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Supplementary Materials for

ARID1A spatially partitions interphase chromosomes

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Supplementary Materials

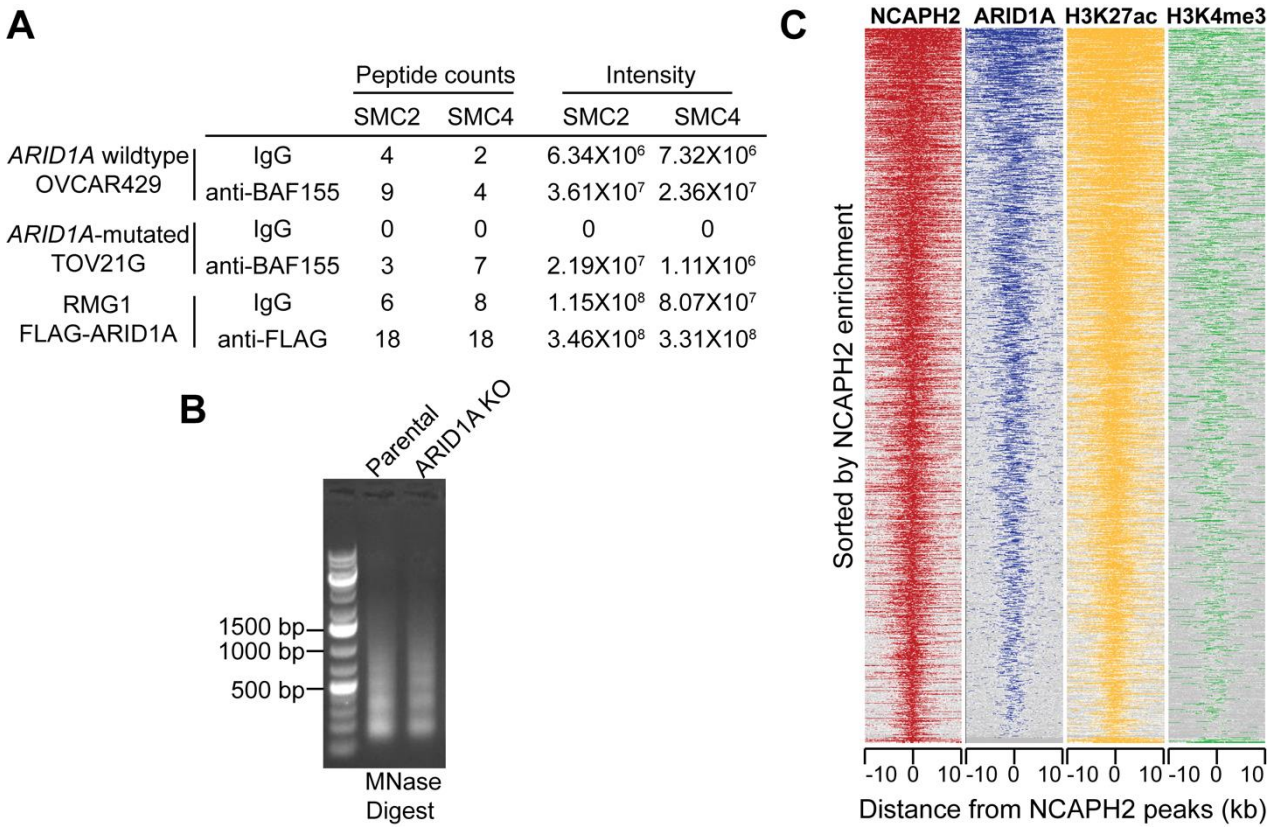

Fig. S1. The SWI/SNF complex colocalizes with the condensin II complex at enhancers. **(A)** Raw peptide counts and intensity of condensin subunits SMC2 and SMC4 identified by LC-MS/MS in pull-downs using an antibody against SNF5, a core subunit of the SWI/SNF complex, from the OVCAR429 and TOV21G OCCC cells or using an anti-FLAG antibody against endogenously FLAG-tagged ARID1A from the RMG1 OCCC cells. **(B)** Gel images of MNase digested chromatin of control parental *ARID1A* wildtype and ARID1A knockout RMG1 cells used for ChIP-seq analysis. **(C)** Heatmaps of ChIP-seq profiles of NCAPH2, ARID1A, H3K27ac and H3K4me3 in *ARID1A* wildtype RMG1 cells sorted by NCAPH2 enrichment.

