SUPPLEMENTARY INFORMATION

NG-LE54374R1

Acute BAF perturbation causes immediate changes in chromatin accessibility

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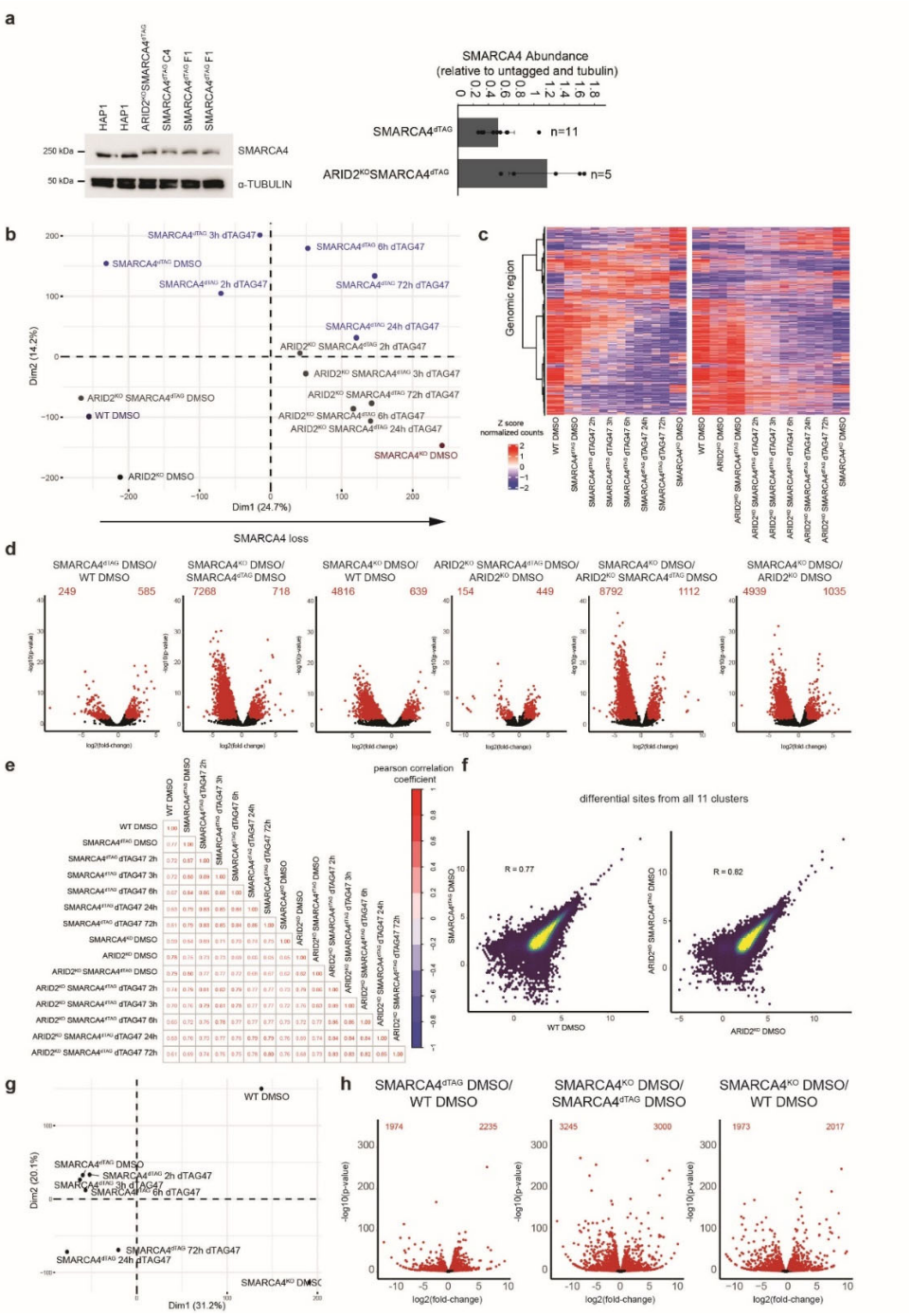
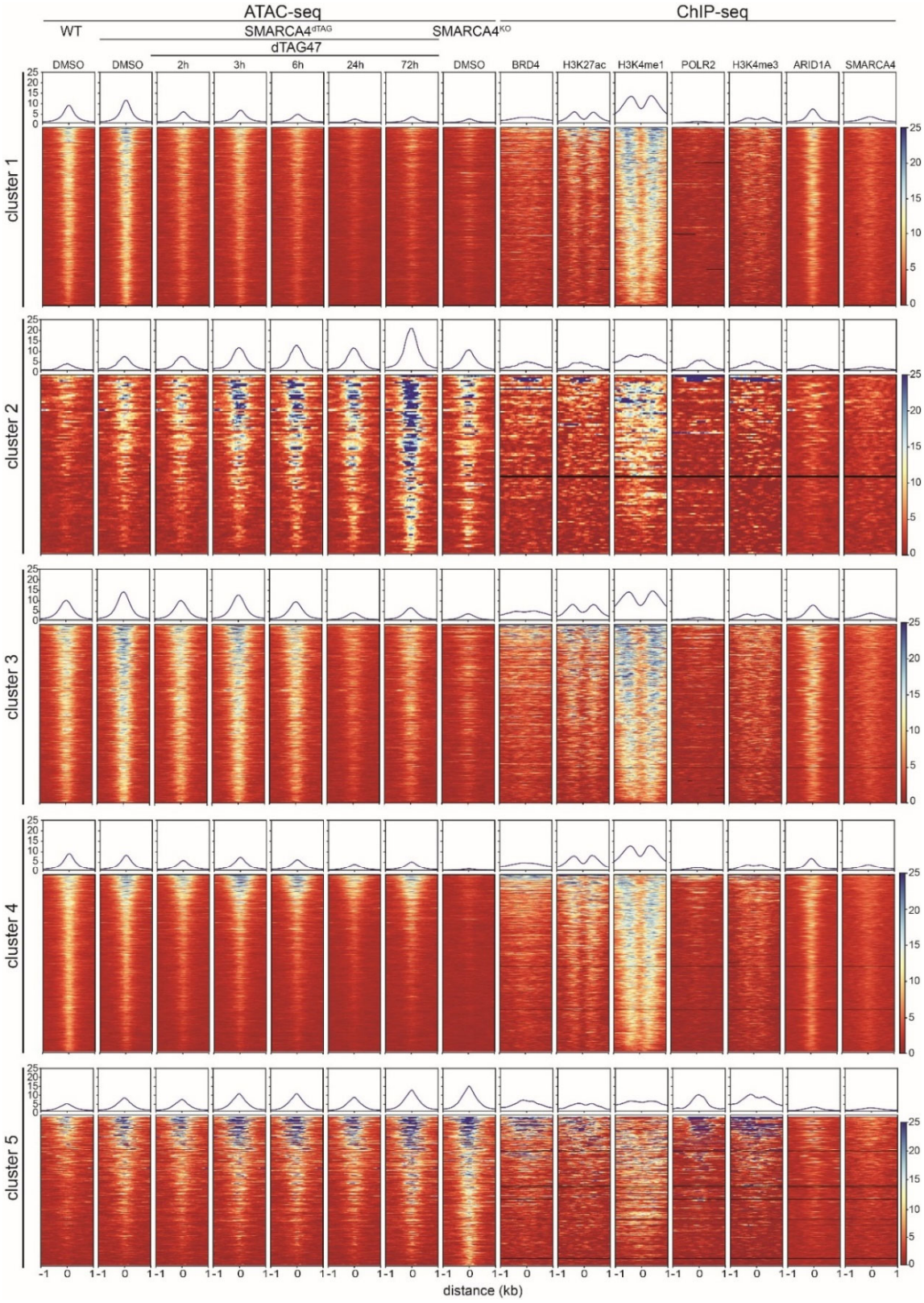


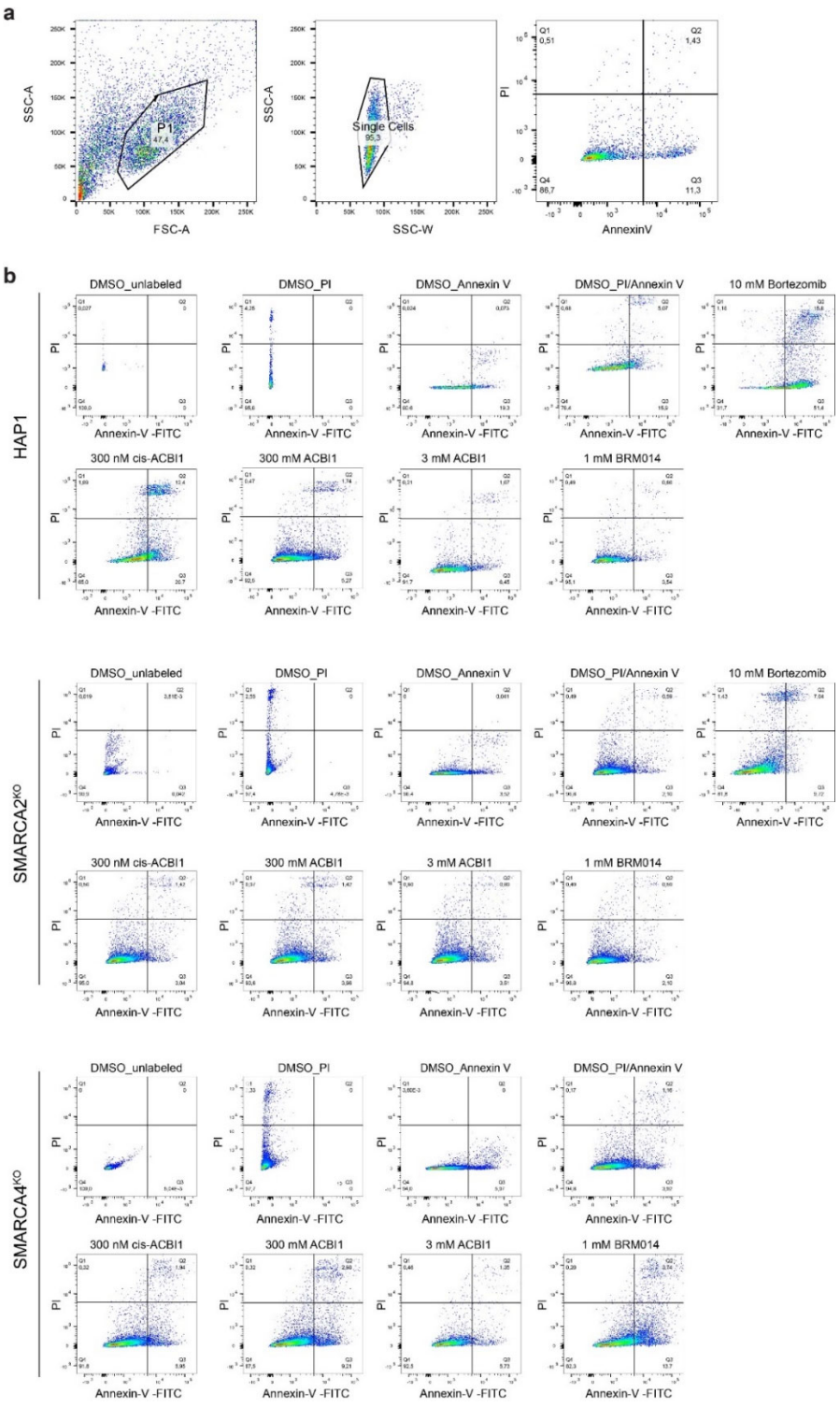
Supplementary Figure 1: Knock-in of the dTAG is not causative for the observed changes after SMARCA4 depletion.

