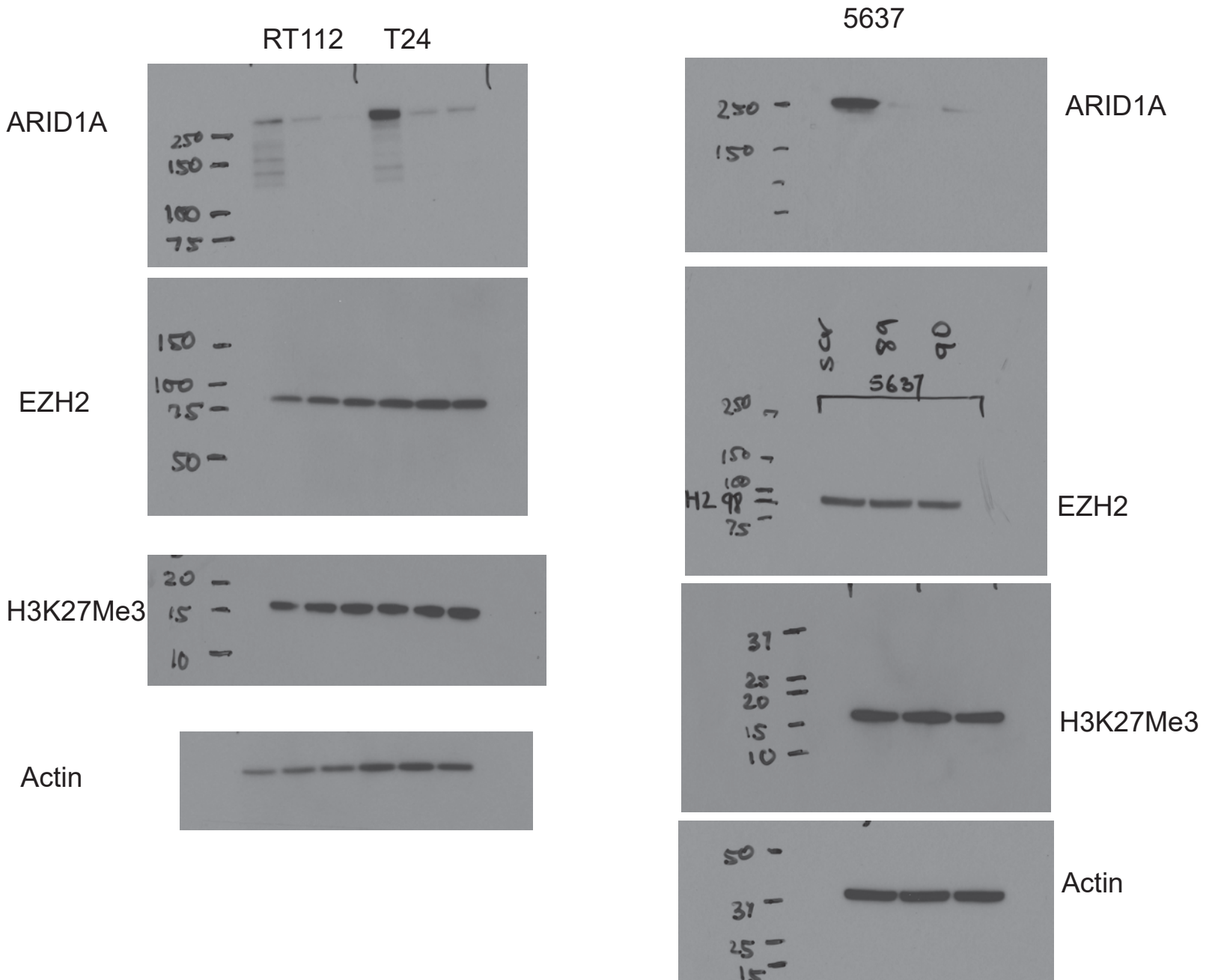
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2.	ON-TARGETplus Human SMARCB1 siRNA (Gene id:6598)	L-010536-00-0005
3.	ON-TARGETplus Human SMARCA2 siRNA (Gene id:6595)	L-017253-00-0005
4.	ON-TARGETplus Human SMARCA4 siRNA (Gene id:6597)	L-010431-00-0005