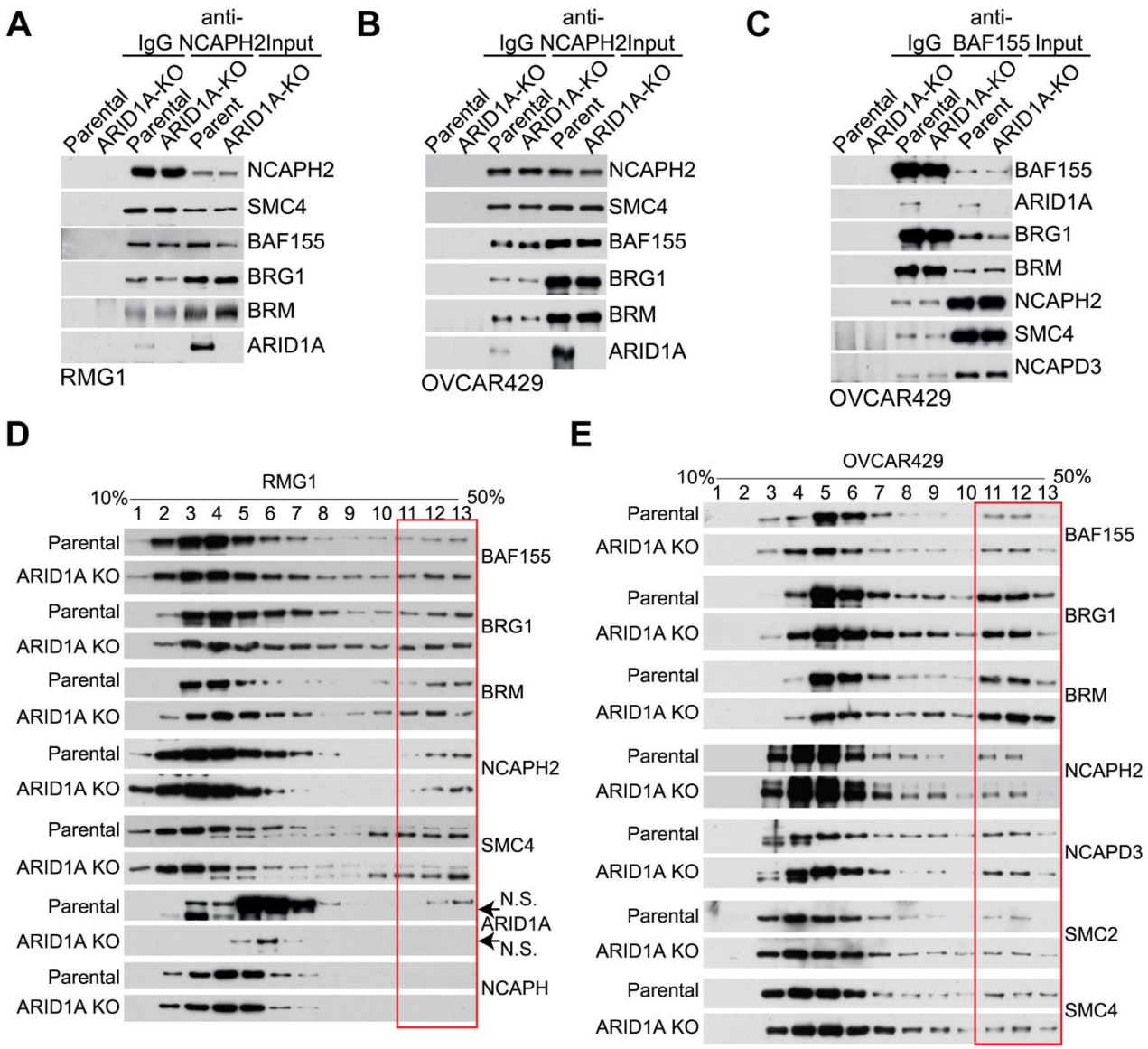

Fig. S2. ARID1A loss does not affect the interaction between the SWI/SNF and condensin II complexes. (A) Parental control and ARID1A knockout RMG1 cells were subjected to co-IP analysis using an antibody against NCAPH2 subunit of the condensin II complex and examined for interactions with the indicated subunits of the condensin II and SWI/SNF complexes by immunoblotting. **(B-C)** Parental control and ARID1A knockout OVCAR429 OCCC cells were subjected to co-IP analysis using an antibody against NCAPH2 subunit of the condensin II complex (**B**) or BAF155 subunit of the SWI/SNF complex (**C**), and examined for interactions with the indicated subunits of the condensin II and SWI/SNF complexes by immunoblotting. **(D-E)** Sucrose sedimentation (10-50%) assay of the SWI/SNF and condensin II complexes from parental control and ARID1A knockout RMG1 cells (**D**), and parental control and ARID1A knockout OVCAR429 cells (**E**). The red box indicates the co-fractionation of SWI/SNF and condensin II complexes. Arrows point to non-specific bands in immunoblotting.