(a) Western Blot analysis for SMARCA4 expression levels after dTAG insertion (cropped images) with quantification (mean±SD). **(b)** Principal component analysis of ATAC-seq data. **(c)** Heatmap of ATAC-seq data displaying quantile normalized counts of all differential regions. **(d)** ATAC-seq volcano plots of DEseq2 analyses. Significant changes (Padj < 0.01 and abs(log2 fold-change) >1) are colored in red. Two-sided Wald test was performed, False discovery rate (FDR) correction as implemented in DESeq2. **(e)** Correlation analyses of quantile normalized ATAC-seq counts. Pearson correlation coefficient is shown. **(f)** ATAC-seq scatter plots of quantile normalized counts displaying all differential sites of all 11 clusters. **(g)** Principal component analysis of RNA-seq data. **(h)** Volcano plots of DEseq2 analyses from RNA-seq data. Significant changes (Padj < 0.01 and abs(log2 fold-change) >1) are colored in red. Two-sided Wald test was performed, False discovery rate (FDR) correction as implemented in DESeq2.



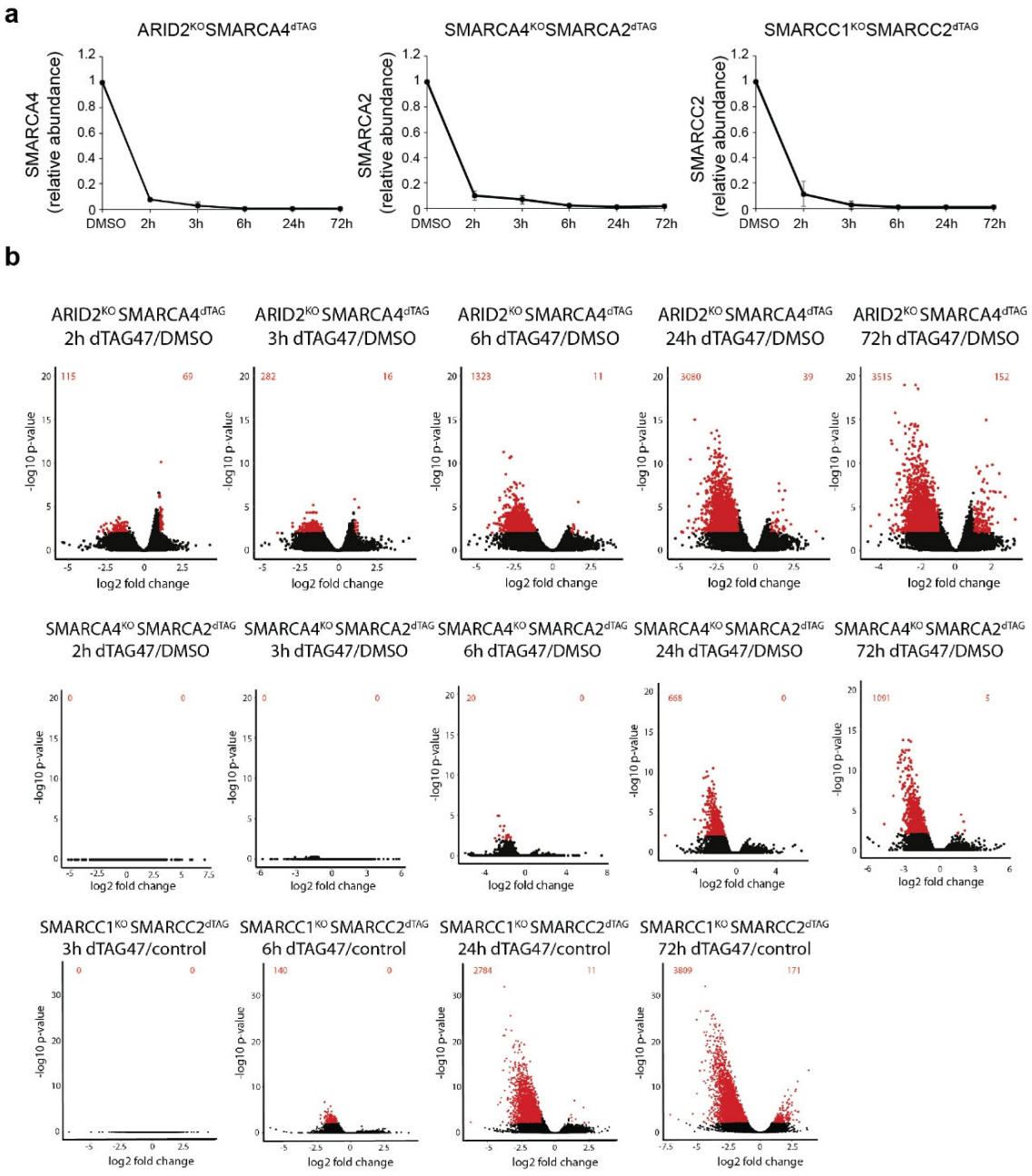
Supplementary Figure 2: Chromatin features of the 5 different clusters.

Chromatin accessibility signal (measured by ATAC-seq) and enrichment of different factors or histone modifications (measured by ChIP-seq) are displayed for differential regions falling into cluster 1-5.



Supplementary Figure 3: SMARCA2/SMARCA4 dual inhibition or degradation do not show cytotoxic effect on HAP1 wildtype, SMARCA2^{KO} and SMARCA4^{KO} cells.

(a) Gating strategy. **(b)** FITC Annexin-V and Propidium Iodide (PI) cell death assay of HAP1 wildtype and SMARCA2^{KO} and SMARCA4^{KO} cells 72h after treatment with ACBI1 and BRM014. The percentage of apoptotic cells stained with Annexin V/PI was measured by flow cytometry. Late apoptotic cell counts in the upper right quadrant (Q2) and lower right quadrant (Q3) for different treatment groups in control HAP1 wildtype (top), SMARCA2^{KO} (middle) and SMARCA4^{KO} (bottom) cells.



Supplementary Figure 4: Chromatin accessibility changes upon targeting of intra-complex synthetic lethalities

(a) Western blot quantitation of protein levels in different dTAG cell lines shows time-dependent degradation of tagged subunits following dTAG47 addition (n=2, mean and standard deviation). **(b)** Volcano plots displaying the chromatin accessibility changes after depletion of synthetic lethal subunits. Significant changes (Padj < 0.01 and abs(log2 fold-change) >1) are colored in red. Two-sided Wald test was performed, False discovery rate (FDR) correction as implemented in DESeq2.

Supplementary Methods

Western blot

Cell pellets were lysed rotating at 4°C for 1h in RIPA buffer (50 mM Tris pH 8, 1% NP-40, 0.5% Sodium deoxycholate, 0.1% SDS, 150 mM NaCl, 5 mM EDTA, 1% Glycerol, 2.5 mM $MgCl_2$, 2 mM Sodiumorthovanadate) containing 1x cOmplete™, EDTA-free Protease Inhibitor Cocktail (Sigma, 4693132001). Protein content of the supernatant after centrifuging for 10 min, 4°C, 13,000 rpm was measured using Bradford assay (AppliChem, A6932). 4x SDS Protein Sample Buffer (0.2 M Tris pH 6.8, 40% glycerol, 4% SDS, Bromphenol Blue, 0.04 % β-Mercaptoethanol) was added to the samples and they were boiled for 10 minutes at 95°C. Proteins were separated by using a 7% SDS-PAGE gel for large sized proteins and 12% for smaller sized proteins. After gel electrophoresis, the proteins were transferred to a 0.45 µm nitrocellulose membrane (Amersham Protran, GE Healthcare) at 200 mA for 1.5 h. The membranes were blocked in 5% milk (Bio-Rad 1706404) in TBS with 0.1% Tween-20 (TBS-Tween, Sigma P1379) for 1h. The membranes were incubated overnight at 4°C with primary antibodies (Supplementary Table 1). After four washing steps with TBS-Tween, the membranes were incubated in secondary antibodies for 1h at room temperature (RT) followed by four washing steps with TBS-Tween. Membranes were then developed using Clarity Western ECL Substrate (Bio-Rad, #170-5060) and imaged on a Bio-Rad ChemiDoc™ MP with Image LabTM Touch Software Version 2.3.0.07. Full scans of all Western Blots are provided in the last section of this document.

Fractionation

PBS-washed cell pellets were resuspended in 2.5x volume Buffer N (300 mM sucrose, 10 mM HEPES pH 7.9, 10 mM KCl, 0.1 mM EDTA, 0.1 mM EGTA, 0.1 mM DTT, 0.75 mM spermidine, 0.15 mM spermine, 0.1% NP-40, 50 mM NaF, 1 mM Na_3VO_4, 1 mM PMSF, 1x cOmpleteTM, EDTA-free protease inhibitors), vortexed and incubated on ice for 5 min. The supernatant after centrifugation at 500 g and 4°C for 5 minutes was collected as cytoplasmic fraction. Nuclear pellets were resuspended in Buffer N and pelleted by centrifugation two more times. The pellets were resuspended in Buffer C420 (20 mM HEPES pH 7.9, 420 mM NaCl, 25 % glycerol, 1 mM EDTA, 1 mM EGTA, 0.1 mM DTT, 50 mM NaF, 1 mM Na_3VO_4, 1 mM PMSF, 1x cOmpleteTM, EDTA-free protease inhibitors) in half of the volume that was used of Buffer N and shaken in a Thermomixer at 4°C, 1,400 rpm for 30 min. After a centrifugation at maximum speed and 4°C for 30 minutes, the supernatant was collected as nuclear fraction. The pellet was resuspended in 0.5 ml of Buffer SB (10 mM HEPES pH7.5, 10 mM KCl, 1.5 mM $MgCl_2$, 0.34 M Sucrose, 0.1% Triton X-100, 1x cOmpleteTM, EDTA-free protease inhibitors) and added to 2.5 ml of Buffer SC (10 mM HEPES

pH7.5, 10 mM KCl, 1.5 mM $MgCl_2$, 2.1 M Sucrose, 1x cOmpleteTM, EDTA-free protease inhibitors). The samples were put in an ultracentrifuge at 44,000 rpm and 4°C overnight. The supernatant of this centrifugation step was discarded, the pellet was resuspended in 0.5 ml of Buffer SB and centrifuged at 20,000 g and 4°C for 45 minutes. This was followed by two washing steps with 0.5 ml of washing buffer (10 mM HEPES pH7.5, 1x cOmpleteTM, EDTA-free protease inhibitors) and 15 minutes centrifugation at 20,000 g and 4°C. The supernatant was discarded and the pellet was resuspended in 250 µl of Benzonase Digest Buffer (15 mM HEPS pH7.5, 1 mM EDTA, 1 mM EGTA, 1x cOmpleteTM, EDTA-free protease inhibitors, 0.1% NP-40). They were sonicated in a Covaris tube using the Covaris S2x sonicator and transferred to new reaction tubes. 1 µl 25 U/ml Benzonase (Novagen 71205) and 2.5 µl 10 µg/ml RNAase (Sigma R4875) were added and the samples incubated for 40 minutes at 4°C.