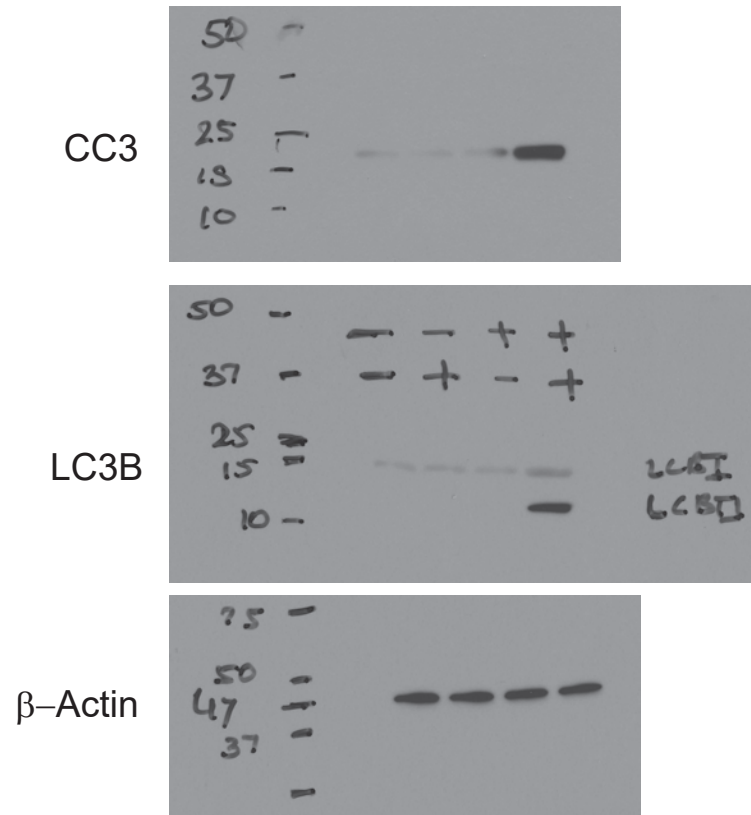
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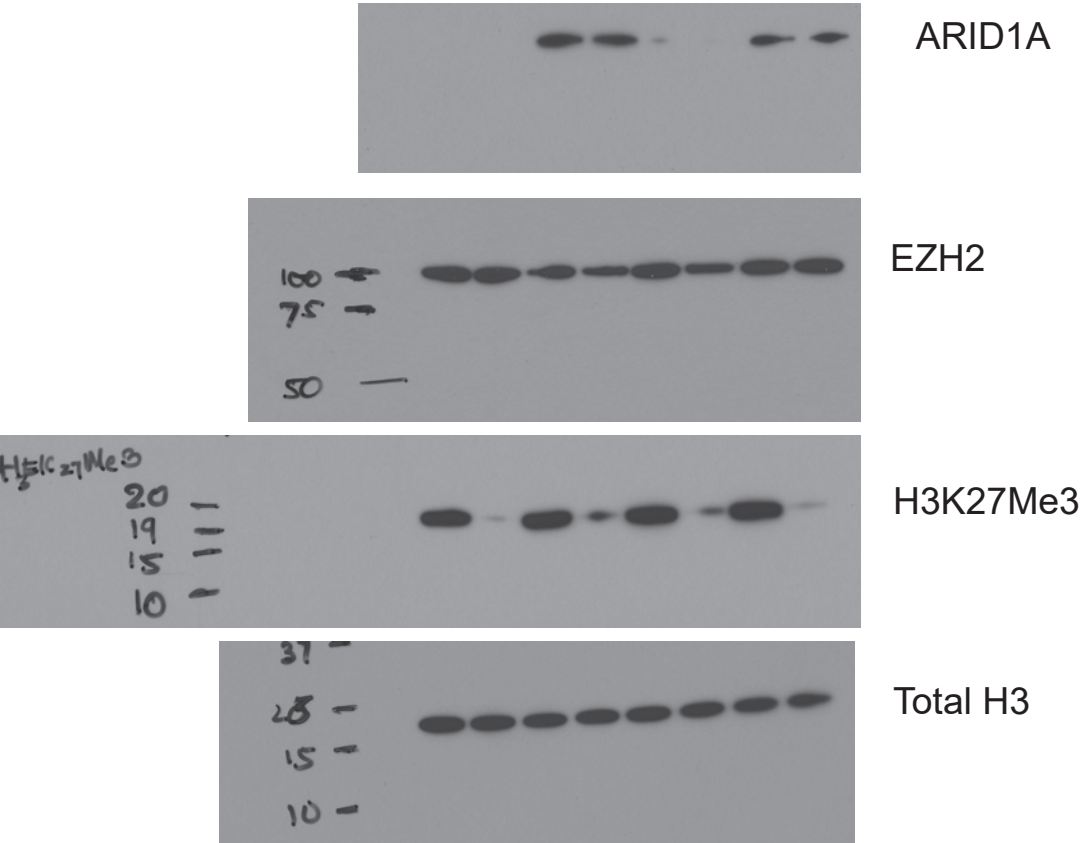
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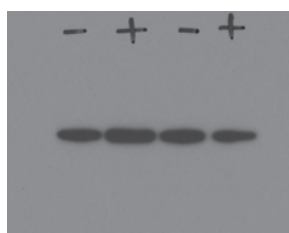
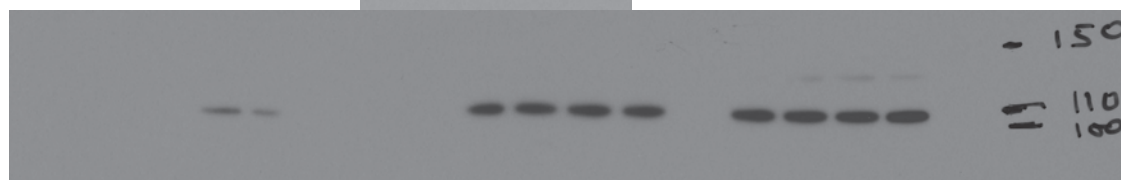
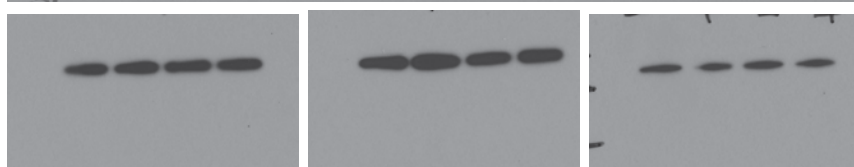
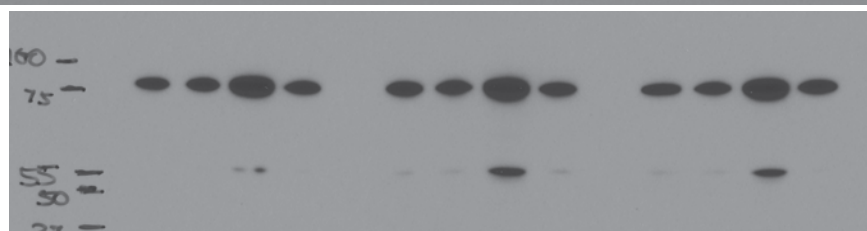
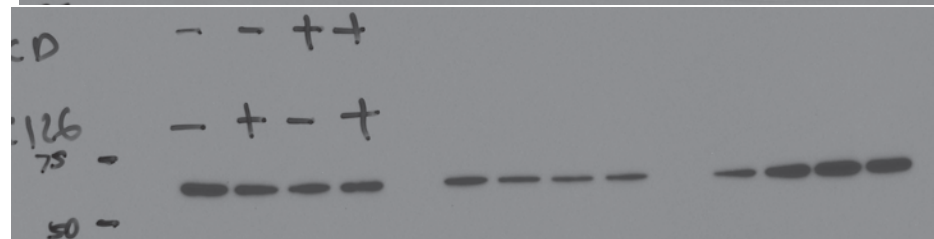
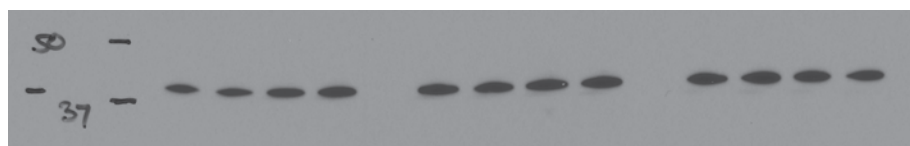


T24

5637

RT112

Uncut blot Figure 3C

P110 α Second Exposure (T24)

T24

5637

RT112

P110 β

pmTOR

p4EBP1 (T37/46)

Total 4EBP1 (T37/46)

pS6K1

Total S6K1

H3K27Me3

Histone

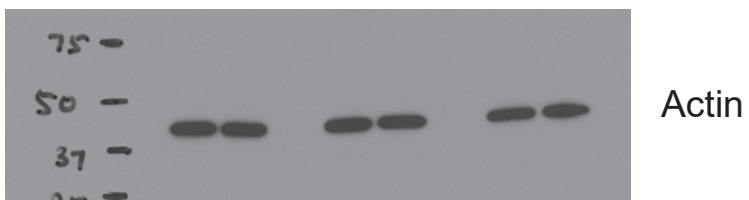
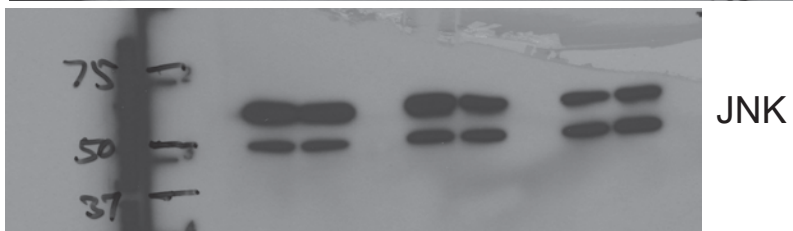
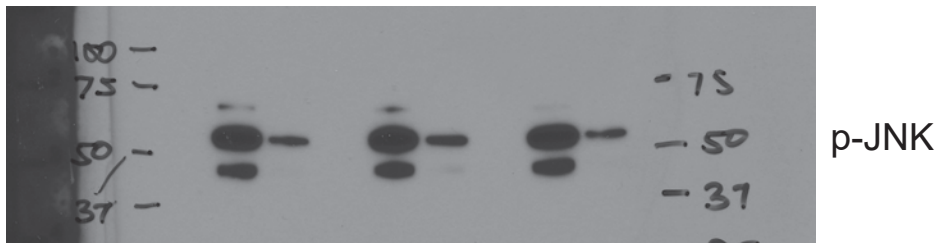
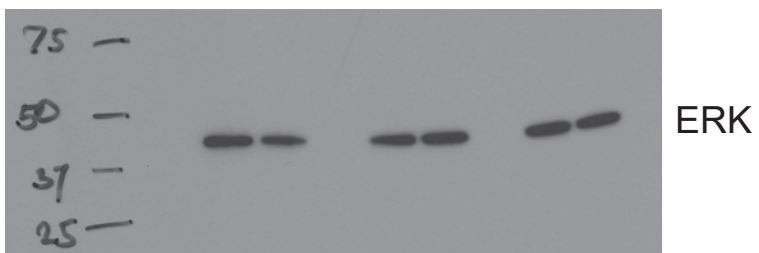
actin

