Supplemental Data

Supplemental Material and Methods

Super-enhancer Identification

Super-enhancers were identified using RANK ORDERING OF SUPER-ENHANCERS (ROSE) (https://bitbucket.org/youngcomputation/rose)(Hnisz et al. 2013; Loven et al. 2013; Whyte et al. 2013). H3k27ac peaks within 12.5 kb of each other, except for those that were fully contained within +/- 2 kb of a transcriptional start site (TSS), were ranked along the x axis based on their H3K27ac enrichment and plotted on the y axis. Super-enhancers were subsequently identified as regions which are to the right of the inflection point based on the resulting curve. Both enhancers and super-enhancers were assigned to the nearest RefSeq genes.

CRISPR/Cas9 Gene Knockout

For genetic knockout experiments, single guide RNA (sgRNA) was designed using the CRISPR Design Tool (http://crispr.mit.edu/) and cloned into lentiCRISPv2 (Addgene plasmid # 52961) or FgH1tUTG (Addgene plasmid # 70183) using BsmB1 enzyme sites. Lentiviruses were produced using the same protocol for shRNA knockdown analysis. Jurkat cells infected with the virus were selected by 0.7 µg/ml of puromycin (Sigma) from day 3 to day 7. To identify genetic deletion, we isolated genomic DNA using the QIAamp DNA Blood Mini Kit (Qiagen) followed by PCR amplification using specific primers flanking the -135 kb element, as follows: forward, 5′-CGT CAA CCA CCA CTG CTT TT-3′; reverse, 5′-TTC CAG TAA CGT GGC AGT CC-3′.

shRNA Sequences

shRNA	Sequence
shGFP	ACA ACA GCC ACA ACG TCT ATA
shLUC	CTT CGA AAT GTC CGT TCG GTT
shARID5B #3	CTA CAC CTG TAG GAA GTT CAT
shARID5B #7	GCC TTC AAA GAG AAC CAT TTA
shTAL1	GCT CAG CAA GAA TGA GAT CCT
shHEB	CCA TCC CAT AAT GCA CCA ATT
shE2A	CCC GGA TCA CTC AAG CAA TAA
shGATA3	GCC TAC ATG CTT TGT GAA CAA
shRUNX1	CAG AGT CAG ATG CAG GAT ACA
shMYB	CCA GAT TGT AAA TGC TCA TTT
shLMO1	CGC GAC TAC CTG AGG CTC TTT

qRT-PCR Primers

Genes	Species	Forward	Reverse
TAL1	Human	TTC CCT ATG TTC ACC ACC AA	AAG ATA CGC CGC ACA ACT TT
GATA3	Human	TTC AGT TGG CCT AAG GTG GT	CGC CGG ACT CTT AGA AGC TA
RUNX1	Human	GTG TCT TCA GCC AGA TG	CGA CTG TGT ACC GTG GAC TG

MYB	Human	TGT TGC ATG GAT CCT GTG TT	AGT TCA GTG CTG GCC ATC TT
ARID5B	Human	CAG AAG AAT GCT GAG CCA AC	TGG GAA ACT ATT GGC ACG TA
MYC	Human	TCA TTG GAA AAT TGA CAG CAT AGT	GTC GTT TCC GCA ACA AGT CCT CTT C
ALDH1A2	Human	AGG CCC TCA CAGTGTCTT CT	ACA TCT TGA ATC CCC CAA AG
MAX	Human	TTC CTC CCT CAT GGA AGA TG	GCT CTT CAG GCT CAG ACT CC
EGR1	Human	CTT CAA CCC TCA GGC GGA CA	GGA AAA GCG GCC AGT ATA GGT
EGR2	Human	GCA TAA GCC CTT CCA GTG TC	TGC TTT TCC GCT CTT TCT GT
CDKN1A	Human	AGG TGG ACC TGG AGA CTC TCA G	TCC TCT TGG AGA AGA TCA GCC G
HnRNPH3	Human	CGA CCG GGA CCA TAT GAT AG	TGA ACT TGC ATC ACC AGC TC
Exon 7 of C10orf107	Human	AAG AGG CCT TTA ATG CAC GA	GAA ACA AAC AAA ACC AGC CA
GAPDH	Human	CTC CTC TGA CTT CAA CAG CGA CAC	TGC TGT AGC CAA ATT CGT TGT CAT
Arid5b	Mouse	GGC CAA CTA CAT TGC CAA CT	GGG ACA TGA TAC CAG GGT TG
Myc	Mouse	AGC TGT TTG AAG GCT GGA TT	AAT AGG GCT GTA CGG AGT CG

β-Actin	Mouse	GGC TGT ATT CCC CTC CAT CG	CCA GTT GGT AAC AAT GCC ATG T
myca	Zebrafish	CGC GCT ACG GGA TGA GAT CCC T	GCA GGG GGT GGG AGT TCT TGG A
mycb	Zebrafish	AAG CGG CCA AAG TGG TGA TCC	CAC TAC TTT GCC ACA CCC TCG C
ef1a	Zebrafish	CTG GAG GCC AGC TCA AAC AT	ATC AAG AAG AGT AGT ACC GCT AGC ATT AC
cd4	Zebrafish	TTT ACG CAC AGG TAG GAG GGA	CTC TGC GGG TTC CTG TTG AT
cd8	Zebrafish	AAT CGC AAA GCA GAC GGA AG	AGT CCG CTG TCT GTC CTT TT
tcra	Zebrafish	ACC AAG TGG GAA ACT CAT GC	TGC CCA GTG ACA AGA AGT TG
lck	Zebrafish	GCC TCC AGT CAG TCA GAA TTT	TTG TAT ATG GCC ACC ACC AG
E130 (ERCC spike-in)	Synthesized RNA	GCT TGA GGA GCT TGA AGC AG	GCG GTC GGT ATA AAA TCA GG