Nuclear extracts for mass spectrometry analyses were supplemented with 0.1% of NP-40 (Sigma I3021), 20 U/ml of Benzonase (Novagen 71205) and 50 ng/ml of RNase A (Sigma R4875). The samples were rotated for 1h at 4°C. 20 µl of 10% SDS (Sigma 75746) (2% SDS final concentration) were added to 50 µg of the nuclear extracts and the samples filled up to 100 µl with HEPES II buffer.

LC-MS/MS analysis

Liquid chromatography mass spectrometry was performed on a Q Exactive™ Hybrid Quadrupole-Orbitrap (ThermoFisher Scientific, Waltham, MA) coupled to a Dionex U3000 RSLC nano system (Thermo Fisher Scientific, San Jose, CA) via nanoflex source interface. Tryptic peptides were loaded onto a trap column (Acclaim™ PepMap™ 100 C18, 3 µm, 5 × 0.3 mm, Fisher Scientific, San Jose, CA) at a flow rate of 10 µl/min using 2% acetonitrile in 0.1% TFA as loading buffer. After loading, the trap column was switched in-line with a 40 cm, 75 µm inner diameter analytical column (packed in-house with ReproSil-Pur 120 C18-AQ, 3 µm, Dr. Maisch, Ammerbuch-Entringen, Germany). Mobile-phase A consisted of 0.4% formic acid in water and mobile-phase B of 0.4% formic acid in a mix of 90% acetonitrile and 10% water. The flow rate was set to 230 nl/min and a 90 min gradient used (4 to 24% solvent B within 82 min, 24 to 36% solvent B within 8 min and, 36 to 100% solvent B within 1 min, 100% solvent B for 6 min before re-equilibrating at 4% solvent B for 18 min).

For the MS/MS experiment, the Q Exactive™ MS was operated in a top 10 data-dependent acquisition mode with a MS^1 scan range of 375 to 1,650 m/z at a resolution of 70,000 (at 200 m/z). Automatic gain control (AGC) was set to a target of 1×10^6 and a maximum injection time of 55 ms. MS^2-scans were acquired at a resolution of 15,000 (at 200 m/z) with AGC settings of 1×10^5 and a

maximum injection time of 110 ms. Precursor isolation width was set to 1.6 Da and the HCD normalized collision energy to 28%. The threshold for selecting precursor ions for MS^2 was set to 3.6×10^4. For enhanced detection of the 29 core BAF complex members, an inclusion list containing 755 peptide m/z values was generated using Skyline (https://skyline.ms/project/home/software/Skyline) for preferential precursor selection using a 10 ppm m/z inclusion tolerance. Dynamic exclusion for selected ions was 90 sec. A single lock mass at m/z 445.120024 was employed[1]. All samples were analysed in duplicates, back-to-back replicates. XCalibur version 4.1.31.9 and Tune 2.9.2926 were used to operate the instrument.

Mass spectrometry data analysis

Acquired raw data files were processed using the Proteome Discoverer 2.4.0.305 platform, utilizing the database search engine Sequest HT. Percolator V3.0 was used to remove false positives with a false discovery rate (FDR) of 1% on peptide and protein level under strict conditions. Searches were performed with full tryptic digestion against the human SwissProt database v2017.06 (20,456 sequences and appended known contaminants) with up to two miscleavage sites. Oxidation (+15.9949 Da) of methionine and phosphorylation of serine, threonine or tyrosine (+79.966331 Da) was set as variable modification, whilst carbamidomethylation (+57.0214 Da) of cysteine residues was set as fixed modifications. All spectra having peptide matches worse than high were send into a second search event using acetylation (+42.010565 Da), methylation (+14.01565 Da), dimetylation (+28.03130 Da) and trimethylation (+42.046950 Da) as well as ubiquitination (+114.042927 Da) as variable modification on lysine. Data was searched with mass tolerances of ±10 ppm and 0.02 Da on the precursor and fragment ions, respectively. Results were filtered to include peptide spectrum matches (PSMs) with Sequest HT cross-correlation factor (Xcorr) scores of ≥1 and high peptide confidence. For calculation of protein areas Minora Feature Detector node and Precursor Ions Quantifier node, both integrated in Thermo Proteome Discoverer were used. Automated chromatographic alignment and feature linking mapping were enabled with total peptide amount used for normalization between individual runs. Normalized protein abundances were used for Principal Component Analysis[2]. For each biological replicate, two technical replicates were summated, followed by $\log_2$ transformation and fold change calculation. Only proteins that were present in both the control and treatment conditions were included in fold change calculation. p values were calculated by Welch Two Sample t-test. Because dTAG experiment affects only a small subset of proteins (BAF complex proteins numbering less than 20 per condition), multiple testing was not considered for significance estimate.

FACS

HAP1 wildtype, SMARCA2^{KO} and SMARCA4^{KO} cells were seeded in 24-well plates (CytoOne, Cat no. CC7682-7524) in a density of 50x10^3 cells per well. Cells were treated with DMSO (Merck Cat. No.41640-1L-M, Lot STBG9935), 300 nM *cis*-ACBI1, 300 nM ACBI1, 3 μM ACBI1 or 1 μM BRM014 and incubated for 72h. For positive control, cell apoptosis was induced by 24h treatment with 10 μM Bortezomib.

On the day of the experiment, both supernatant and cells were collected and pelleted for 5min. The cell pellet was diluted in 500μl of the buffer provided in the Annexin V-FITC Apoptosis Detection kit (Abcam, Cat no. ab 14085, Lot no. GR230157-3). 50 μg/ml of Propidium Iodide (PI) and 5 μl of Annexin-V as was added to each tube as suggested by the manufacturer protocol. The control tubes contained single staining or no staining. The mixture was incubated for 5 min in the dark at room temperature. Samples were analyzed by BD LSRFortessa™ Flow Cytometer using FITC signal detector for Annexin-V and phycoerythrin emission signal detector for PI. The data was analyzed using FlowJo software (BD, version 10).

Processing of ATAC-seq reads

Read trimming was performed using Skewer[3]. Read mapping was performed to GRCh38/hg38 using Bowtie2[4] and the "-very sensitive" parameter. Only unique reads with a mapping quality >30 and alignment to the nuclear genome were included in the downstream analyses. Fastqc was used for quality control. Peak calling was performed using MACS2[5] with the "-nomodel" and "extsize 147" parameters. Peaks overlapping blacklisted features defined in the ENCODE project[6] were discarded from the analyses.

Processing of ChIP-seq reads

Read trimming was performed using Skewer[3]. Read mapping was performed to GRCh38/hg38 using Bowtie2[4] and the "-very sensitive" parameter. Only unique reads with a mapping quality >30 and alignment to the nuclear genome were included in the downstream analyses.

Processing of transcriptome analysis

Raw data acquisition (HiSeq Control Software, HCS, HD 3.4.0.38) and base calling (Real-Time Analysis Software, RTA, 2.7.7) was performed on HiSeq 3000/4000 instruments (Illumina, San Diego, CA, USA), while the subsequent raw data processing involved two custom programs based on Picard tools (2.19.2) (https://github.com/DanieleBarreca/picard/, https://broadinstitute.github.io/picard/). In a first step, base calls were converted into lane-specific, multiplexed, unaligned BAM files suitable for long-term archival

(IlluminaBasecallsToMultiplexSam, 2.19.2-CeMM). In a second step, archive BAM files were demultiplexed into sample-specific, unaligned BAM files (IlluminaSamDemux, 2.19.2-CeMM).

NGS reads were mapped to the Genome Reference Consortium GRCh38 assembly via “Spliced Transcripts Alignment to a Reference” (STAR, 2.7.5a)[7] utilising the “basic” Ensembl transcript annotation from version e100 (April 2020) as reference transcriptome. Since the hg38 assembly flavour of the UCSC Genome Browser was preferred for downstream data processing with Bioconductor packages for entirely technical reasons, Ensembl transcript annotation had to be adjusted to UCSC Genome Browser sequence region names. STAR was run with options suggested by the ENCODE project. Aligned NGS reads overlapping Ensembl transcript features were counted with the Bioconductor (3.11) GenomicAlignments[8] (1.24.0) package via the summarizeOverlaps function in Union mode, taking into account that the Illumina TruSeq stranded mRNA protocol leads to sequencing of the second strand so that all reads needed inverting before counting. Transcript-level counts were aggregated to gene-level counts and the Bioconductor DESeq2 package[9] (1.28.1) was used to test for differential expression based on a model using the negative binomial distribution. Biologically meaningful results were extracted from the model, log2-fold values where shrunk with the CRAN ashr package (2.2-47)[10], while two-tailed p-values obtained from Wald testing were adjusted with the Benjamini-Hochberg procedure.

Processing of PRO-seq reads

Raw data were processed as published[11]. Briefly, reads were adapter-trimmed using cutadapt (version 1.9.1), converted to reverse complements using fastx reverse_complement (version 0.014), aligned to a concatenated hg38_dm6_rDNA-U13369 index using bowtie2 (version 2.2.9), and MAPQ20 filtered (samtools version 1.7). Reads aligning to dm6 chromosome scaffolds were counted to calculate global normalization factors alpha=1e6/dm6_count as described[11,12]. Signal originating from human hg38 scaffolds was now trimmed to only retain a single 3’ base corresponding to biotinylated nucleotides last incorporated by polymerase active centers during the run-on reaction (bedtools v2.26.0).