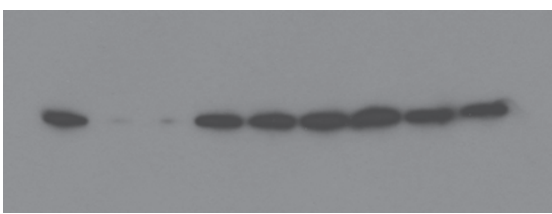
Exposure 1



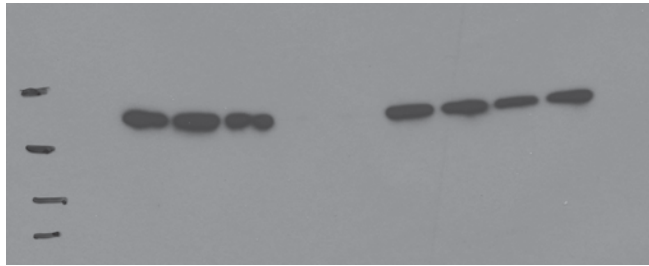
Uncut blot Figure 3D

Exposure 2

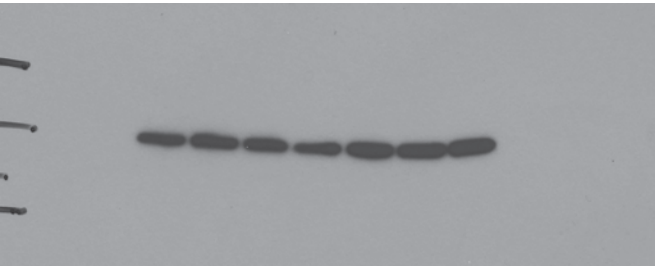




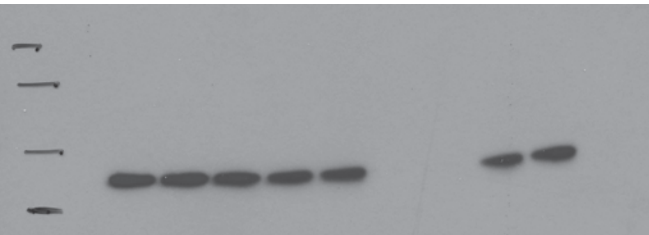
ARID1A_siRNA



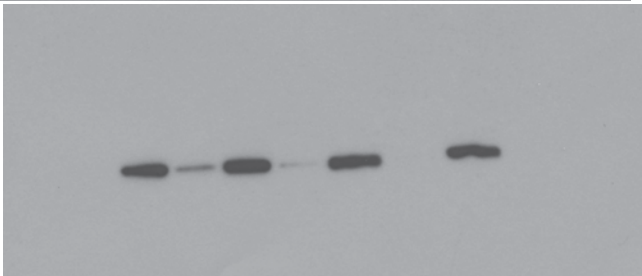
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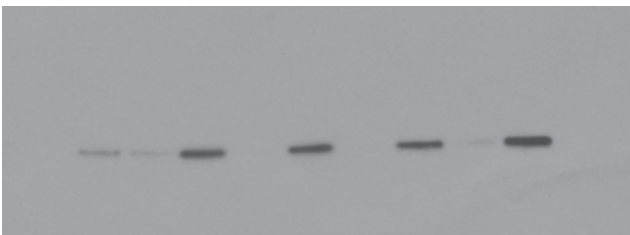
BRM_siRNA



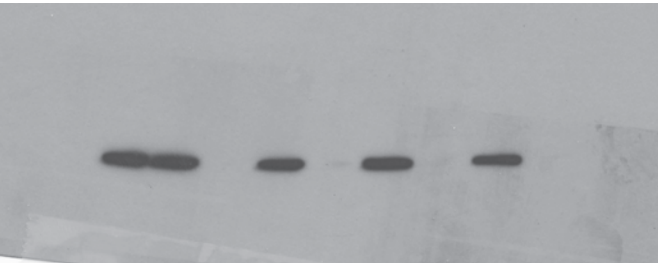
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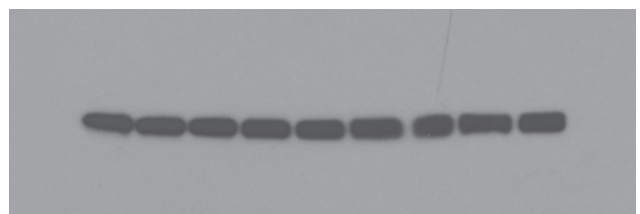
PIK3R3