ChIP-PCR Primers

Primers for the analysis of transcription factor binding

Targets	Forward	Reverse

N-Me (1)	ATG GGG TTC CCA TGG TAT TT	GCC CTG CTG TTT CAT GAT TT
TAL1 (1)	CCT CTC ACC ACT TGC TCT CC	CCC CAC CCC ATT CCT ATT AC
GATA3 (1)	CGC ACG GTA AGC AGG AAG	AGC TCA GCA TGT TTC TGC AA
RUNX1 (1)	CCT GTG GTT TTC TCG CTC TC	TGC ACC TGC AGA GTT TTC AC
MYB (1)	ATA ATG TCT CCG CGA TGG AT	GCT TTG GTT TCA GCT GCT CT
IGFBP3 (control locus)	AGT ACT CTG CAC TTA GAG AAT CGA G	TCT TCT CAC TGG AGA TAA TAT GTG G

Primers for the analysis of H3K27ac

Targets	Forward	Reverse
N-Me (2)	CTT AGG TTG GAG GCA CGA AA	CAT GTC CAA GCA GGA AGG TT
TAL1 (2)	CTG TCA CCA CTC CCA GCT AA	ATG CAG AAA GTT CCC TGT GC
GATA3 (2)	TTG TTC AGC AGA GGA TGC AG	GCC CTT CTC AAC AGT TCC TG
RUNX1 (2)	GGG GGT CAA ATC TTT TGG TT	AGA GAG TTG ACC TGG CCT GA

MYB (2)	GAT ATG GCA GTG GCT GCA C	ATG GAG GTC TGG CTT TGT TG

CRISPR/CAS9 sgRNA Sequences

sgRNA	Sequence
EGFP	CAA GTT CAG CGT GTC CGG CG
sgRNA #1	AGG TTT TGT GAT TGC CGA GG
sgRNA #2	AGG CCT AAG GAC TTG GTA CA
sgARID5B Exon 6	GAA AAA CCA AAG GTT GCC AT

Cell Cycle Analysis

Cells transduced with lentivirus expressing shRNA were harvested after 3 days, washed with PBS and fixed overnight using 70% ethanol. The cells were subsequently incubated with propidium iodide and analyzed with a BD™ LSR II flow cytometer using BD FACSDiva™ software.

Immunoprecipitation

Cells were lysed using IP lysis buffer containing 150 mM NaCl, 20 mM Tris pH 7.5, 1 mM EDTA, 0.5% NP40, 10% glycerol and 1x protease inhibitor (Roche). One microgram of a primary antibody, IgG (Santa Cruz) or ARID5B (Bethyl Laboratories), TAL1 (Santa Cruz) was added to 1 mg of protein. The protein lysate and antibody were incubated overnight at 4°C. The immune complex was precipitated using Dynabeads® Protein G (Thermo

Fisher Scientific), and this was followed by Western blot analysis with specific antibodies against ARID5B (Bethyl Laboratories) and PHF2, HDAC1, HDAC2, HDAC3 and HDAC4 (Cell Signaling Technology).

HDAC Inhibitor Treatment

Jurkat cells were treated with either DMSO or SAHA (2μM) (Sigma Aldrich) in RPMI-1640 medium. At 24 hours after drug treatment, cells were harvested for qPCR analysis as described in RNA Extraction and Gene Expression Analysis.

Western Blot Analysis for H3K27Ac

Jurkat cells were infected with shLUC (Control), shARID5B #3 and #7 lentivirus in the presence of polybrene (8 μg/ml; Millipore) by centrifugation at 1,300 rcf for 1.5 hr. The infected cells were selected with 0.7 μg/ml of puromycin (Sigma) in RPMI-1640 medium for at least 36 hr after infection. At day 3 after infection, cells were harvested for protein extraction using radioimmunoprecipitation assay (RIPA) buffer. Twenty microgram of protein lysate was used for western blot analysis with antibodies specific to H3K27ac and H3 (Cell Signalling Technology).

Overexpression of ARID5B and PHF2 in 293T Cells

Six microgram of pCS2+ mammalian expression constructs for expression of human *ARID5B* and *PHF2* cDNAs were transfected into 293T cells grown in 10 cm Petri dish

using FuGENE® 6 Transfection Reagent (Promega). At 48 hours after transfection, cells were harvested for immunoprecipitation.

Inducible shRNA Knockdown

The shRNA sequences targeting the *ARID5B* mRNA were designed according to the RNA Consortium's recommendation (http://www.broadinstitute.org/rnai/trc) and cloned into the inducible lentivirus expression vector Tet-pLKO-puro. Lentiviruses were produced by co-transfecting individual shRNA constructs with the packaging plasmids pMDLg/pRRE and pRSV-Rev and the envelope plasmid pMD2.G into 293T cells by using FuGENE 6 transfection reagent (Promega). Supernatants containing lentivirus particles were collected and filtered through a 0.45 µm filter (Thermo). Jurkat cells expressing doxcycyline dependent shRNA were established through lentiviral infection in the presence of polybrene (8 µg/ml; Millipore) by centrifugation at 1,300 rcf for 1.5 hr. The infected cells were selected with 0.7 µg/ml of puromycin (Sigma) in RPMI-1640 medium for at least 36 hrs after infection. shRNA knockdown was induced by culturing the cells with 1 µg/mL of Doxycycline in RPMI-1640 media for at least 24 hrs followed by Western blot analysis.

Overexpression of BCL2 and MYC in T-ALL Cells

Retrovirus were produced by co-transfecting the retrovirus vector, MSCV-IRES-GFP containing the *BCL2* or *MYC* cDNA with packaging plasmids, pMD-MLV and the envelope plasmid pCMV-VSV-G into 293T cells by using FuGENE 6 transfection reagent

(Promega). Supernatants containing retrovirus particles were collected and filtered through a 0.45 µm filter (Thermo). Jurkat cells were infected in the presence of polybrene (8 µg/ml; Millipore) by centrifugation at 1,300 rcf for 1.5 hr. After 3 days, infected cells were analyzed with a BD™ LSR II flow cytometer using BD FACSDiva™ software. Successfully infected cells that express GFP were sorted using FACS Aria Flow Cytometer (BD Biosciences).