R packages

The following R packages were used for figure generation in custom scripts publicly available in our GitHub repository: corrplot, GetoptLong, colorspace, circlize, ComplexHeatmap, NbClust, ggplot2, data.table, SummarizedExperiment, DelayedArray, BiocParallel, matrixStats, Biobase, GenomicRanges, GenomeInfoDb, IRanges, S4Vectors, BiocGenerics, DOSE, org.Hs.eg.db, AnnotationDbi, enrichplot, stringr, viridis, GO.db, RColorBrewer, scales, reshape, ggrepel, simpleCache, ggsci, BSDA, lawstat, hexbin, plyr.

Supplementary Methods – References

1. Olsen, J.V. *et al.* Parts per million mass accuracy on an Orbitrap mass spectrometer via lock mass injection into a C-trap. *Mol Cell Proteomics* **4**, 2010-21 (2005).
2. Stacklies, W., Redestig, H., Scholz, M., Walther, D. & Selbig, J. pcaMethods--a bioconductor package providing PCA methods for incomplete data. *Bioinformatics* **23**, 1164-7 (2007).
3. Jiang, H., Lei, R., Ding, S.W. & Zhu, S. Skewer: a fast and accurate adapter trimmer for next-generation sequencing paired-end reads. *BMC Bioinformatics* **15**, 182 (2014).
4. Langmead, B. & Salzberg, S.L. Fast gapped-read alignment with Bowtie 2. *Nat Methods* **9**, 357-9 (2012).
5. Zhang, Y. *et al.* Model-based analysis of ChIP-Seq (MACS). *Genome Biol* **9**, R137 (2008).
6. Hoffman, M.M. *et al.* Integrative annotation of chromatin elements from ENCODE data. *Nucleic Acids Res* **41**, 827-41 (2013).
7. Dobin, A. *et al.* STAR: ultrafast universal RNA-seq aligner. *Bioinformatics* **29**, 15-21 (2013).
8. Lawrence, M. *et al.* Software for computing and annotating genomic ranges. *PLoS Comput Biol* **9**, e1003118 (2013).
9. Love, M.I., Huber, W. & Anders, S. Moderated estimation of fold change and dispersion for RNA-seq data with DESeq2. *Genome Biol* **15**, 550 (2014).
10. Stephens, M. False discovery rates: a new deal. *Biostatistics* **18**, 275-294 (2016).
11. Jaeger, M.G. *et al.* Selective Mediator dependence of cell-type-specifying transcription. *Nat Genet* **52**, 719-727 (2020).
12. Orlando, D.A. *et al.* Quantitative ChIP-Seq normalization reveals global modulation of the epigenome. *Cell Rep* **9**, 1163-70 (2014).

Full Scans of all Western blots

Fig. 1b/ ED2a

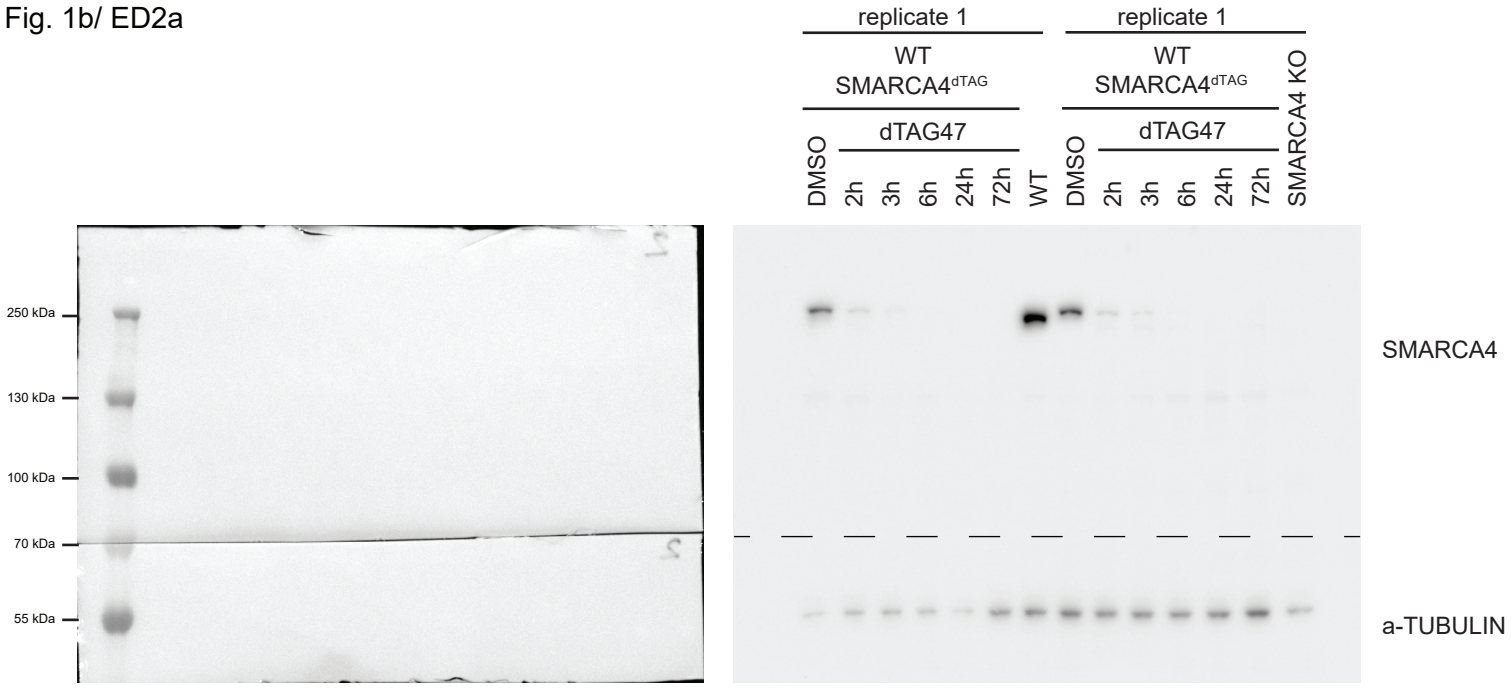
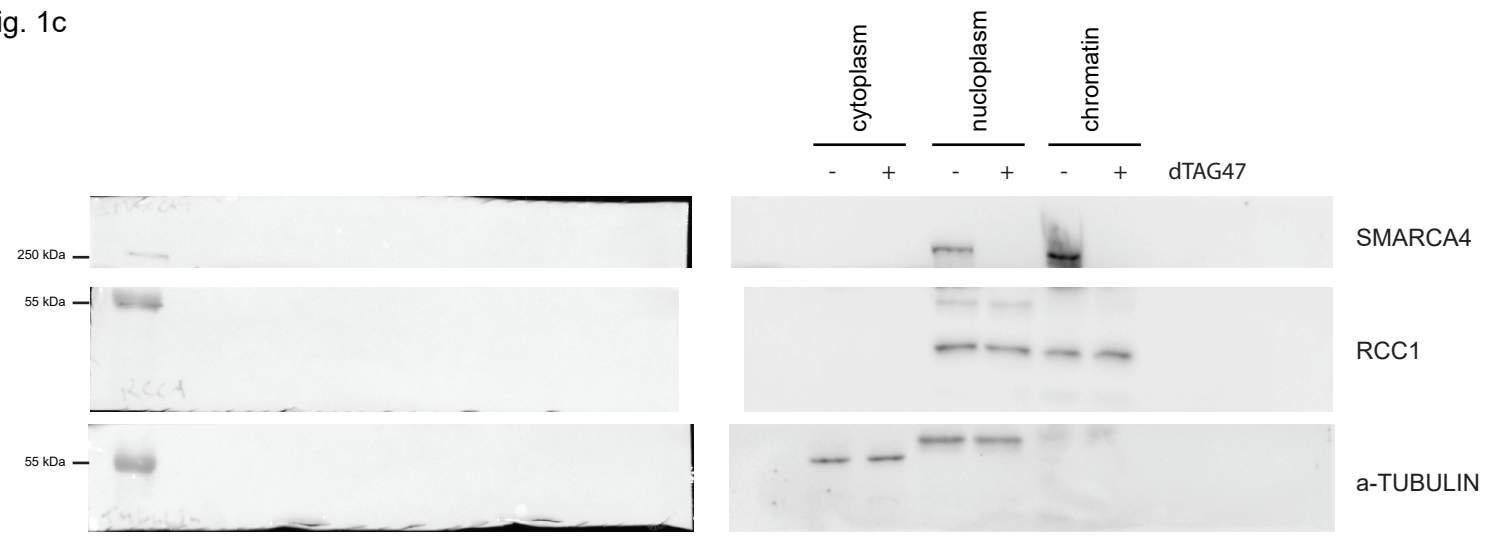