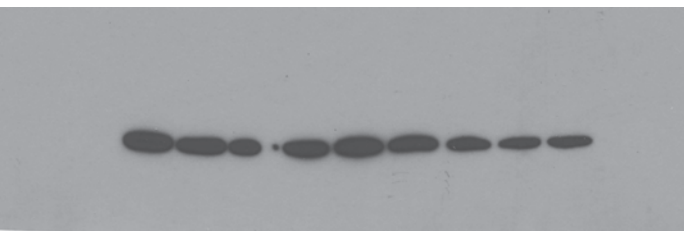
PIK3IP1



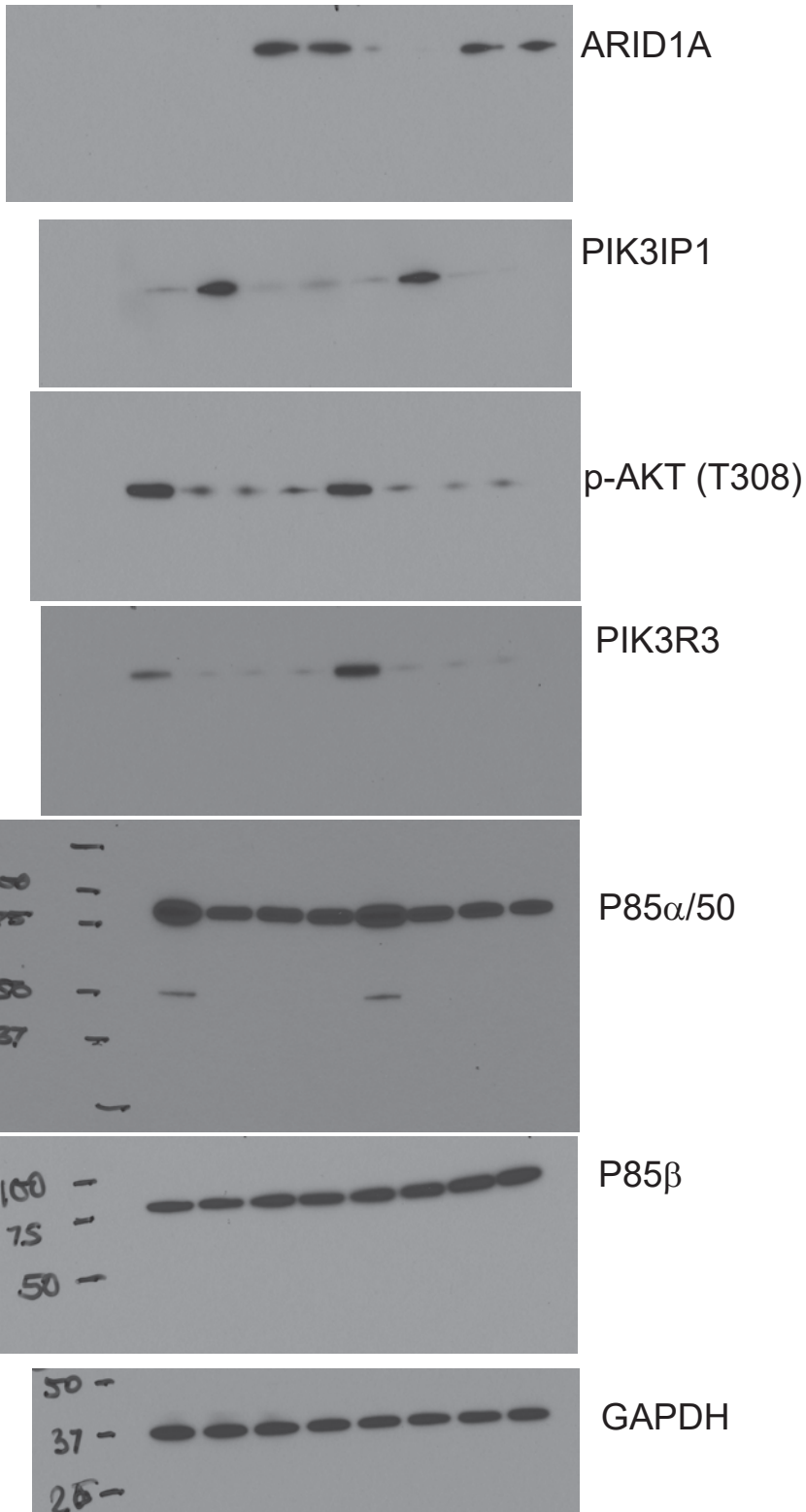
H3k27Me3



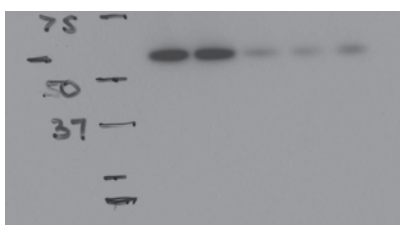
actin



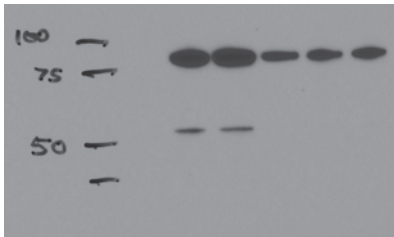
Total Histone



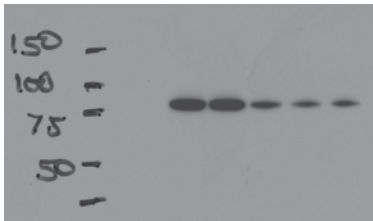
Uncut blot Figure 4B



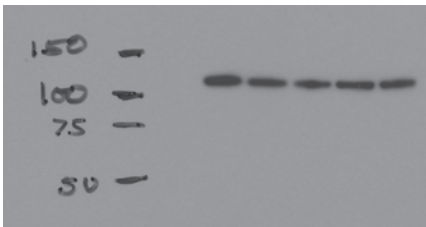
PIK3R3



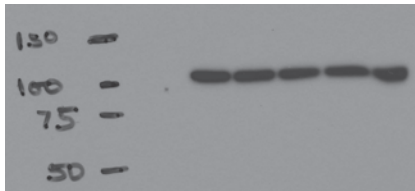
p85α/p50α



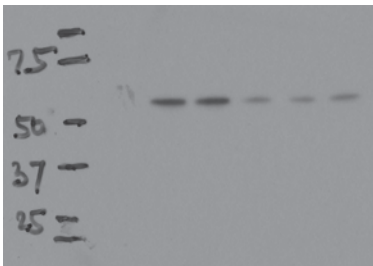
p85β



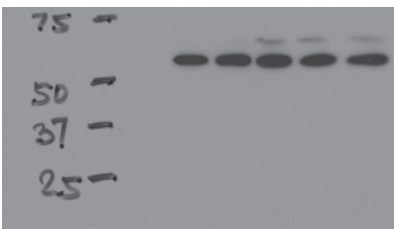
p110α



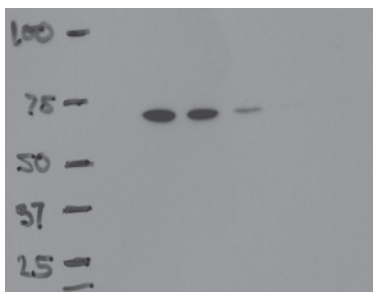
p110β



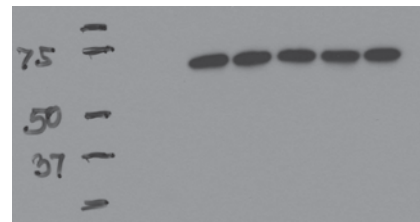
p-AKT



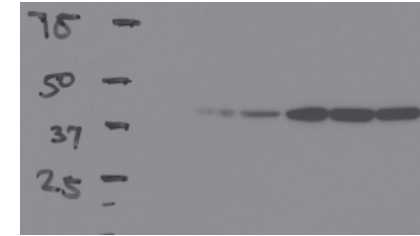
AKT



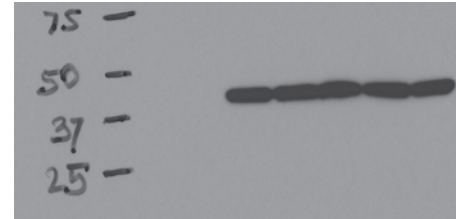
p-pS6K



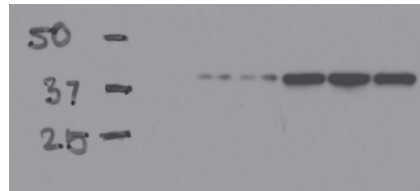
Total pS6K



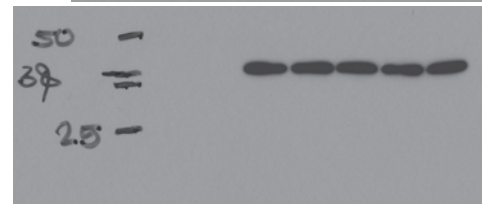
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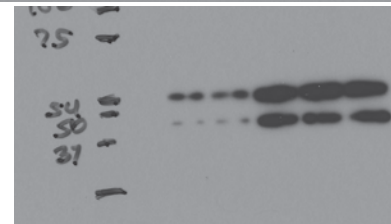
Total ERK