Inducible CRISPR/Cas9 Knockout

Cas9-expressing Jurkat cells was established using lentiviruses produced through co-transfecting of FUCas9Cherry (Addgene plasmid #70182) with the packaging plasmids pMDLg/pRRE and pRSV-Rev and the envelope plasmid pMD2.G into 293T cells. after infection, cells were analyzed with a BD™ LSR II flow cytometer using BD FACSDiva™ software. Successfully infected cells that express mCherry were sorted using FACS Aria Flow Cytometer (BD Biosciences). The single guide RNA (sgRNA) sequences targeting the *ARID5B* genomic DNA were designed using the CRISPR Design Tool (http://crispr.mit.edu/) and cloned into the inducible lentivirus expression vector FgH1tUTG (Addgene plasmid # 70183) using BsMB1 enzyme sites. Lentivirus was produced using the same protocol as described for shRNA knockdown and infected into Jurkat cells that stably expresses Cas9. After 3 days, cells were analyzed with a BD™ LSR II flow cytometer. Dual-positive eGFP and mCherry cells were sorted using FACS Aria Flow Cytometer (BD Biosciences). sgRNA expression was induced by culturing cells with 1 µg/mL of Doxycycline in RPMI-1640 media for 6 days and protein expression was analyzed using Western blot.

Zebrafish Genotyping

Zebrafish were selected based on the presence of mCherry fluorescence and subsequently genotyped using primers targeting the human *ARID5B* gene. The primers used for sequencing were as follows: rag2, forward, 5'-AAA TGG AAG GCC TGG AAG CAT CGG-3'; ARID5B, reverse, 5'-GTC CTC TCT TCC CAC AAC AGC-3'.

Supplemental Figure Legend

Supplemental Figure S1. ARID5B is regulated under the -135kb enhancer in T-ALL

(A) Gene expression changes of 13 target genes of the TAL1 transcriptional complex after *TAL1* knockdown. See Figure 1A legend for details. **(B)** Schematic diagram indicating the sgRNA target sites (blue arrows), PCR primers (gray arrows) and the size of the deleted region (green arrows). **(C)** The sgRNAs (#1 and 2) targeting the 5' and 3' ends of the -135 kb element, respectively, or a control sgRNA targeting *EGFP* were transduced into Jurkat cells by lentiviral infection. Genomic DNA was harvested at day 6 after lentivirus infection and amplified using specific primers by PCR. **(D)** Sanger sequence chromatogram in the knockout cells. The black arrowheads indicate the genomic DNA cleavage site targeted by the sgRNA (#1 or #2). **(E)** The mRNA expression of exon 7 of the *C10orf107* gene, which is located under the -135 kb element, was measured by qRT-PCR analysis in control and knockout samples. *p<0.05 by two-sample, two-tailed t-test. **(F)** Gene Expression Commons database showing mouse *Arid5b* mRNA expression in hematopoietic cell subpopulations. **(G)** Expression of human *ARID5B* gene in different stage of human hematopoietic cells were analyzed using an RNA-seq dataset reported by Casero *et al*. (Casero et al. 2015). Two samples were included for each fraction in the original dataset. Expressions are shown by FPKM values. Hematopoietic stem cells (HSC), $CD34^{+}CD38^{neg}lin^{neg}$; lymphoid-primed multipotent progenitors (LMPP), $CD34^{+}CD38^{+}CD10^{neg}CD45RA^{+}CD62L^{high}lin^{neg}$; common lymphoid progenitor (CLP), $CD34^{+}CD38^{+}CD10^{+}CD45RA^{+}lin^{neg}$; Thy1, $CD34^{+}CD7^{neg}CD1a^{neg}CD4^{neg}CD8^{neg}$; Thy2, $CD34^{+}CD7^{+}CD1a^{neg}CD4^{neg}CD8^{neg}$; Thy3, $CD34^{+}CD7^{+}CD1a^{+}CD4^{neg}CD8^{neg}$; Thy4, $CD4^{+}CD8^{+}$; Thy5, $CD3^{+}CD4^{+}CD8^{neg}$; and Thy6, $CD3^{+}CD4^{neg}CD8^{+}$. Thy1-3, Thy4, Thy5

and Thy6 represent double-negative (DN), double-positive (DP), CD4 single-positive (SP) and CD8 SP cells, respectively.

Supplemental Figure S2. ARID5B overexpression supports the survival of TAL1-positive T-ALL cells

(A) Cell viability of Jurkat cells overexpressing BCL2 was measured by CellTiter Glo assay at days 3, 5 and 7 post-infection with lentivirus expressing shLUC, (control), shARID5B-3 or shARID5B-7. Cell growth rates (fold-change compared to day 3) are shown as the mean ±standard deviation (SD) of duplicate samples. **(B)** Western blot analysis for protein expressions of ARID5B, PARP and α-tubulin (loading control) in Jurkat cells on day 3 after shRNA-expressing lentivirus infection. **(C)** Cell cycle distribution of Jurkat cells on day 3 after shRNA-expressing lentivirus infection was measured by flow cytometry using propidium iodide DNA staining. The data represent the mean ± SD of duplicate samples. **(D)** Detection of apoptosis by Annexin V staining in CCRF-CEM, PF-382, MOLT-4 and LOUCY cells on day 3 after transduction with shRNA-expressing lentivirus. The data represent the mean of duplicate samples. See Figure 2B legend for details.