Fig. 1c



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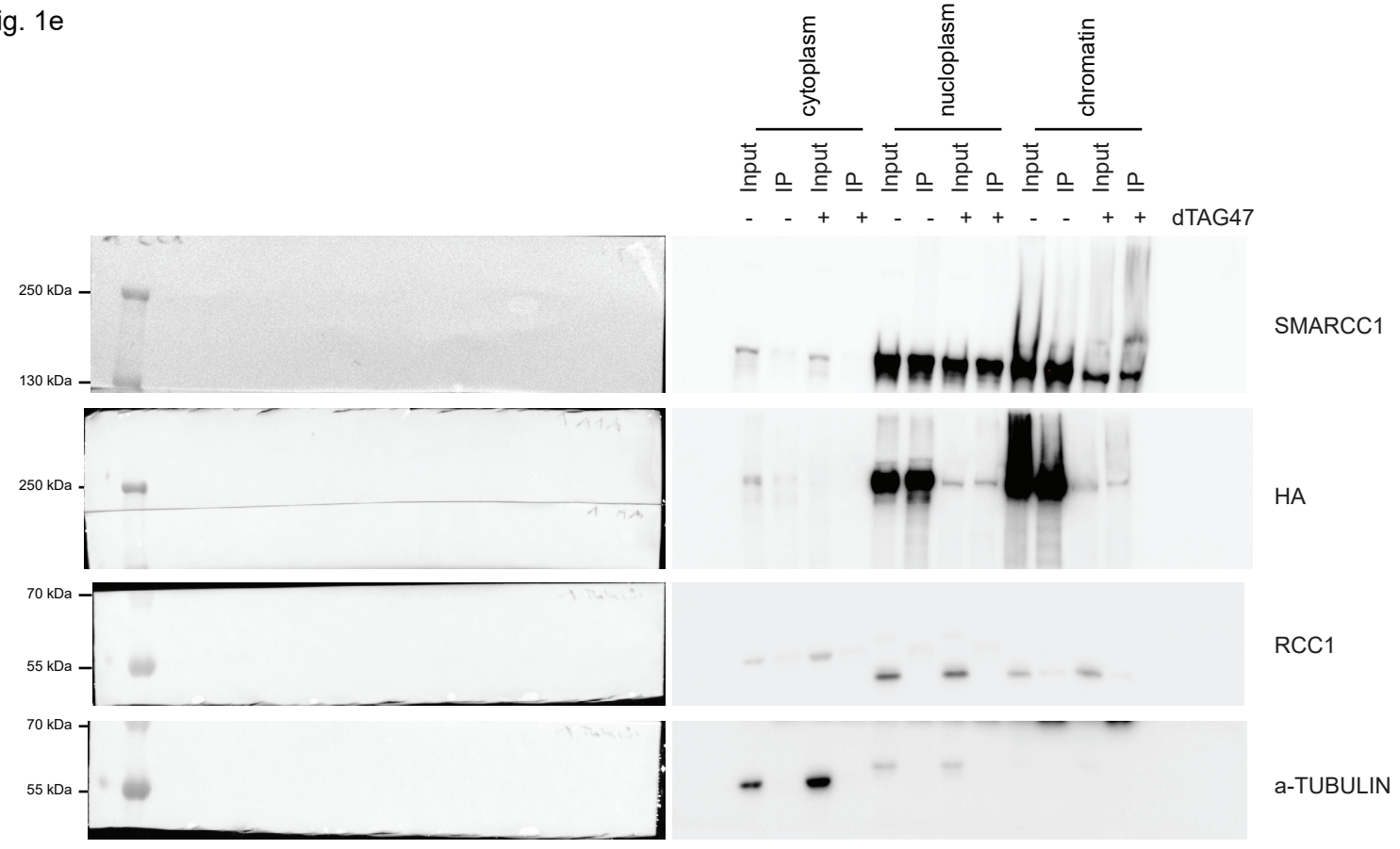
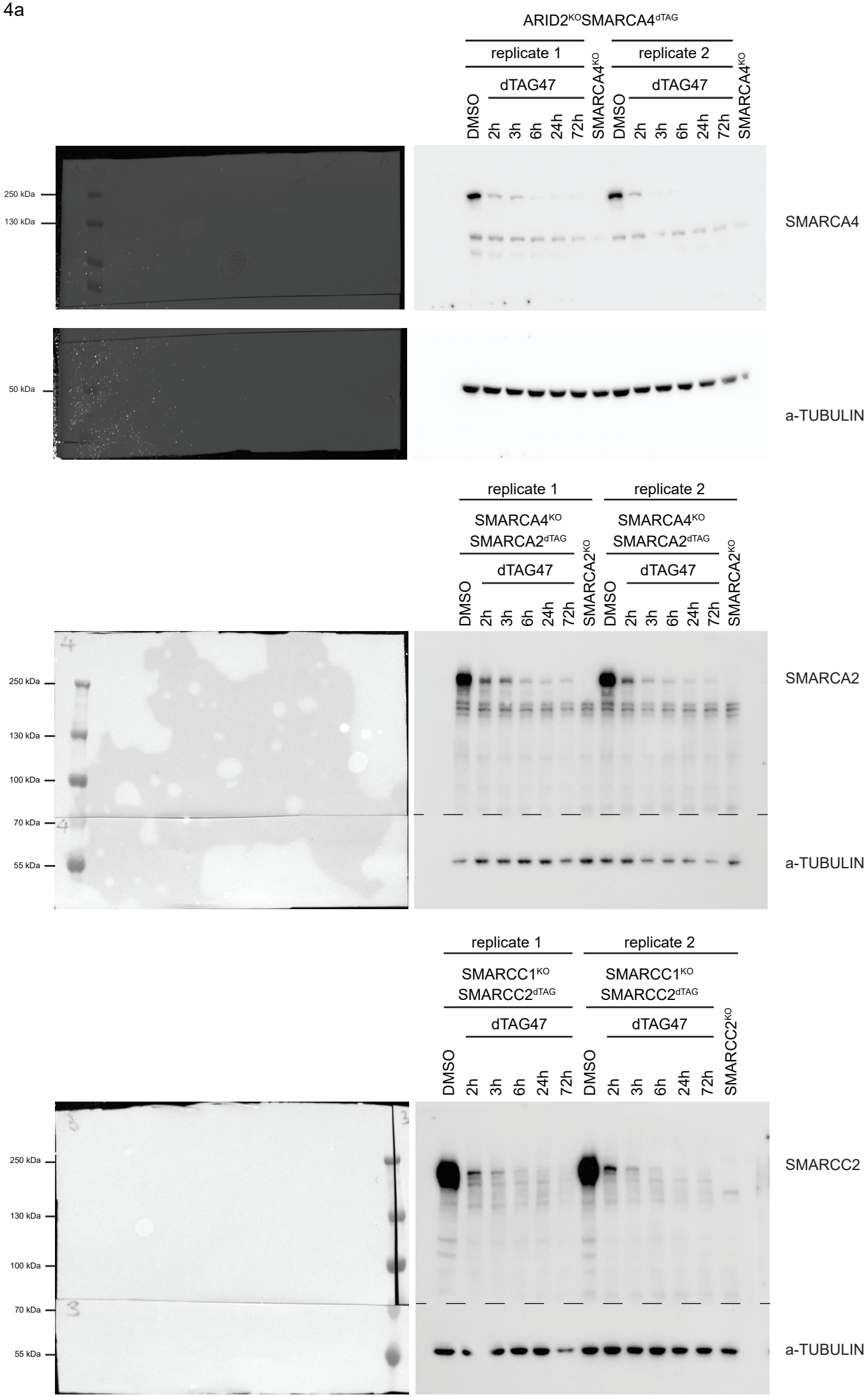


Fig. 4a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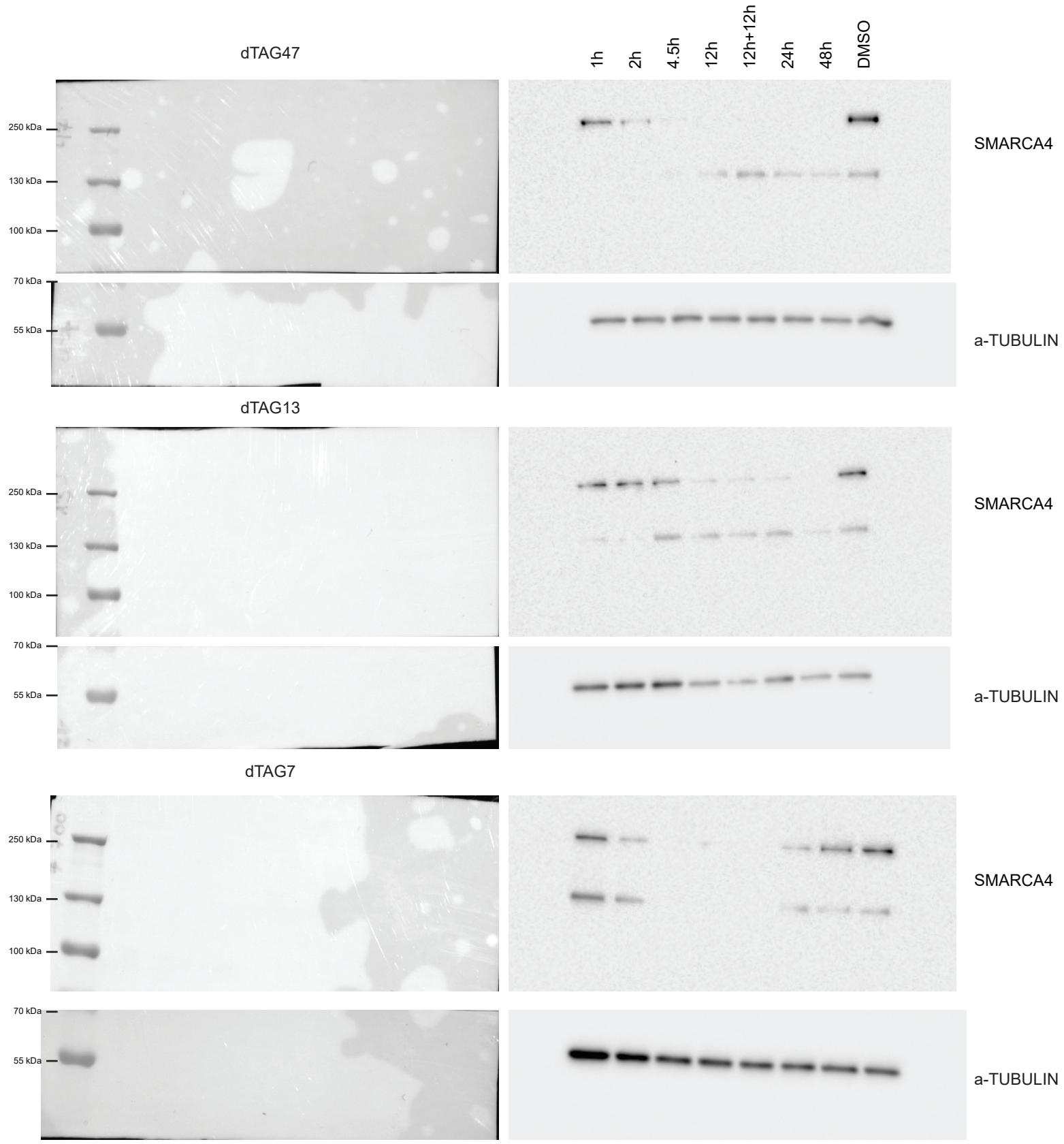
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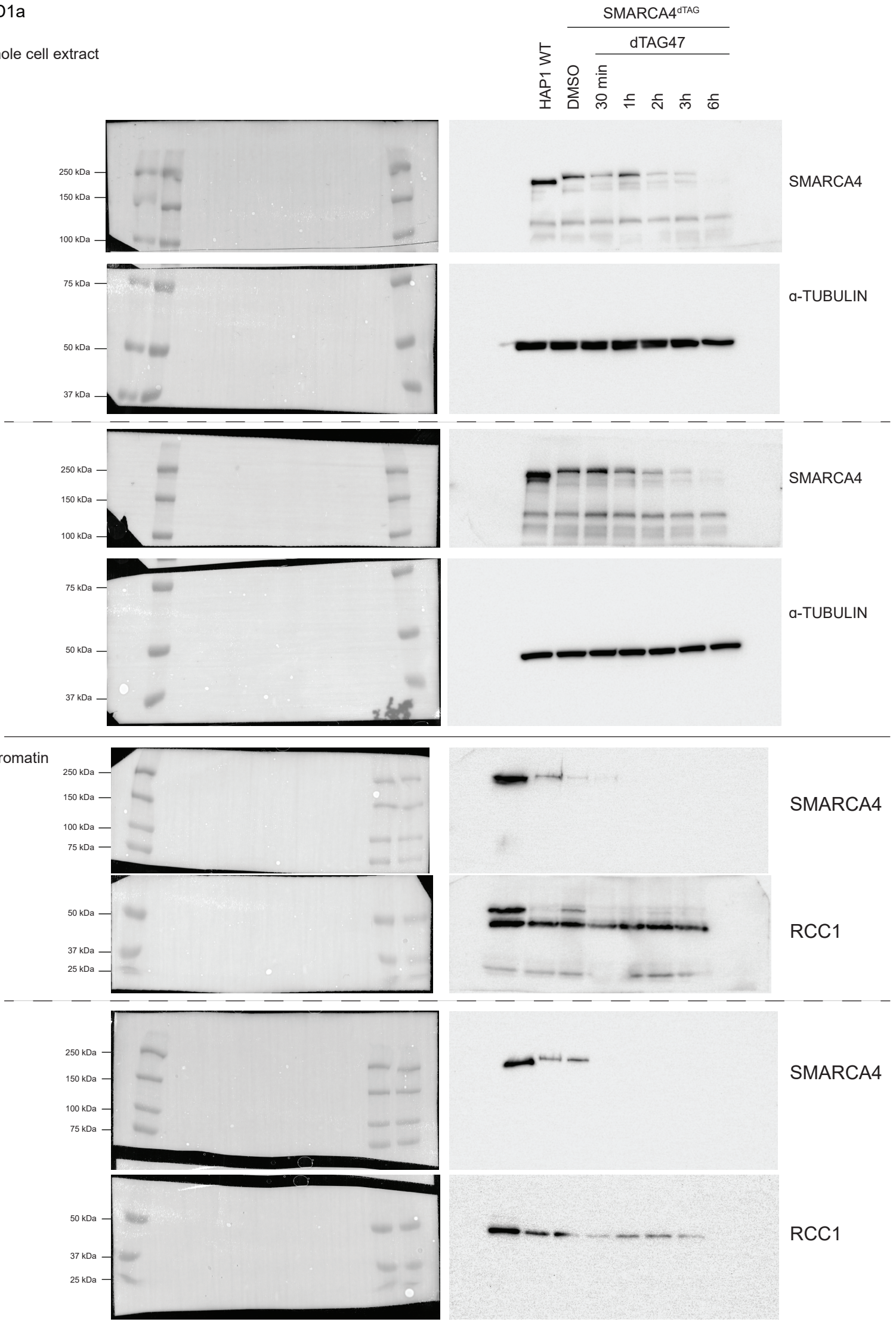
whole cell extract