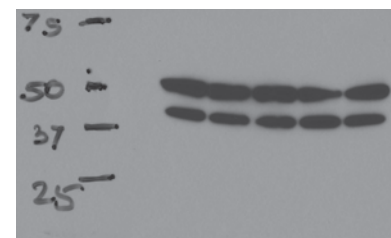
p-p38



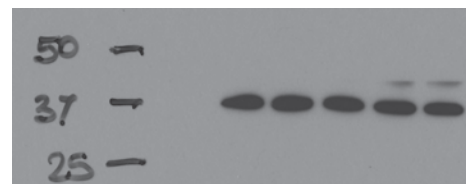
Total p38



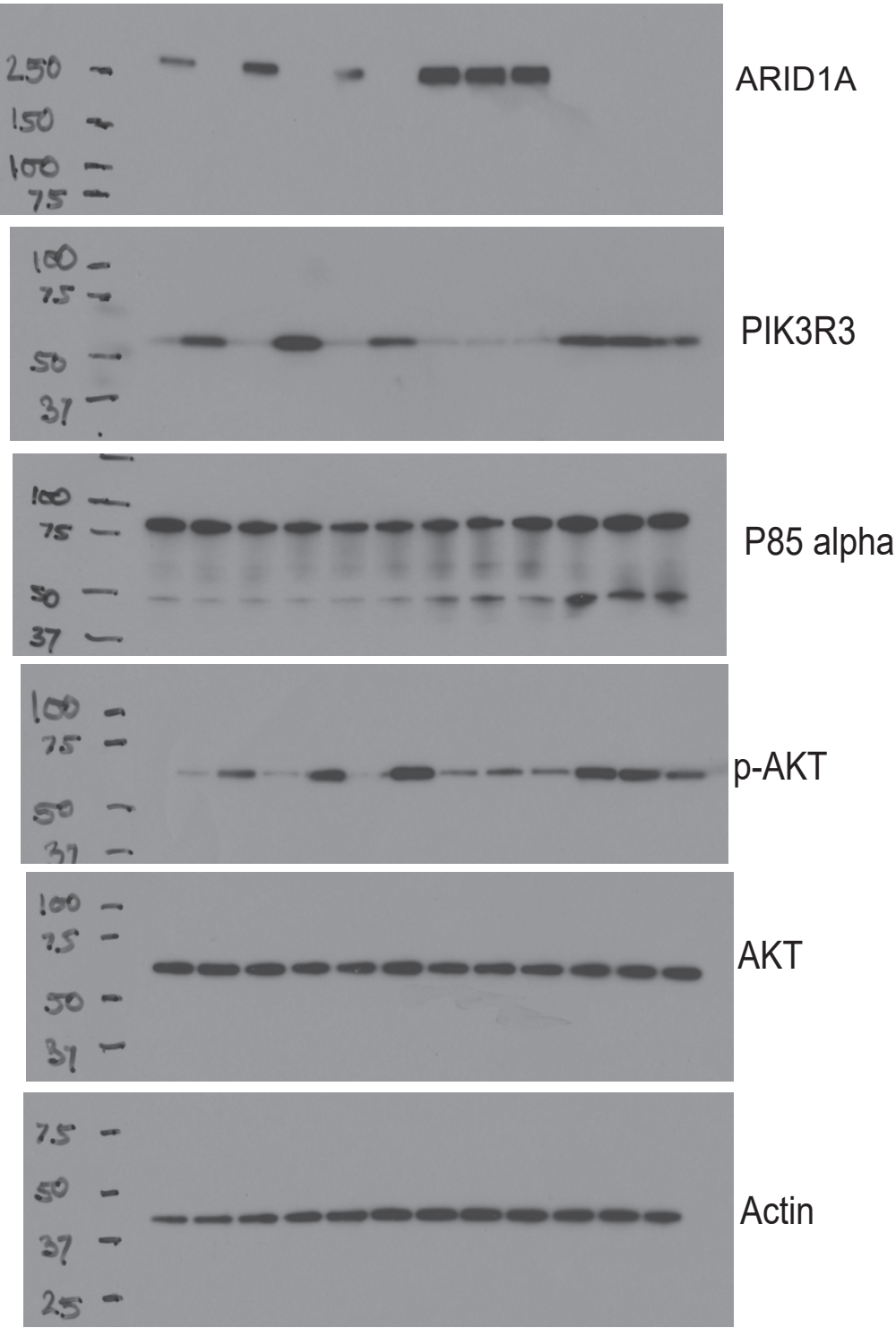
p-JNK

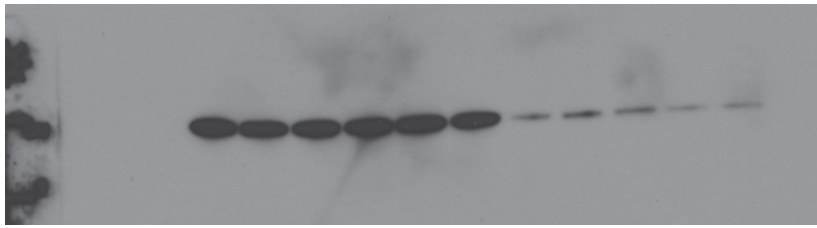


Total JNK



GAPDH

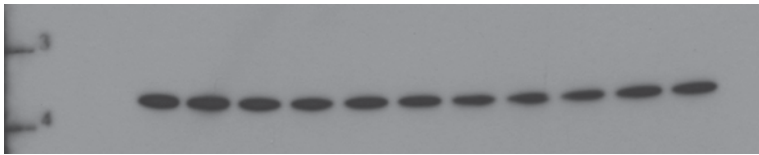




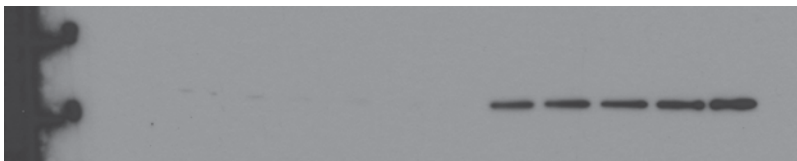
ARID1A



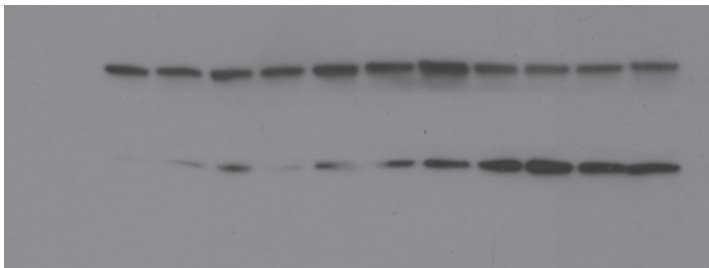
p-AKT (T308)