Supplemental Figure S3: ARID5B-bound regions are predominantly associated with active histone marks. (A) Immunoprecipitation assay performed in Jurkat cells with IgG, ARID5B or TAL1-specific antibodies. The whole-cell lysate (WCL), immunoprecipitate (IP) and flow-through (FT) were analyzed by immunoblotting (IB) with

HDAC1-, HDAC2-, HDAC3- and HDAC4-specific antibodies. Of note, TAL1 did not interact with any of HDAC proteins in this analysis. **(B)** mRNA expression of *EGR1*, *EGR2* and *CDKN1A* in Jurkat cells on day 3 after infection with shGFP and shARID5B-3 was analyzed by qRT-PCR. The relative gene expression was normalized to the ERCC Spike-in exogenous control (*E130*). The data represent the mean ± SD of duplicate samples. *p<0.05 by two-sample, two-tailed t-test. **(C)** mRNA expression of *EGR1*, *EGR2* and *CDKN1A.* Jurkat cells treated for 24 hrs with DMSO or a small-molecule HDAC inhibitor (SAHA) at a concentration of 2 μM were analyzed by qRT-PCR. The relative gene expression was normalized to the ERCC Spike-in exogenous control *(E130).* The data represent the mean ± SD of duplicate samples. *p<0.05, **p<0.01 by two-sample, two-tailed t-test. **(D)** "Active" genes in Jurkat were defined as those bound by RNA polymerase II and H3K4me3 within +/- 2.5 kb of the TSS and also bound by H3K79me2 in the first 5 kb of the gene. All selected genes were then ranked by the ARID5B signals. Top 500 genes with the highest ARID5B signals (ARID5B targets). Bottom 500 genes with the lowest ARID5B signals (non-ARID5B targets). **(E)** Jurkat cells were transduced with shLUC (control), shARID5B-3 or shARID5B-7 for 3 days. Protein expression of ARID5B, H3K27ac and total H3 (loading control) were analysed by Western blot. **(F)** WCL was subjected to immunoprecipitation using an anti-ARID5B antibody or control IgG followed by immunoblotting (IB) analysis with an anti-ARID5B or PHF2 antibody. IP, immunoprecipitant; FT, flowthrough. **(G)** 293T cells were transfected with constructs for expression of human *ARID5B* and *PHF2* cDNAs. At 48 hours after transfection, WCL were subjected to immunoprecipitation using an anti-ARID5B antibody followed by immunoblotting (IB) with an anti-ARID5B or PHF2 antibody.

Supplemental Figure S4. ARID5B transcriptionally activates *MYC* oncogene in T-ALL cells

(A) ChIP enrichment analysis (ChEA) and Gene ontology (GO) analysis were performed in the Enricher program by using genes that were significantly downregulated after knockdown of each of transcription factors (TAL1, GATA3, RUNX1, MYB) (with an adjusted p-value<0.05 and a log2 fold-change<-0.5 between 2 control and 2 knockdown samples). The top 10 terms ranked by the combined score are shown. **(B)** Western blot analysis for protein expression of MYC in Jurkat cells overexpressing BCL2 on day 3 after the transduction of lentivirus expressing shLUC, shARID5B-3 or shARID5B-7. **(C)** The sgRNA targeting exon 6 of *ARID5B* gene was induced using a doxycycline-induced system in Jurkat cells expressing Cas9 protein to knock out ARID5B protein. Protein expression of MYC was analyzed by Western blot in control and knockout samples. **(D)** ChIP analysis was performed using an anti-ARID5B antibody or control IgG in Jurkat, CCRF-CEM, RPMI-8042 and LOUCY cells. Fold enrichment of ChIP samples compared to input (whole cell lysate) at the NOTCH1-driven *MYC* enhancer region was measured by PCR. Negative control (*IGFBP3* genomic region) that is not bound by TAL1 or ARID5B and is not associated with active histone marks was used for normalization. The error bars represent the SD of the fold enrichment. *p<0.05, ***p<0.001 by two-sample, two-tailed t-test. **(E)** Jurkat cells were transduced with a doxycycline-inducible shRNA targeting ARID5B. The cells were treated with or without doxycycline for 48 hours. ChIP analysis was performed using an anti-H3K27ac antibody or control IgG in control and knockdown samples. Fold enrichment of ChIP samples compared to input (whole cell

lysate) around the NOTCH1-driven *MYC* enhancer region was measured by PCR. **(F)** Cell viability of Jurkat cells overexpressing *MYC* was measured by CellTiter Glo assay at days 3, 5 and 7 post-infection with lentivirus expressing shLUC (control), shARID5B-3 or shARID5B-7. Cell growth rates (fold-change compared to day 3) are shown as the mean ± SD of duplicate samples. **(G)** mRNA expression of *MYC* in T-ALL cell lines was determined by microarray analysis. **(H)** Relative expression of mouse *Myc* in DN1, DN2 and DN3 populations harvested from the thymus of 8 to 10 weeks old *NOD-Rag1*^{null} *IL2rg*^{null} (*NRG*) mice. See Figure 1H legend for details. *p<0.05, **p<0.01 by two-sample, two-tailed t-test.

Supplemental Figure S5. ARID5B coordinately regulates the expression of TAL1 targets in T-ALL cells

Heatmap image representing the expression levels of TAL1 target genes in shGFP (control) and shARID5B knockdown samples.

Supplemental Figure S6. ARID5B positively regulates the expression of the TAL1 complex in T-ALL cells

(A-D) Occupancy of ARID5B at the *TAL1* (A), *GATA3* (B), *RUNX1* (C) and *MYB* (D) enhancer regions in Jurkat, CCRF-CEM, RPMI-8042 and LOUCY cells was analyzed by ChIP-PCR. See Supplemental Figure S4D legend for details. *p<0.05, ***p<0.001 by two-sample, two-tailed t-test. **(E-H)** H3K27ac signals at the *TAL1* (E), *GATA3* (F), *RUNX1* (G) and *MYB* (H) enhancer regions in Jurkat. See Supplemental Figure S4E legend for details.