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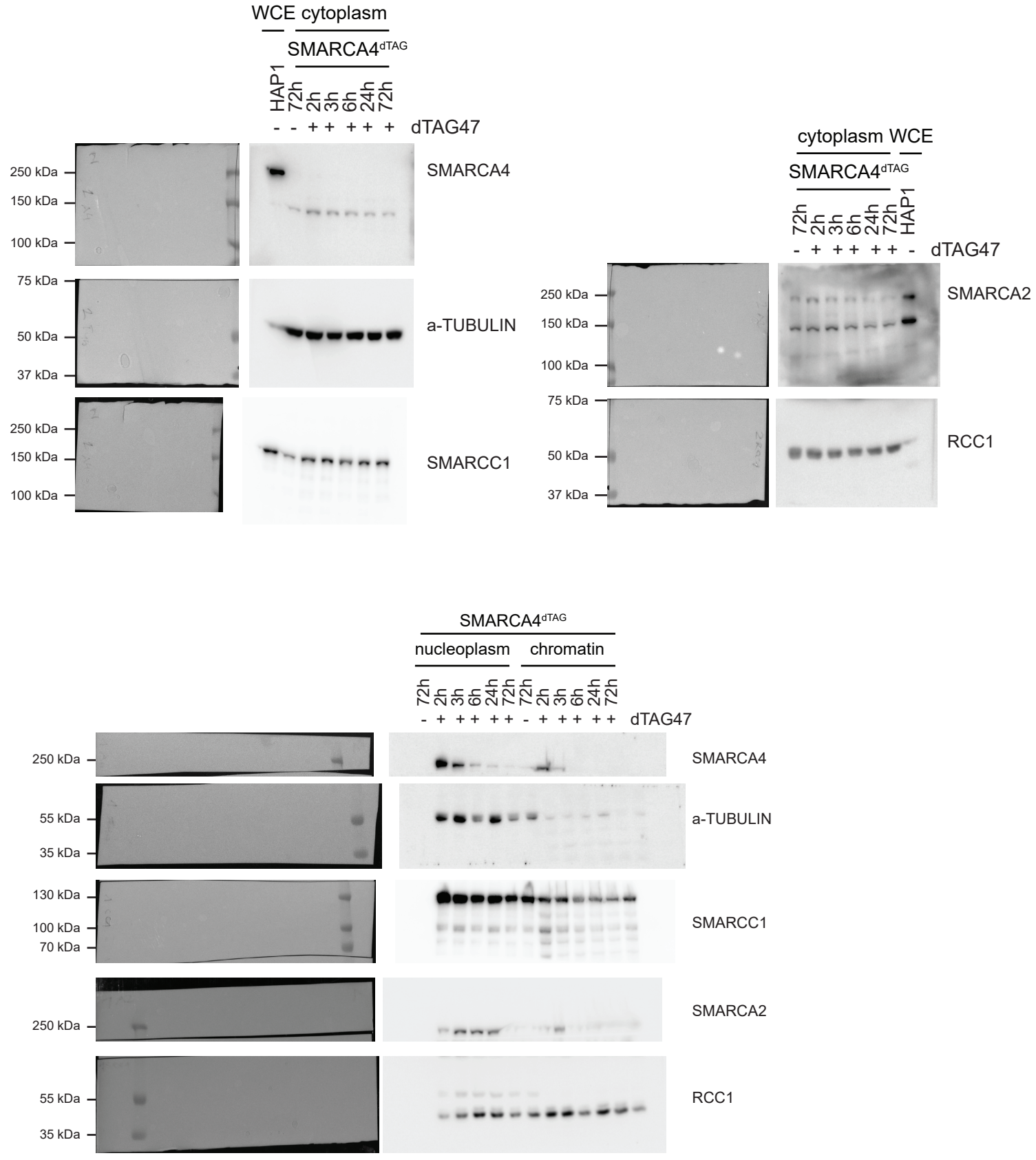
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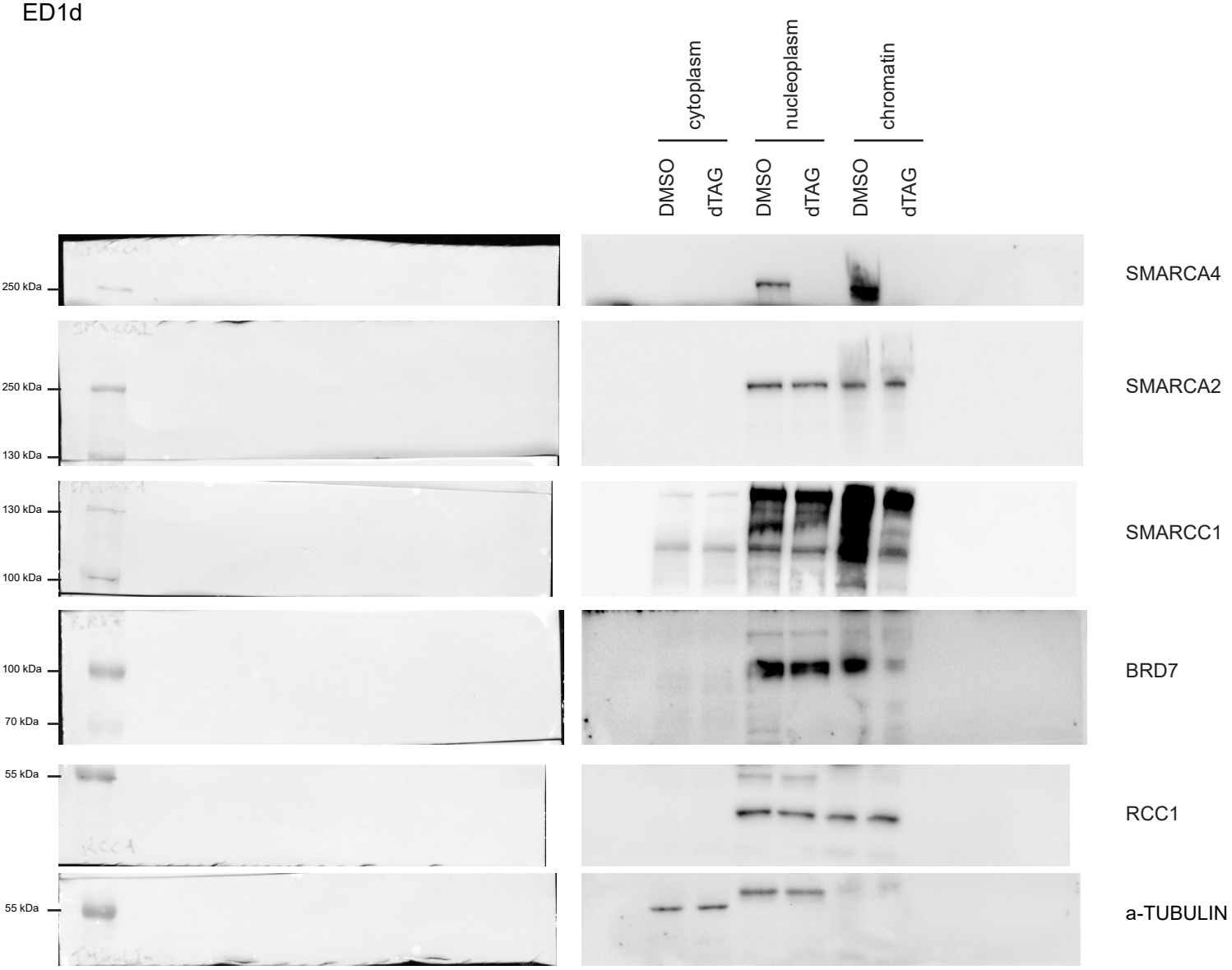
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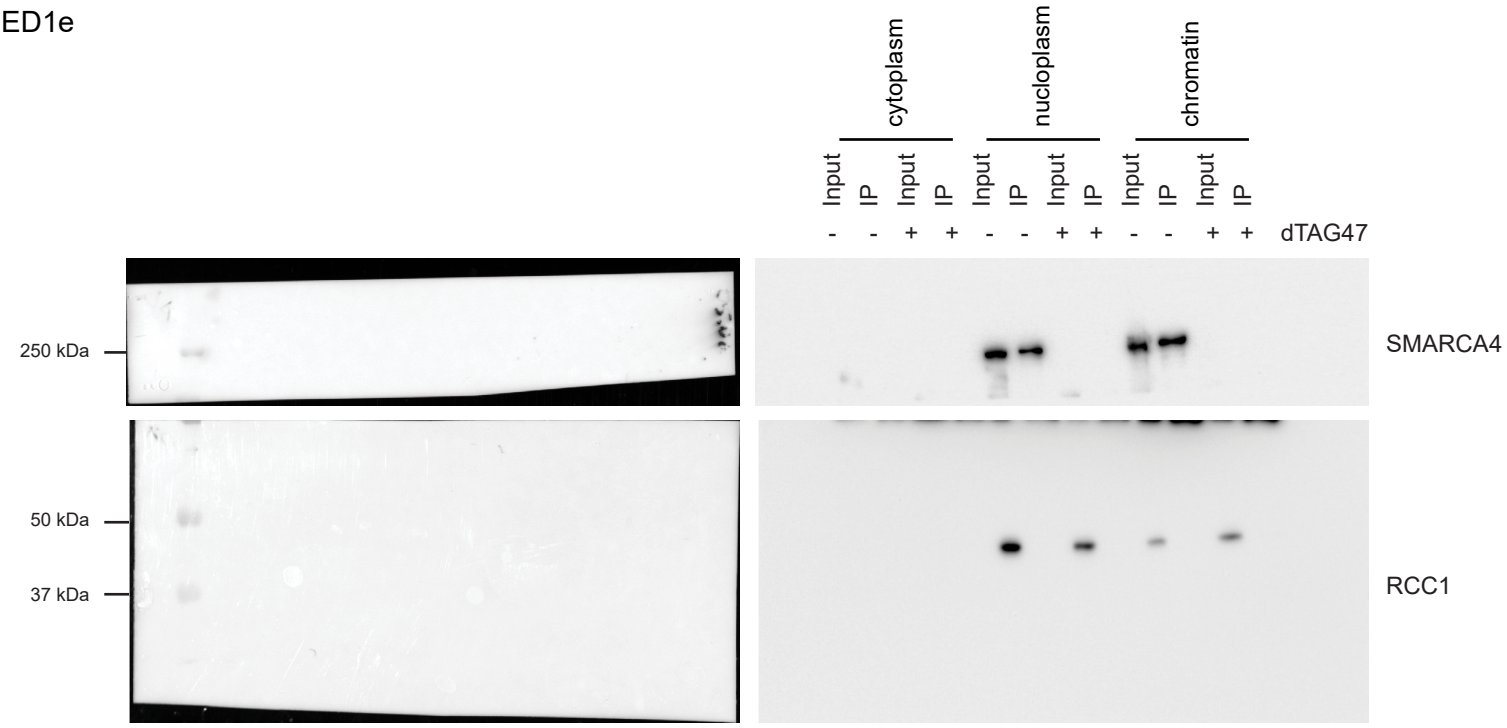