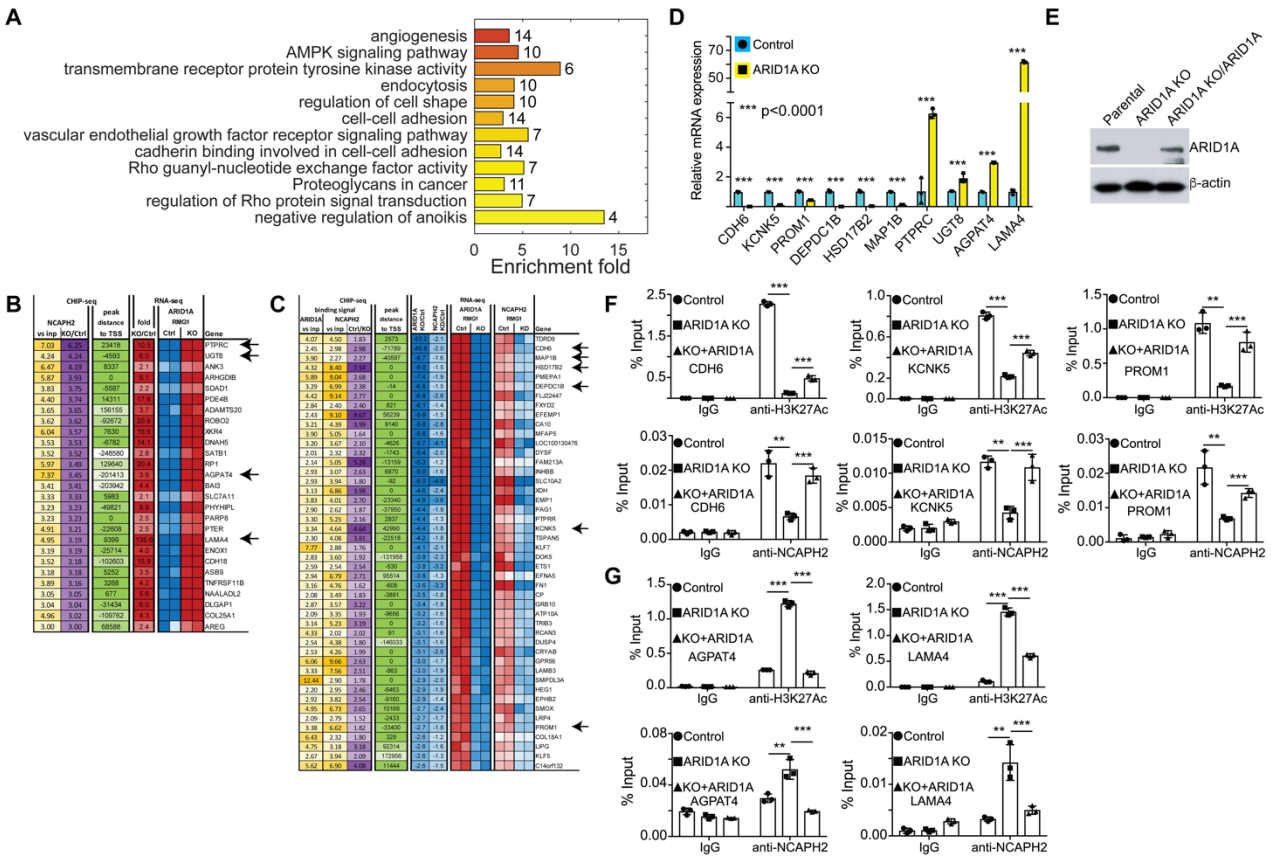

Fig. S3. ARID1A loss redistributes condensin II complex binding at enhancers to regulate gene expression. (A) List of functions and pathways enriched in the genes whose loci showed a decrease in NCAPH2 occupancy at least 2-fold in ARID1A knockout RMG1 cells. Numbers within the box indicate number of genes in the indicated functions and pathways. **(B)** List of overlapped genes that are direct ARID1A/NCAPH2 targets and upregulated in both ARID1A knockout and NCAPH2 knockdown cells with an increase in NCAPH2 binding induced by ARID1A knockout in RMG1 cells. **(C)** List of genes for which both NCAPH2 binding and expression were decreased by ARID1A knockout. Arrows point to genes that were randomly selected for validation. **(D)** Validation of the selected down or upregulated NCAPH2 target genes induced by ARID1A knockout in RMG1 cells using qRT-PCR. **(E-G)** Expression of ARID1A in the indicated RMG1 cells determined by immunoblot **(E)**. Validation of changes in the association of H3K27ac and NCAPH2 with the indicated NCAPH2 target downregulated (**F**) and upregulated (**G**) genes in control, ARID1A knockout and ARID1A rescued RMG1 cells. *** p<0.0001 and ** p<0.001