*p<0.05, **p<0.01 by two-sample, two-tailed t-test. **(I)** Protein expression of ARID5B, TAL1, GATA3, RUNX1, MYB and α-tubulin on day 6 after the doxycycline-induced expression of sgRNA targeting ARID5B. See Supplemental Figure S4C legend for details. **(J)** Western blot analysis for protein expression of TAL1 in Jurkat cells overexpressing BCL2 on day 3 after the transduction of lentivirus expressing shLUC, shARID5B-3 or shARID5B-7

Supplemental Figure S7. Overexpression of ARID5B leads to thymus retention and the development of T-cell lymphoma in zebrafish

(A) Clustal Omega protein sequence alignment of the full-length human *ARID5B* and zebrafish *arid5b* proteins. **(B)** Schematic diagram of the plasmids that were co-injected into one-cell-stage embryos. Meganuclease I-SceI was used to digest and insert the zebrafish *rag2* promoter into the target gene sequences of the zebrafish genomic DNA. **(C)** Genotype of *rag2-ARID5B* transgenic zebrafish. Genomic DNA extracted from the zebrafish fin was subjected to PCR using *rag2* forward, *ARID5B* forward and *ARID5B* reverse primers.

Supplemental Tables, provided as Excel files

Supplemental Table 1. Genes significantly downregulated or upregulated by ARID5B knockdown

Supplemental Table 2. ChEA and gene ontology analysis for genes differentially-regulated by transcription factors

Supplemental Table 3. Genes significantly downregulated after TAL1 knockdown

Supplemental References

Casero D, Sandoval S, Seet CS, Scholes J, Zhu Y, Ha VL, Luong A, Parekh C, Crooks GM. 2015. Long non-coding RNA profiling of human lymphoid progenitor cells reveals transcriptional divergence of B cell and T cell lineages. *Nat Immunol* **16**: 1282-1291.