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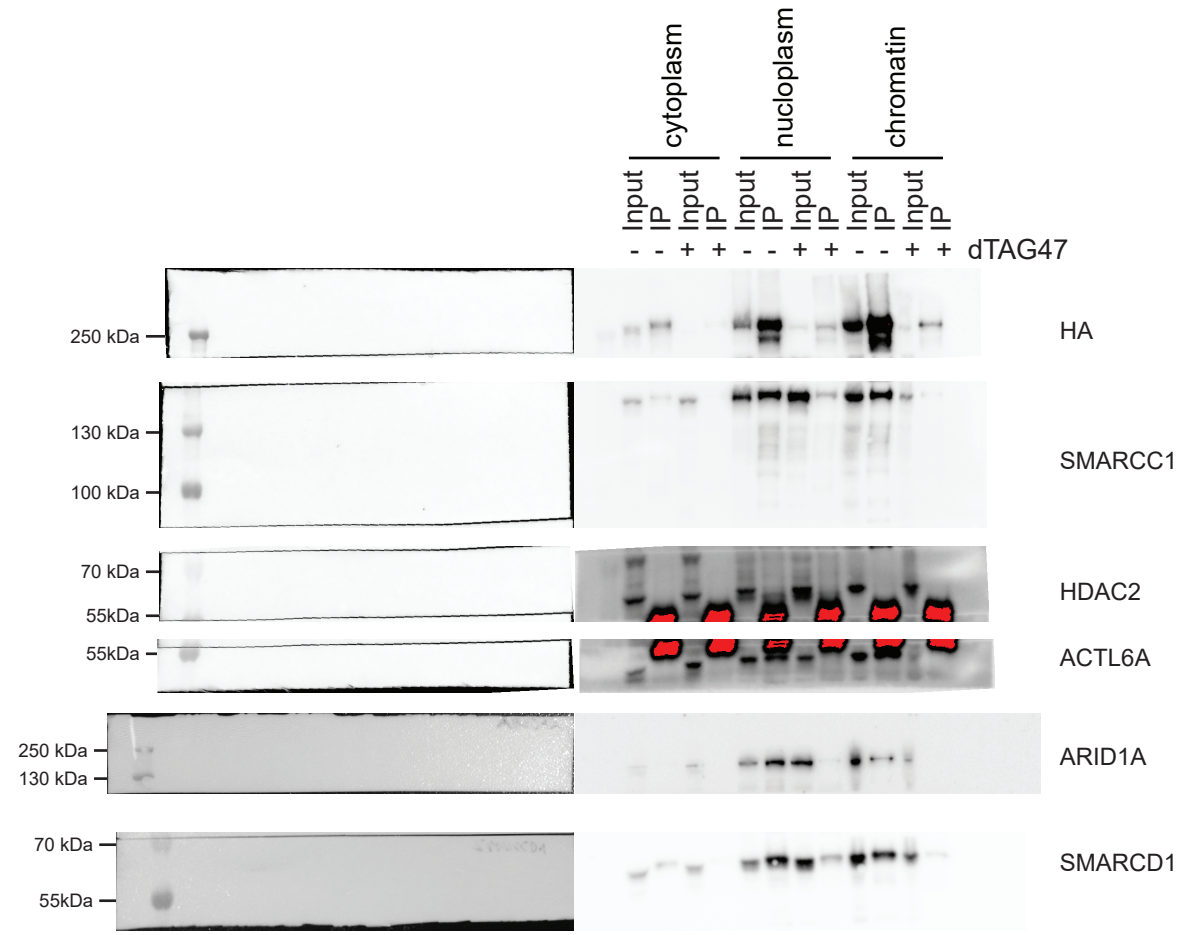
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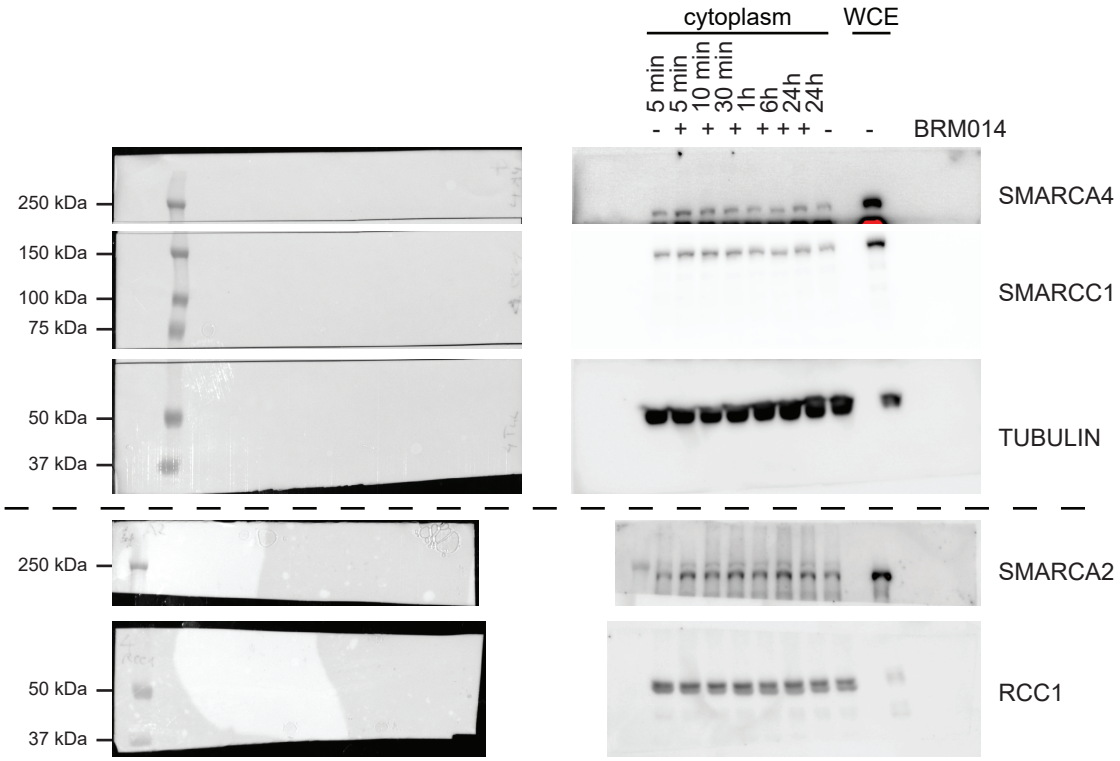
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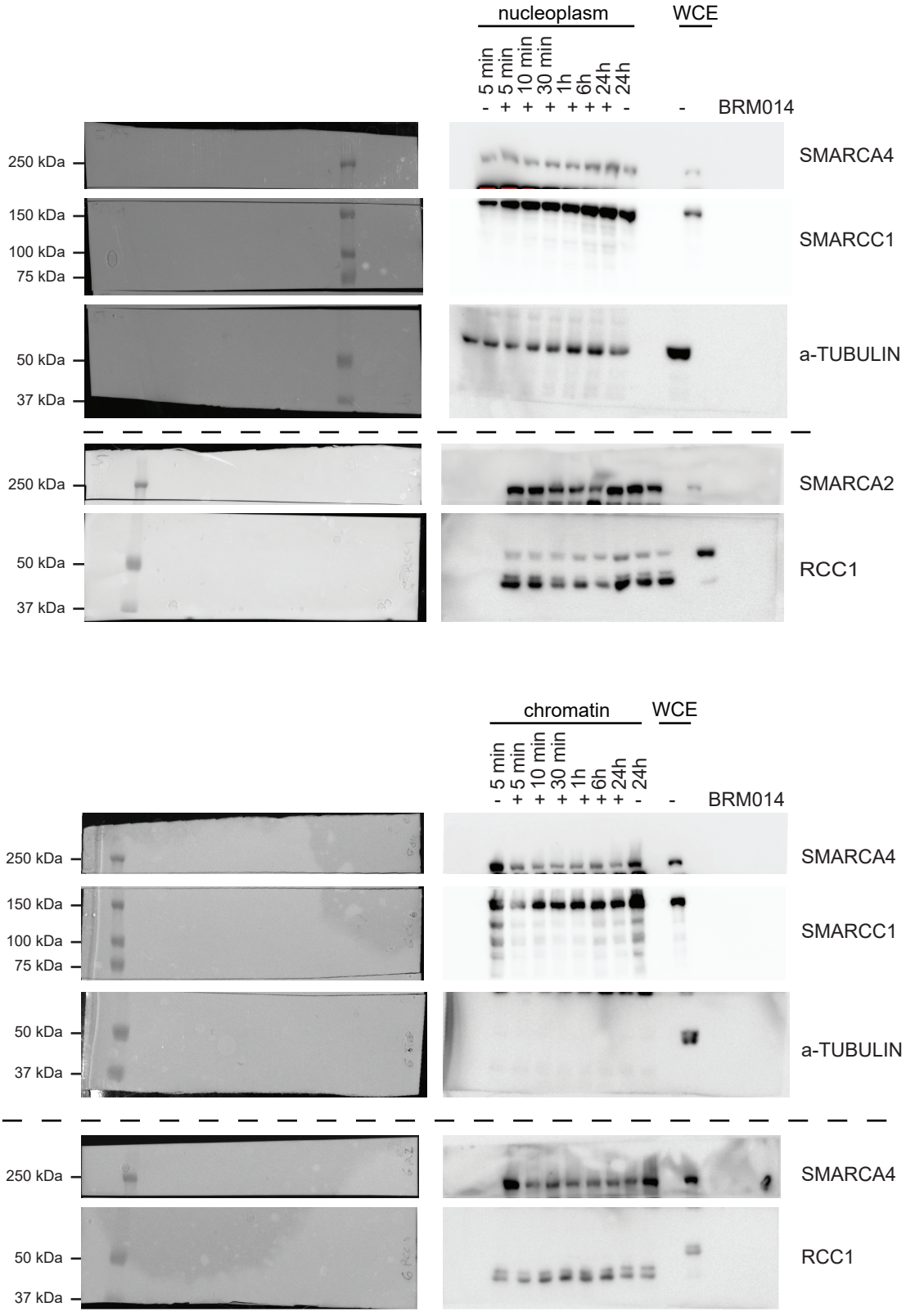
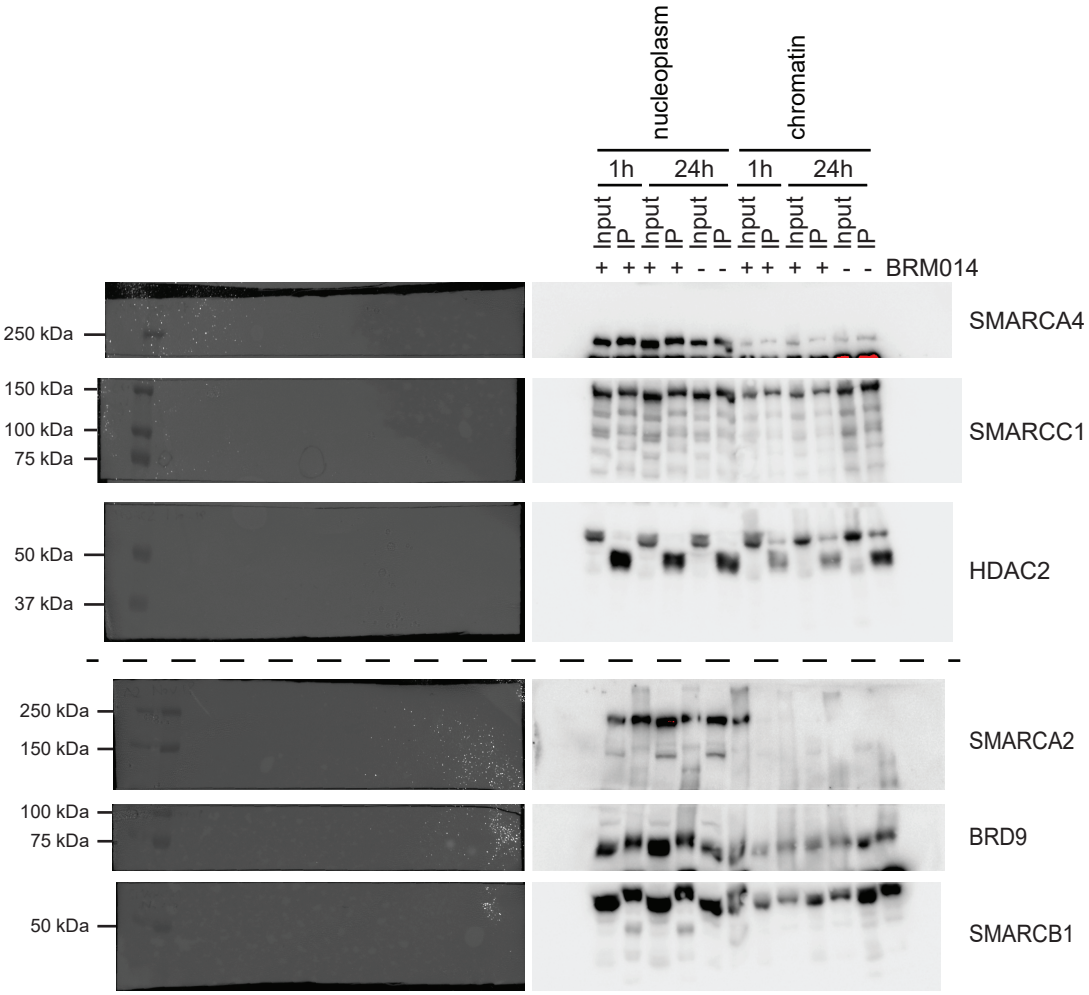
250 kDa
SMARCA2
50 kDa
37 kDa
RCC1