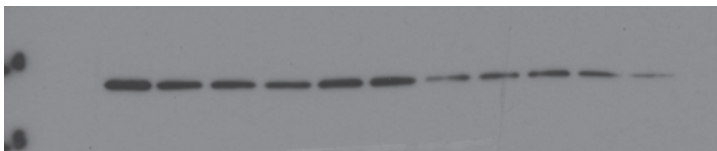
Total AKT



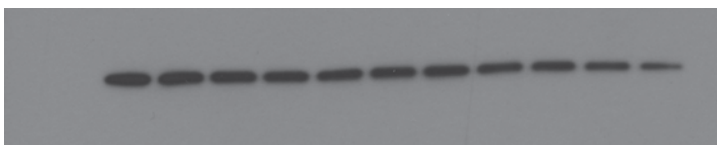
PIK3R3



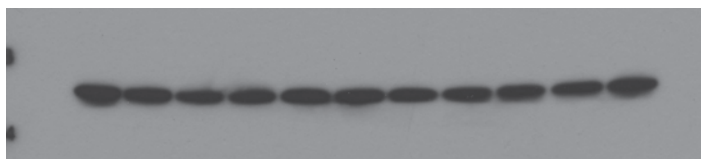
p85alpha/p50



p-ERK



ERK

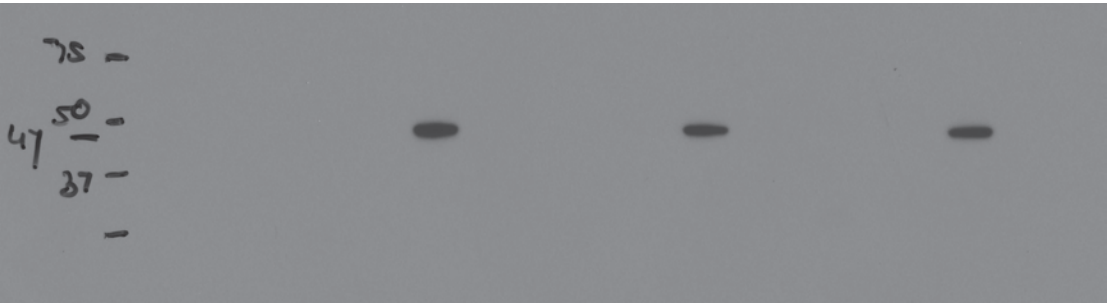


β -actin

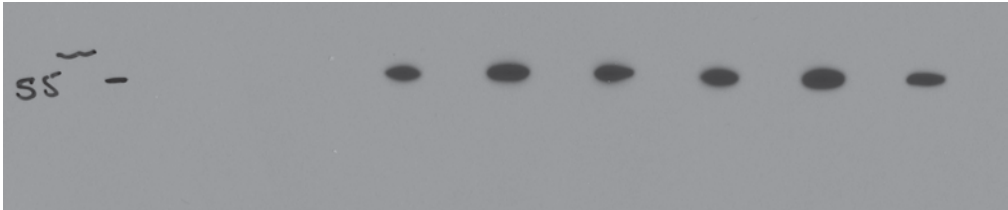
T24

5637

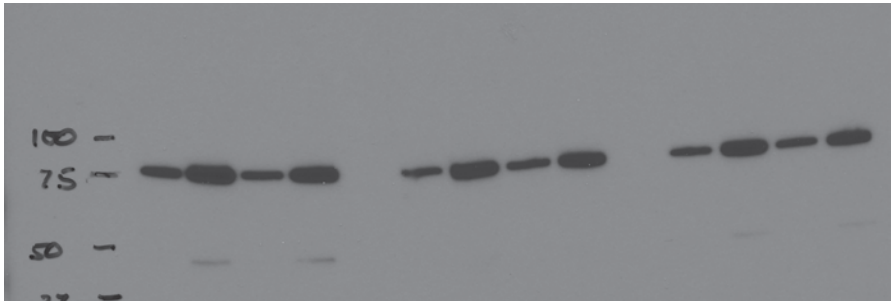
RT112



PIK3IP1

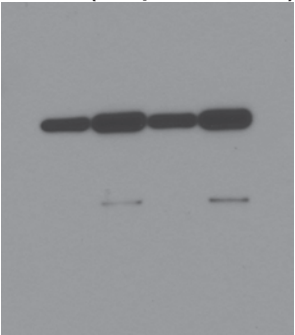


PIK3R3

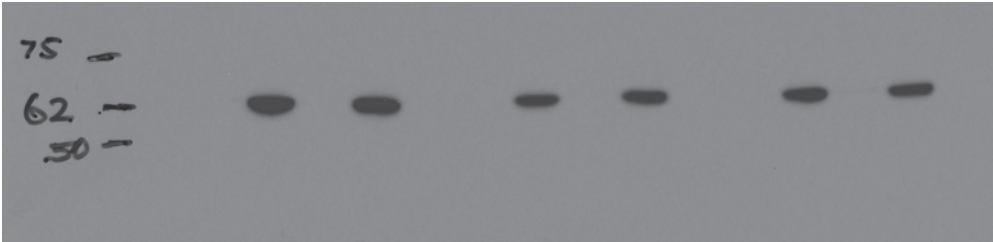


P85 alpha/P50

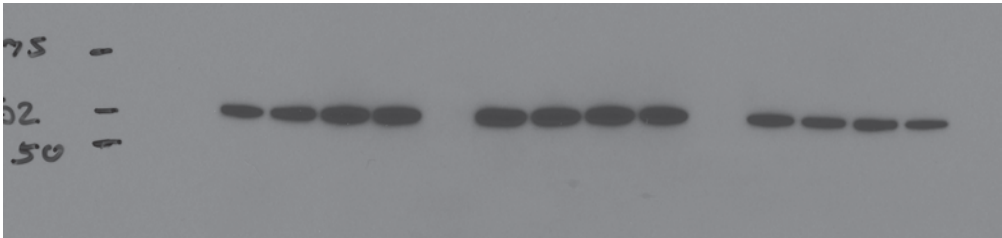
5637(Exposure 2)



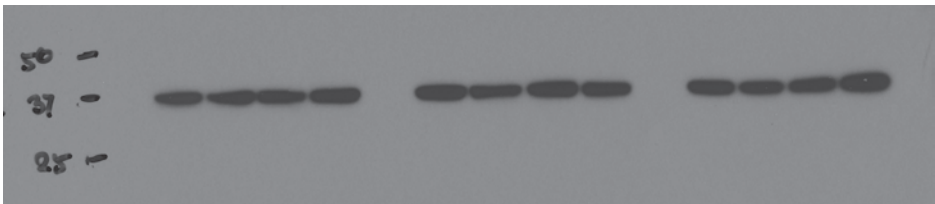
P85 alpha/P50



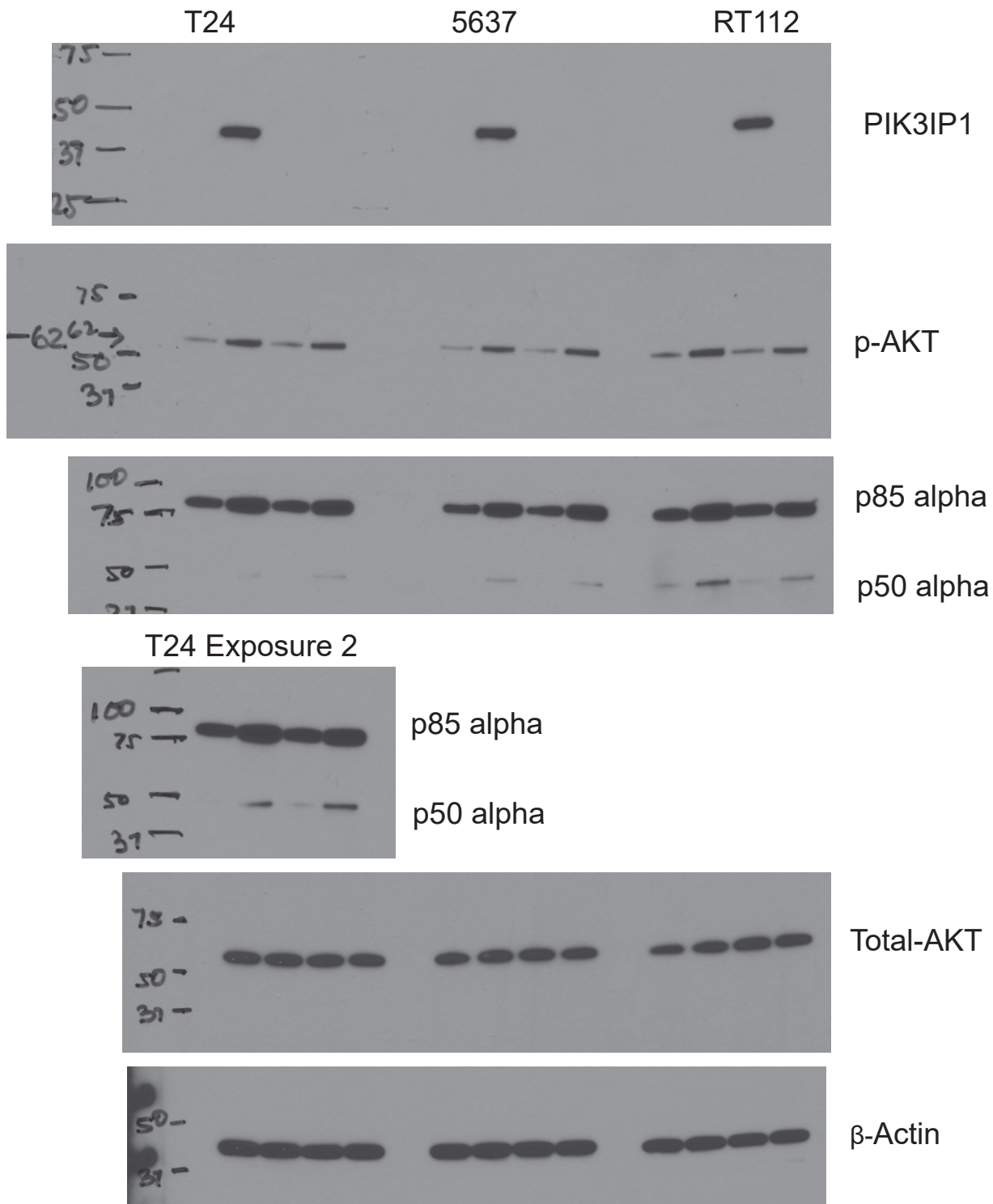
p-AKT (T308)