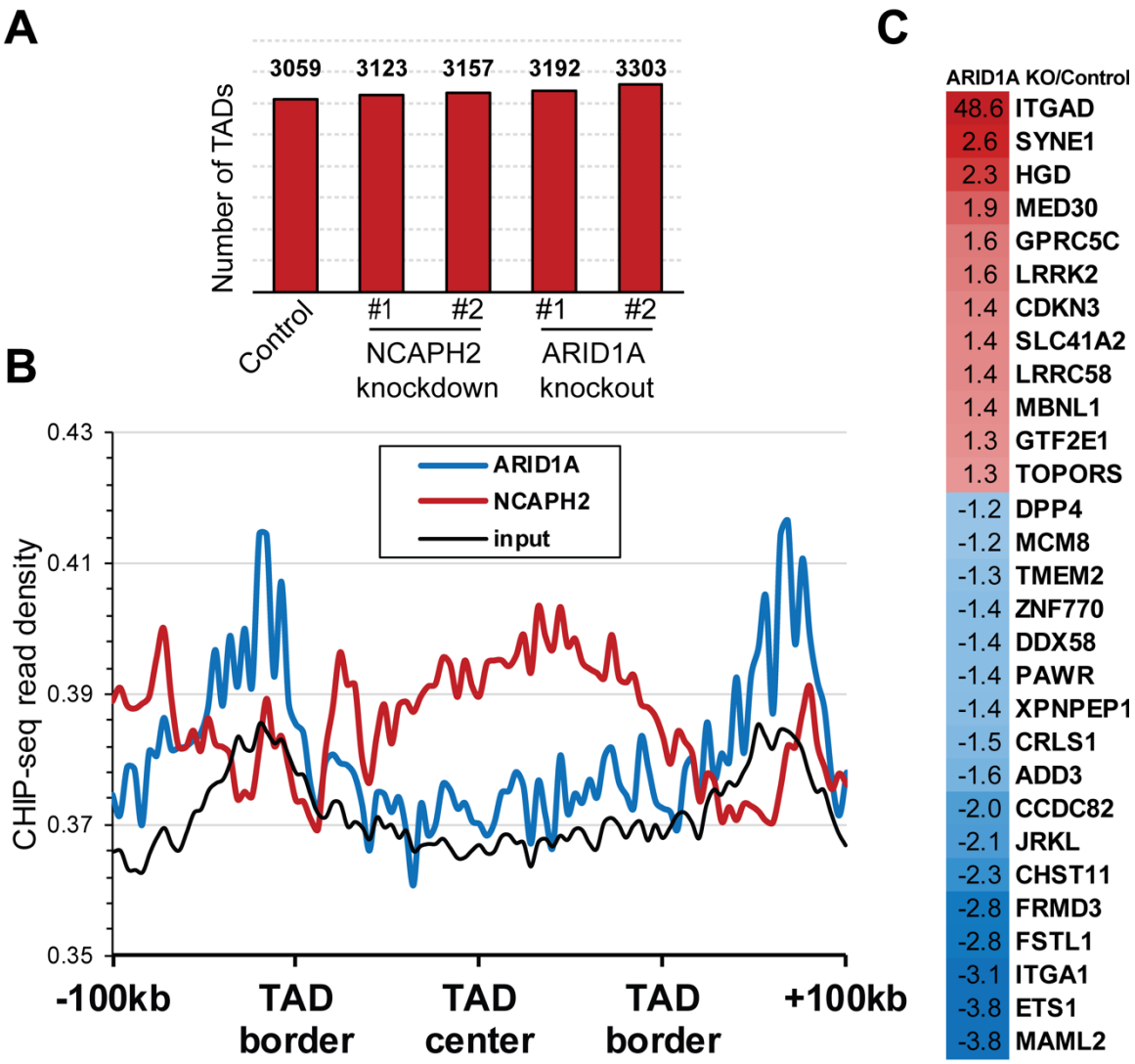

Fig. S4. ARID1A suppresses insulation of TADs. (A) Unique number of TADs identified in control, NCAPH2 knockdown and ARID1A knockout RMG1 cells. **(B)** Average ChIP-seq peak intensity of ARID1A and NCAPH2 across the TADs. Note that this is the same graph as Fig. 5B with different scale after removing H3K27ac and H3K4me3 plots. **(C)** List of genes whose changes in expression correlates with the switch of compartments induced by ARID1A knockout in RMG1 cells based on RNA-seq analysis. Those upregulated genes are associated with a B to A compartment switch, while those downregulated genes are associated with an A to B compartment switch.