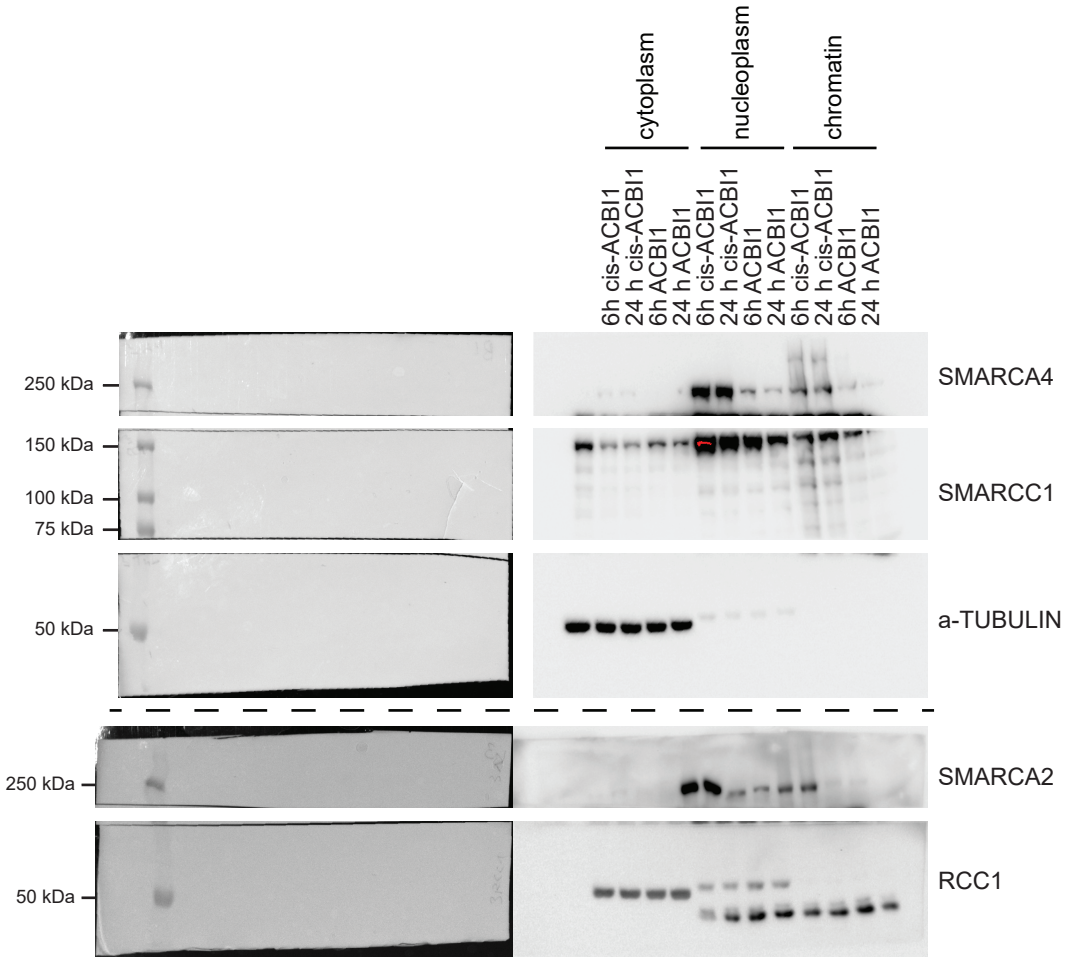
ED5a


nucleoplasm
WCE
- 5 min
+ 5 min
+ 10 min
+ 30 min
+ 1h
+ 6h
+ 24h
- 24h
- BRM014
250 kDa
150 kDa
100 kDa
75 kDa
50 kDa
37 kDa
SMARCA4
SMARCC1
a-TUBULIN
250 kDa
50 kDa
37 kDa
SMARCA2
RCC1
chromatin
WCE
- 5 min
+ 5 min
+ 10 min
+ 30 min
+ 1h
+ 6h
+ 24h
- 24h
- BRM014
250 kDa
150 kDa
100 kDa
75 kDa
50 kDa
37 kDa
SMARCA4
SMARCC1
a-TUBULIN
250 kDa
50 kDa
37 kDa
SMARCA4
RCC1

ED5b


nucleoplasm
chromatin
1h
24h
1h
24h
Input
IP
Input
IP
Input
IP
Input
IP
Input
IP
+ + + + - - + + + + - - BRM014
250 kDa
SMARCA4
150 kDa
100 kDa
75 kDa
SMARCC1
50 kDa
37 kDa
HDAC2
250 kDa
150 kDa
SMARCA2
100 kDa
75 kDa
BRD9
50 kDa
SMARCB1

ED8a


cytoplasm
nucleoplasm
chromatin
6h cis-ACBI1
24 h cis-ACBI1
6h ACBI1
24 h ACBI1
6h cis-ACBI1
24 h cis-ACBI1
6h ACBI1
24 h ACBI1
6h cis-ACBI1
24 h cis-ACBI1
6h ACBI1
24 h ACBI1
250 kDa
SMARCA4
150 kDa
100 kDa
75 kDa
SMARCC1
50 kDa
a-TUBULIN
250 kDa
SMARCA2
50 kDa
RCC1

Supplementary Figure 1a