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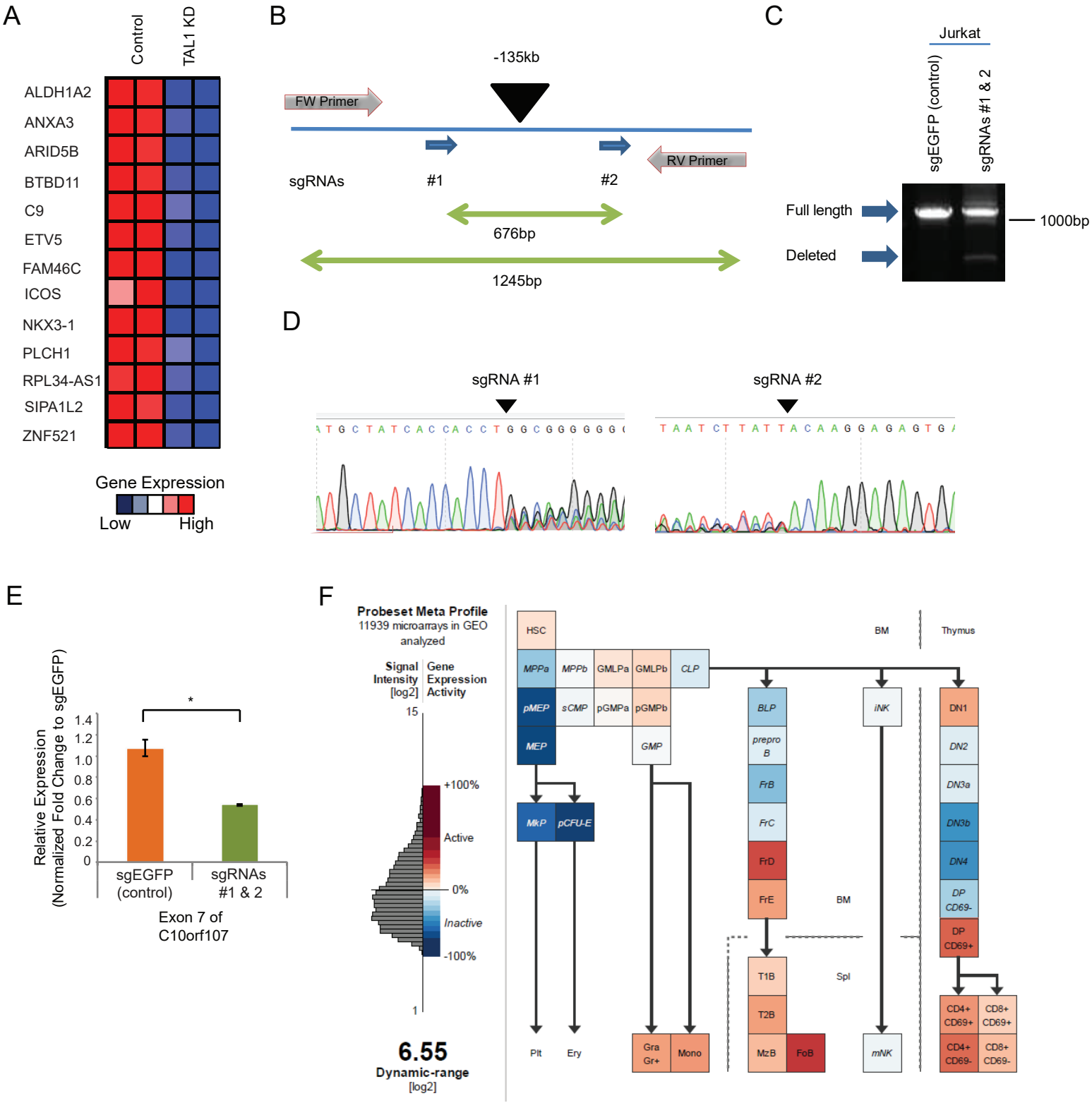