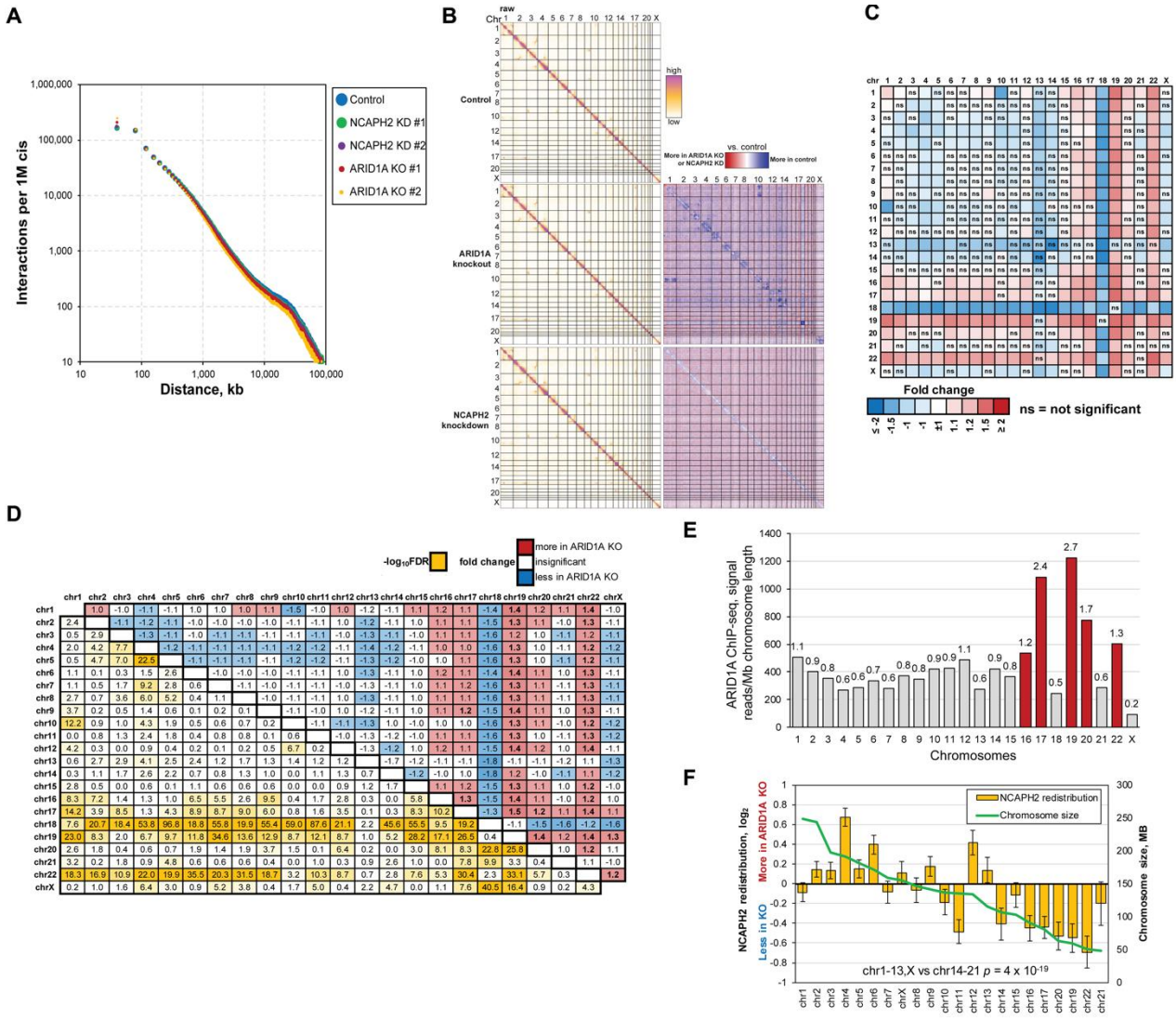

Fig. S5. ARID1A partitions chromosomal territories. (A) Genome-wide interaction frequency (per 1M intra-chromosomal reads) versus genomic distance in the indicated parental control, NCAPH2 knockdown and ARID1A knockout RMG1 cells. **(B)** Heatmap of genome-wide Hi-C interaction and comparison of the interaction between parental control and ARID1A knockout or NCAPH2 knockdown RMG1 cells. **(C)** Genome-wide intra- and inter-chromosomal interaction change in ARID1A knockout cells relative to parental RMG1 controls. **(D)** Genome-wide inter-chromosomal interaction change in ARID1A knockout relative to parental control RMG1. **(E)** ARID1A ChIP-seq signal reads within significant peaks per Mb of chromosome length across each of individual chromosomes in *ARID1A* wildtype RMG1 cells. Numbers show the signal ratio versus average across chromosomes with red bars indicating chromosomes with the ratio >1.2. **(F)**, Correlation between NCAPH2 redistribution and chromosome size.