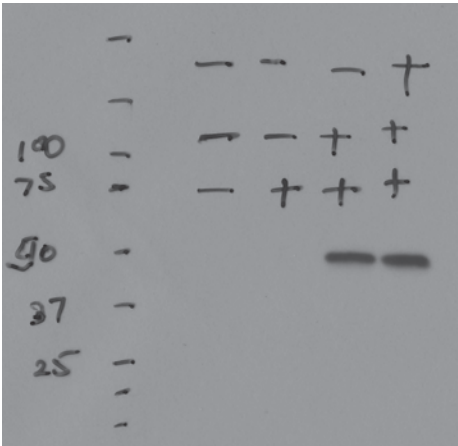


AKT

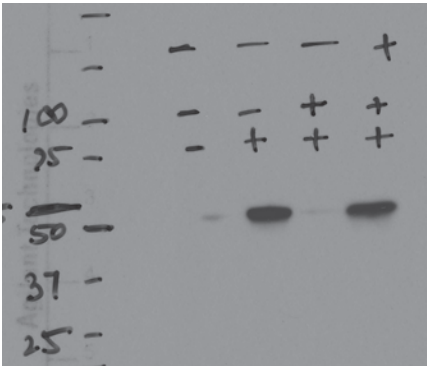


GAPDH

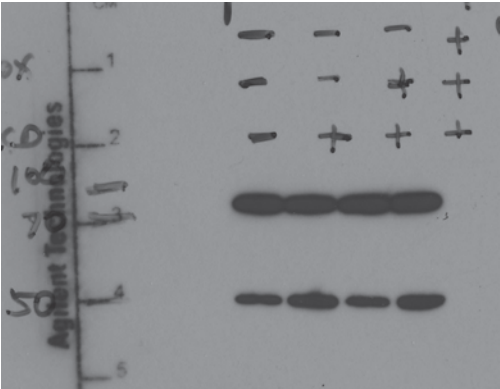




PIK3IP1

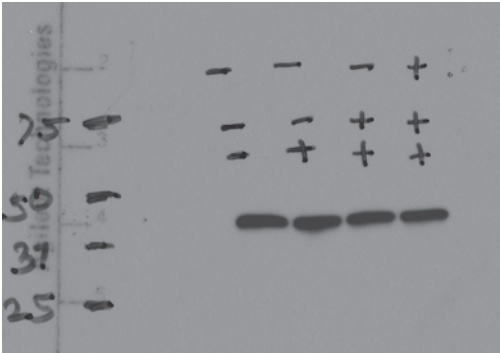


PIK3R3

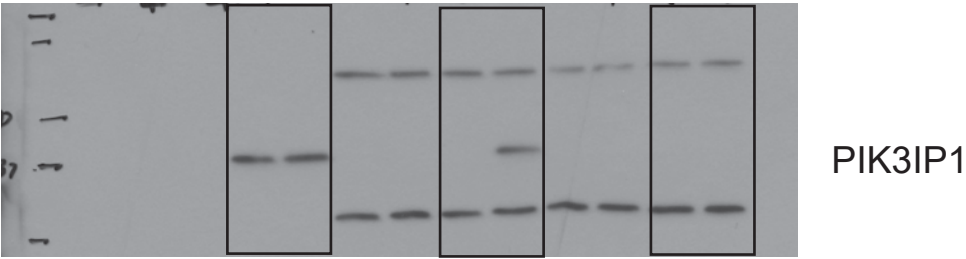
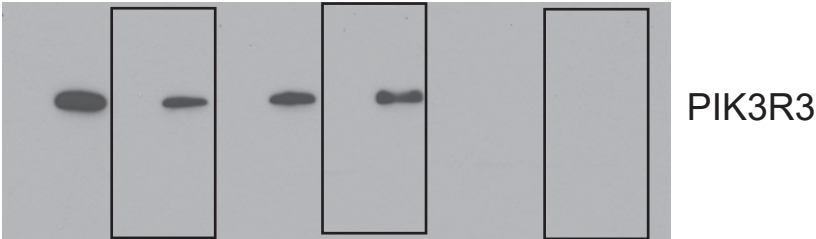
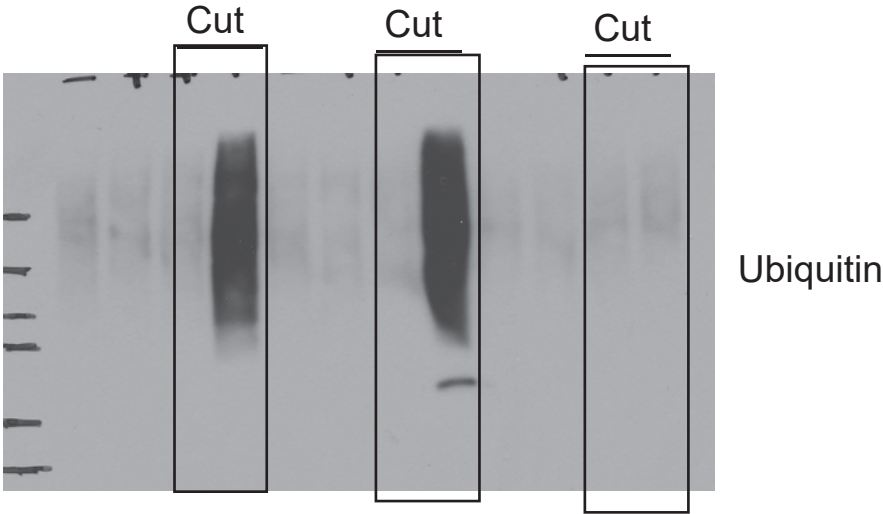


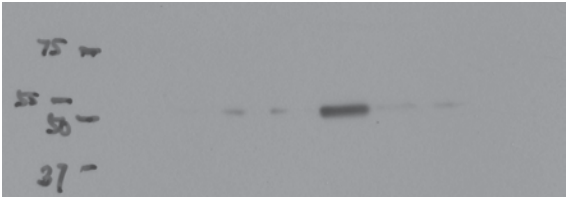
p85 alpha

p50 alpha

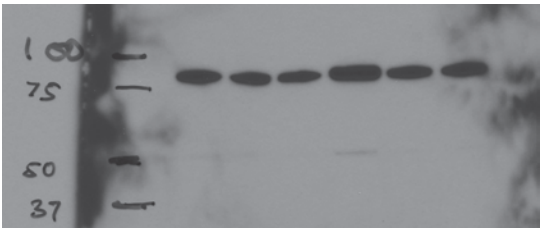


β-Actin



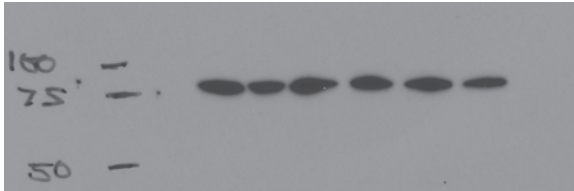


PIK3R3

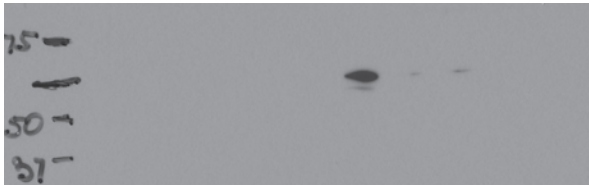


p85 alpha

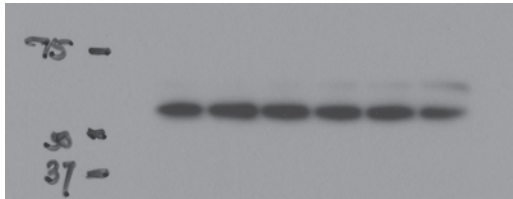
p50 alpha



p85 beta



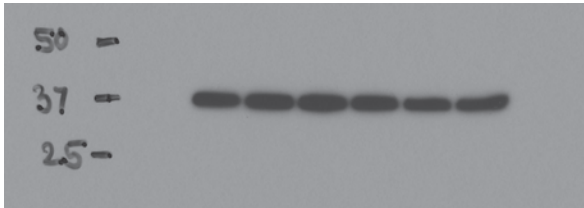
p-AKT (T308)



Total AKT



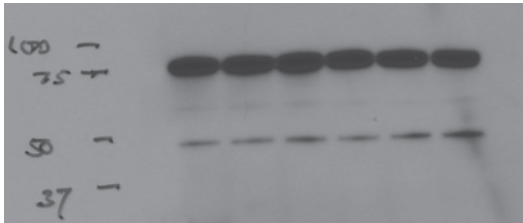
pS6K1 (T389)