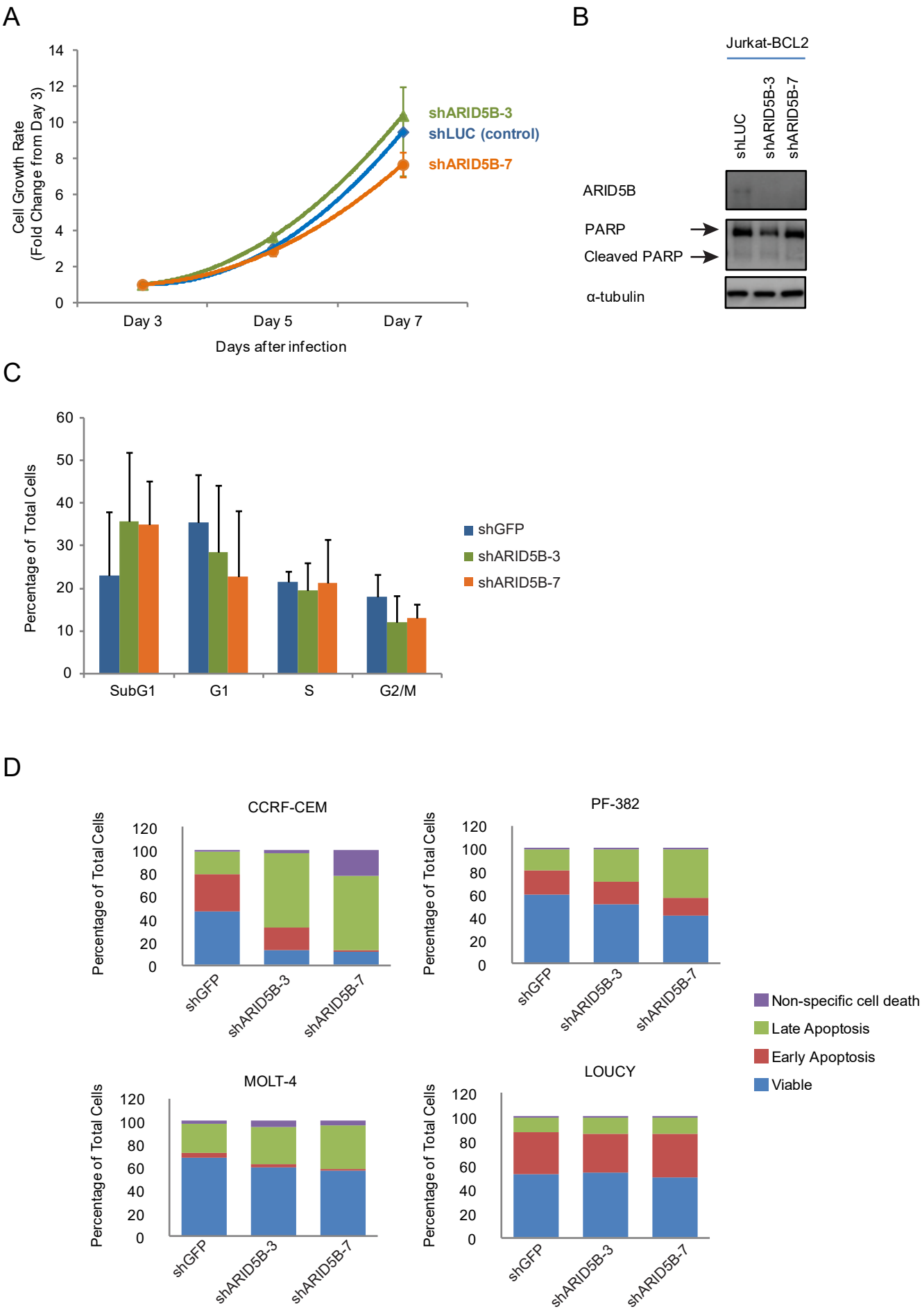
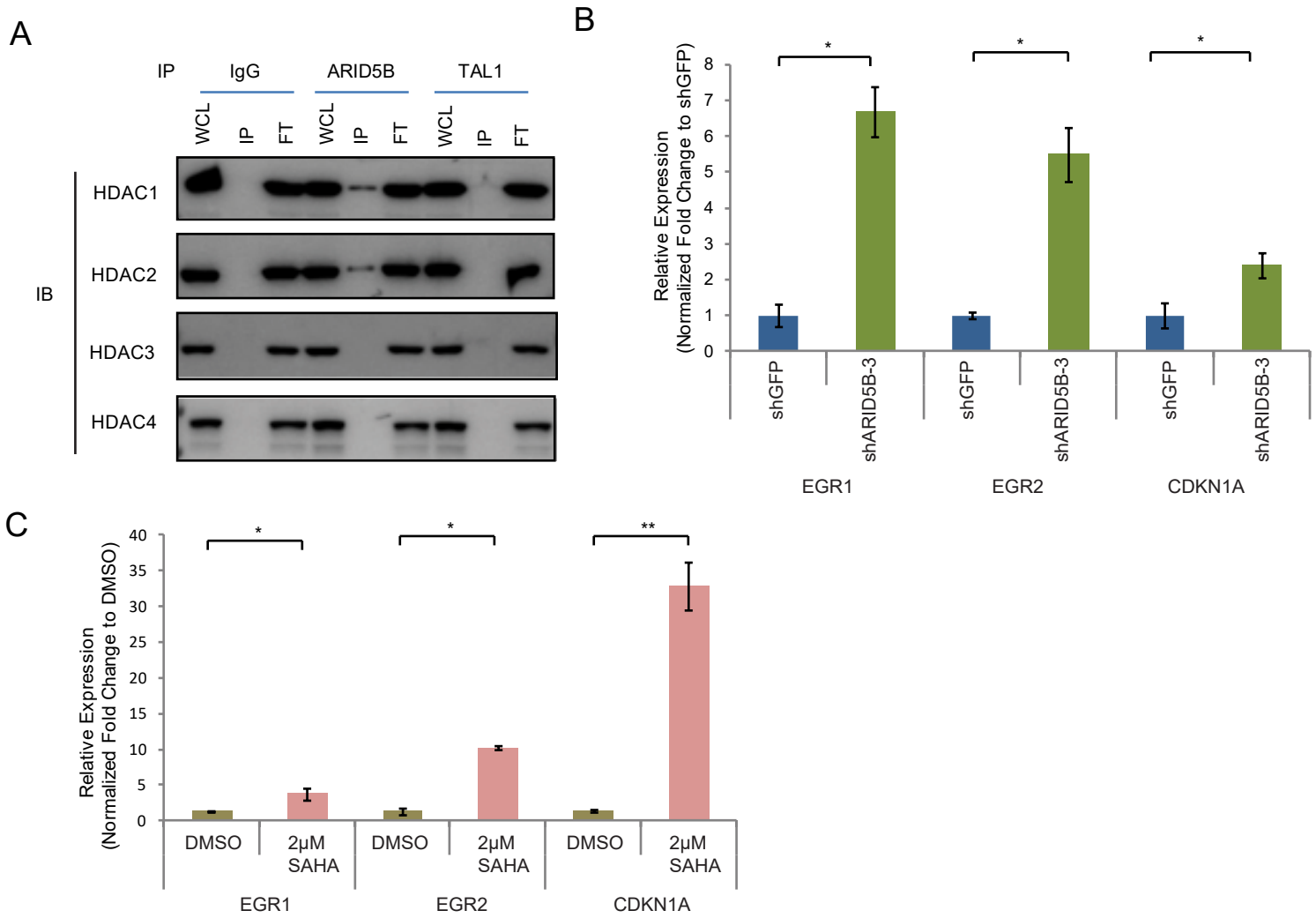
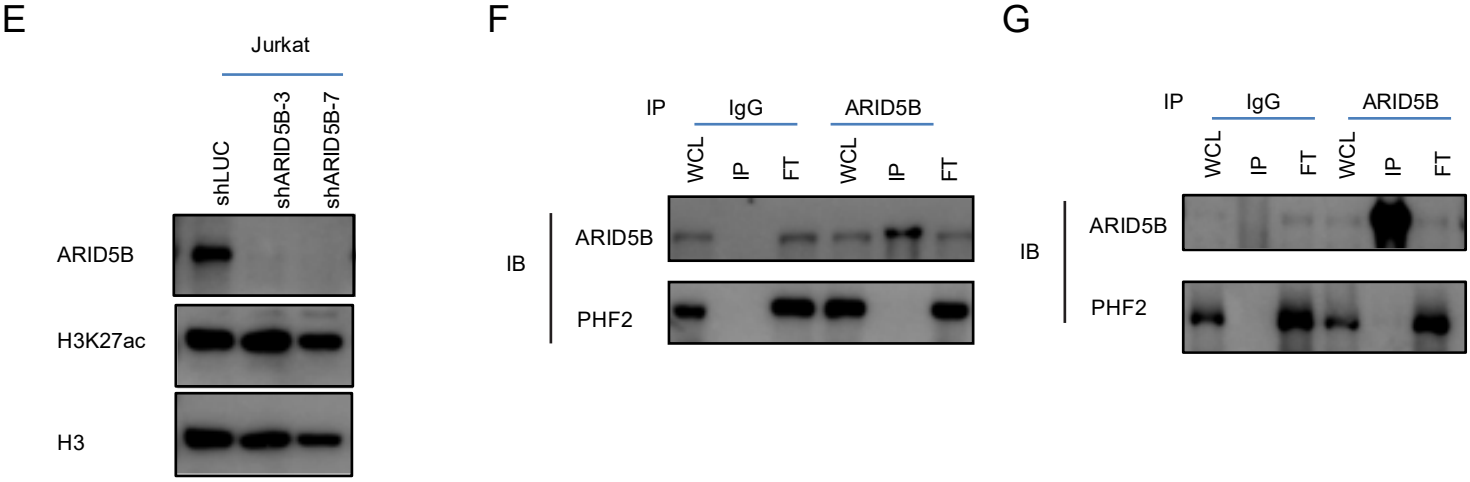