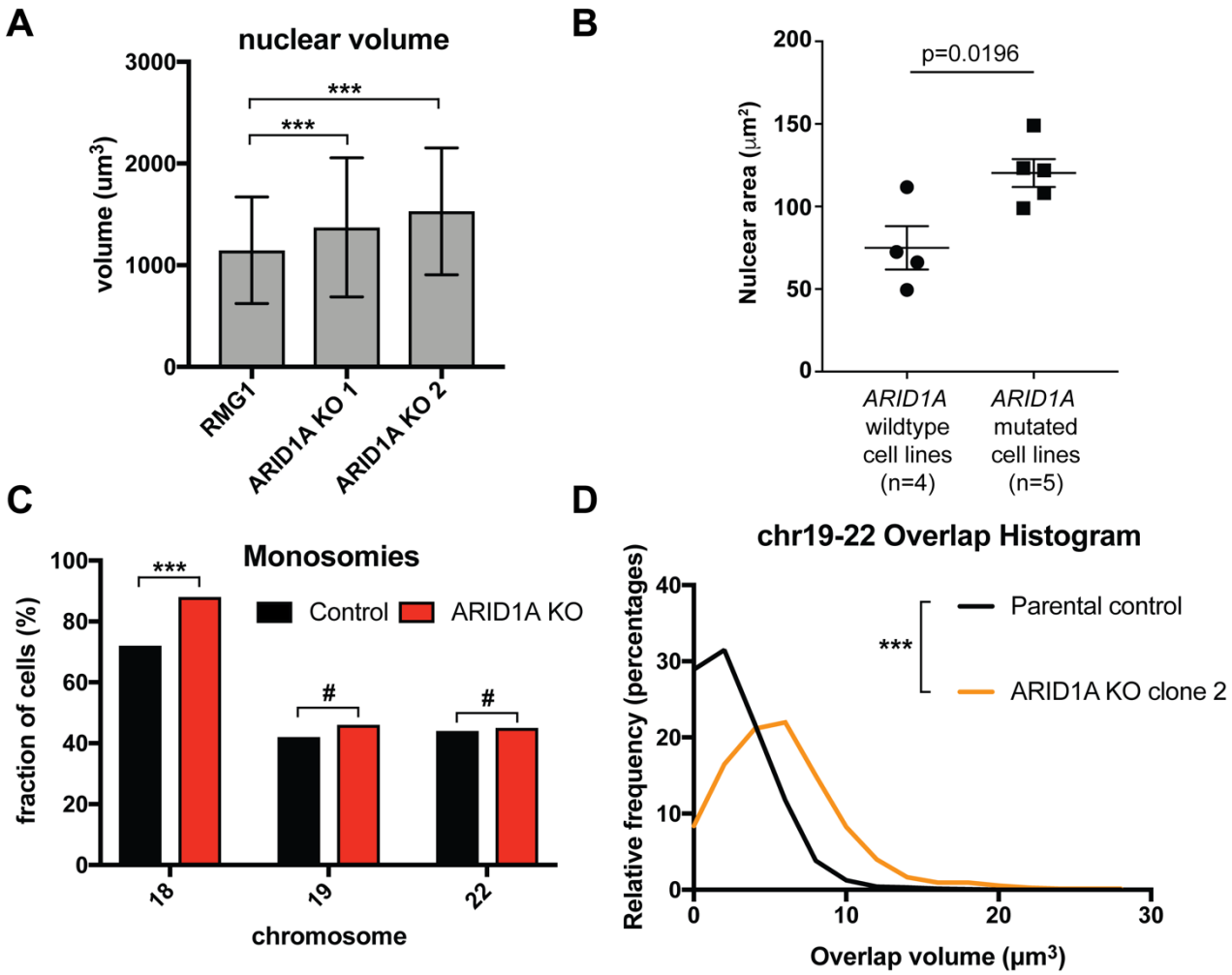

Fig. S6. ARID1A loss increases chromosome and nuclei volume and promotes small chromosome intermixing in trans. (A) Nuclear volume of parental control and two independent ARID1A knockout RMG1 clones (measured from at least 576 nuclei). **(B)** Comparison of nuclear area between four individual *ARID1A* wildtype OCCC cell lines and five different *ARID1A*-mutated OCCC cell lines. Error bars = mean with SEM. **(C)** Percentage of monosomies for chr18, chr19, and chr22 in the indicated parental control and ARID1A knockout RMG1 cells (based on 3D chromosome painting from at least 747 nuclei from each of the indicated groups). **(D)** Distribution of 3D chromosome overlap area (from > 747 nuclei) between chr19 and chr22 in parental control and the second ARID1A knockout RMG1 clone. *** $p<0.0001$ and # $p>0.05$.