GAPDH

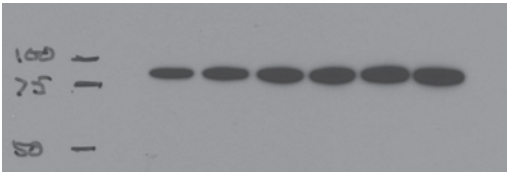


PIK3R3



p85 alpha

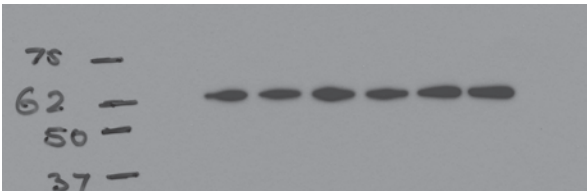
p50 alpha



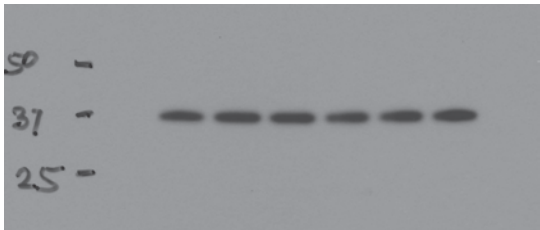
p85 beta



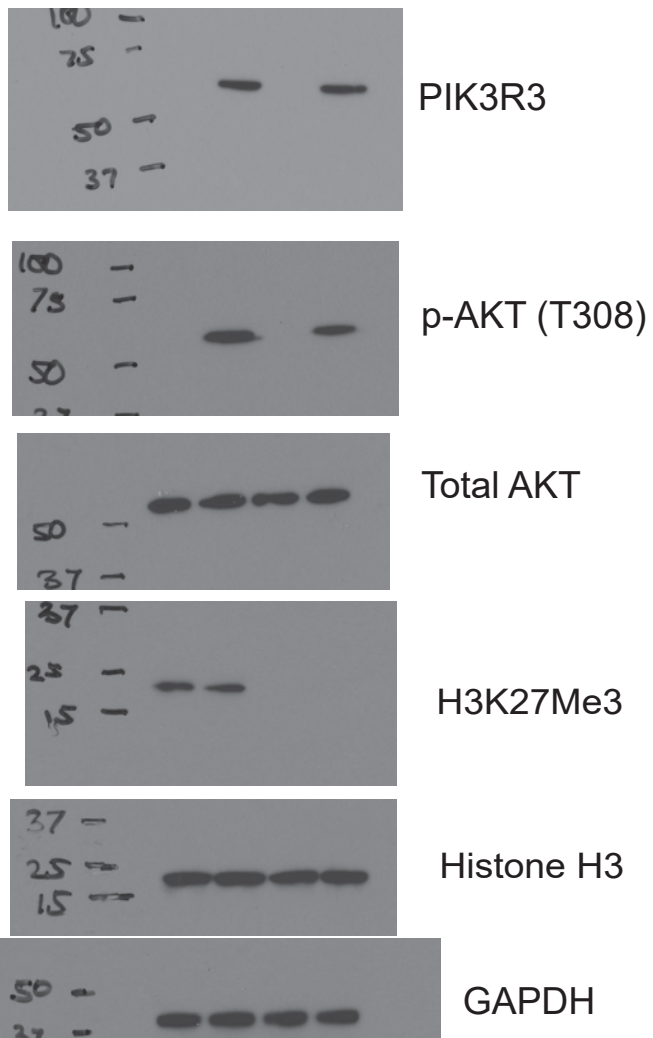
p-AKT (T308)



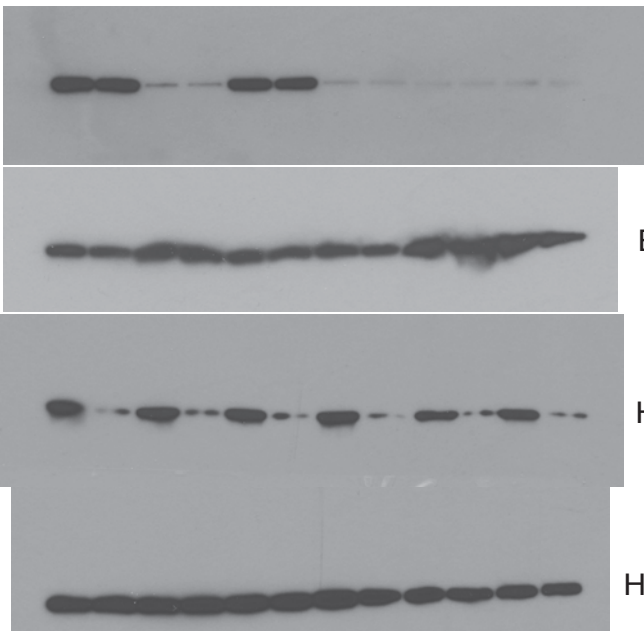
Total AKT



GAPDH



CPI-1205



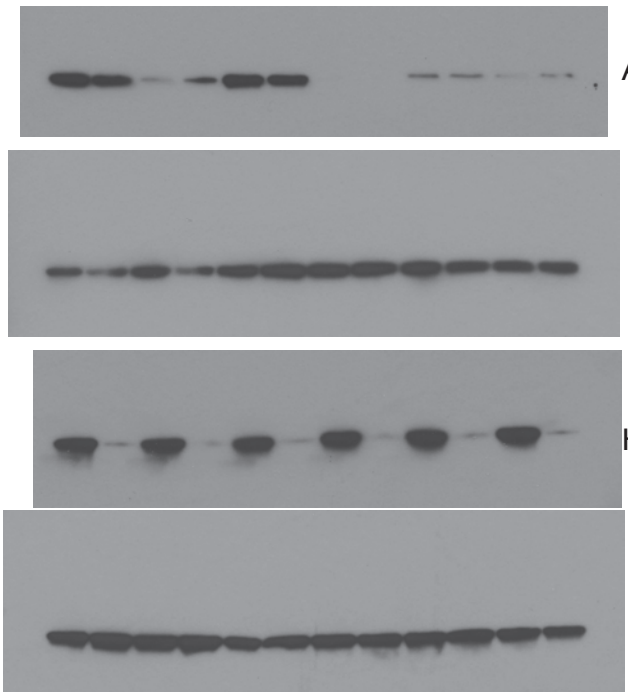
ARID1A

EZH2

H3K27Me3

Histone

EPZ-6428



ARID1A

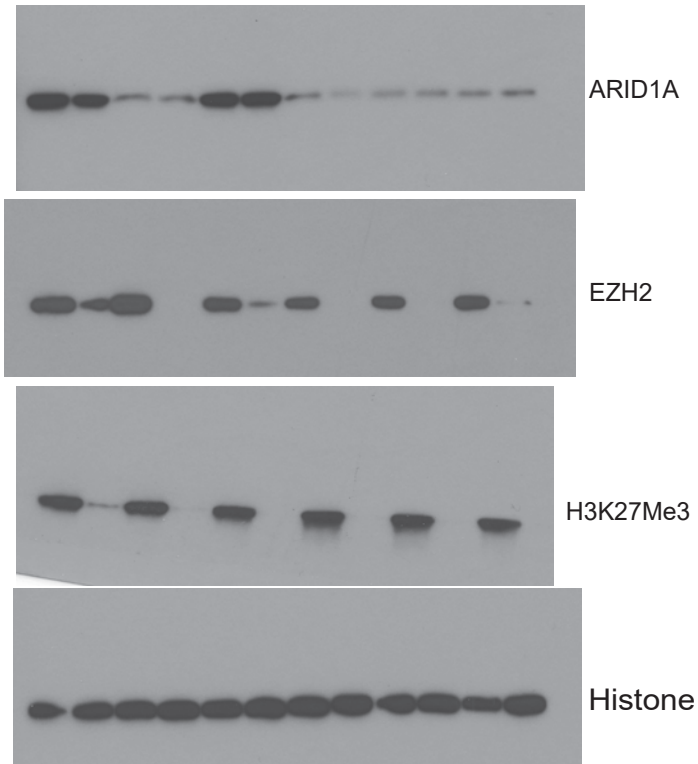
EZH2

H3K27Me3

Histone

Uncut blot Figure S4D

MAK-683



ARID1A

EZH2

H3K27Me3

Histone