Supplemental Figure 1



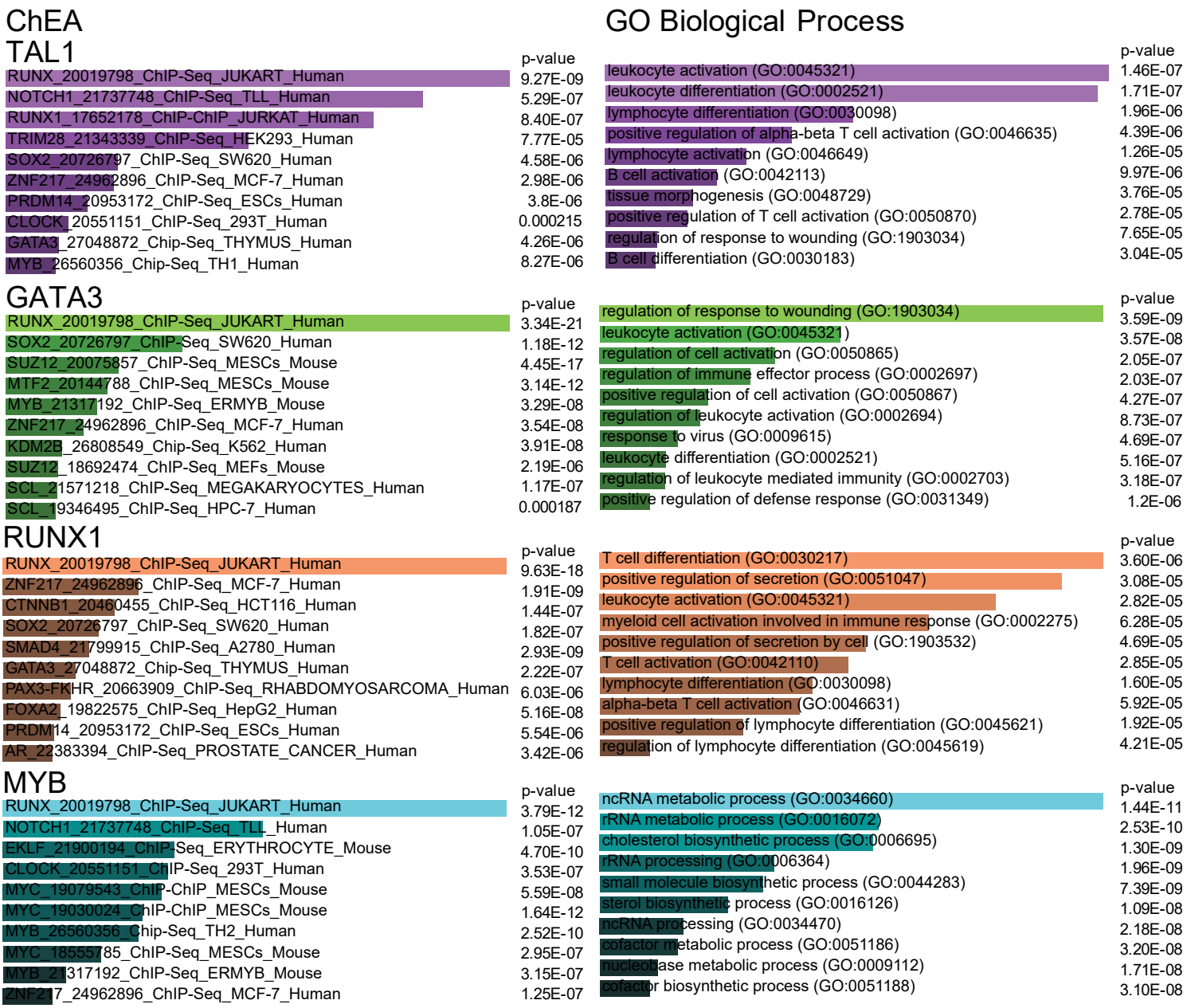
Supplemental Figure 2



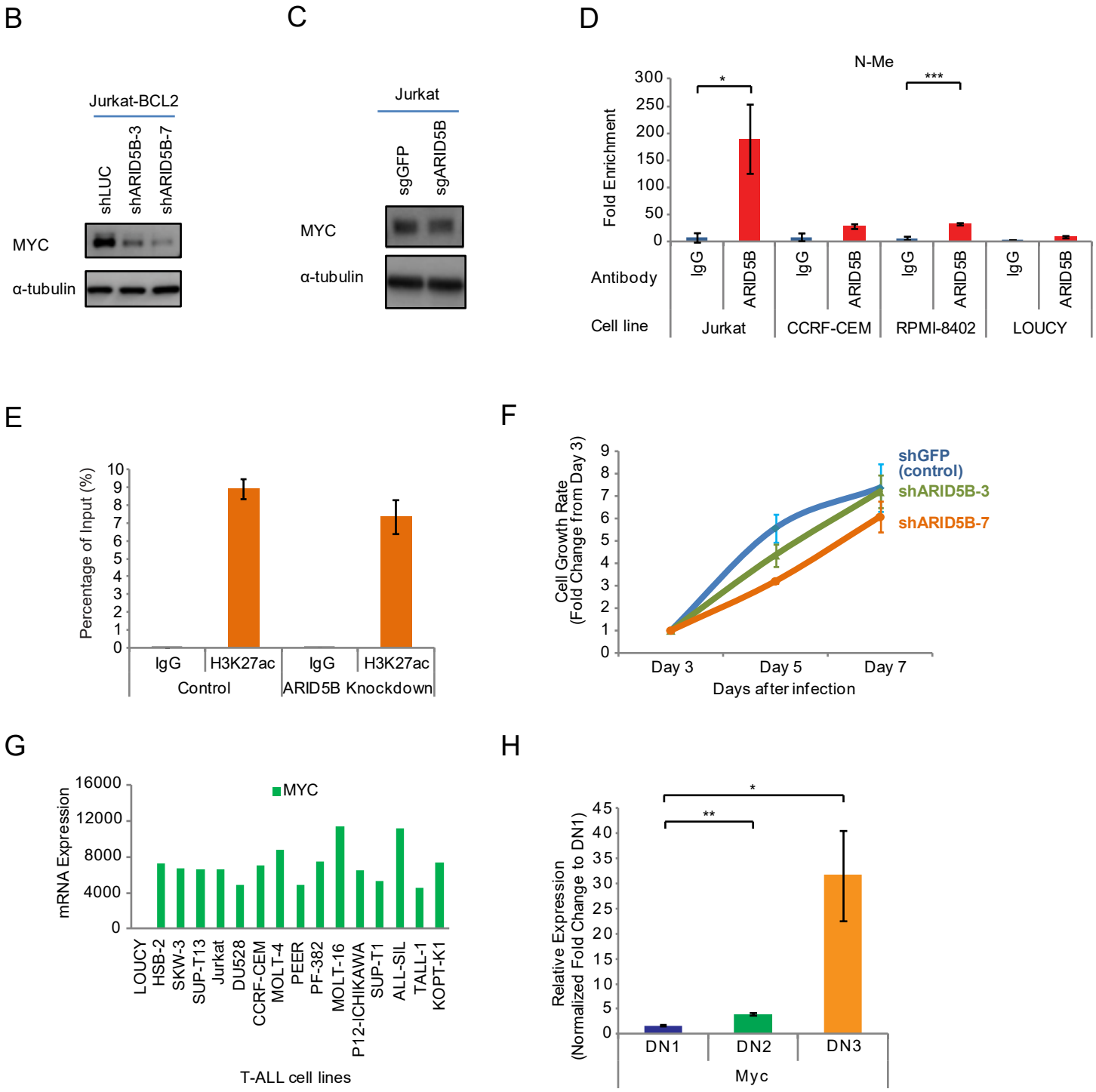


Supplemental Figure 3

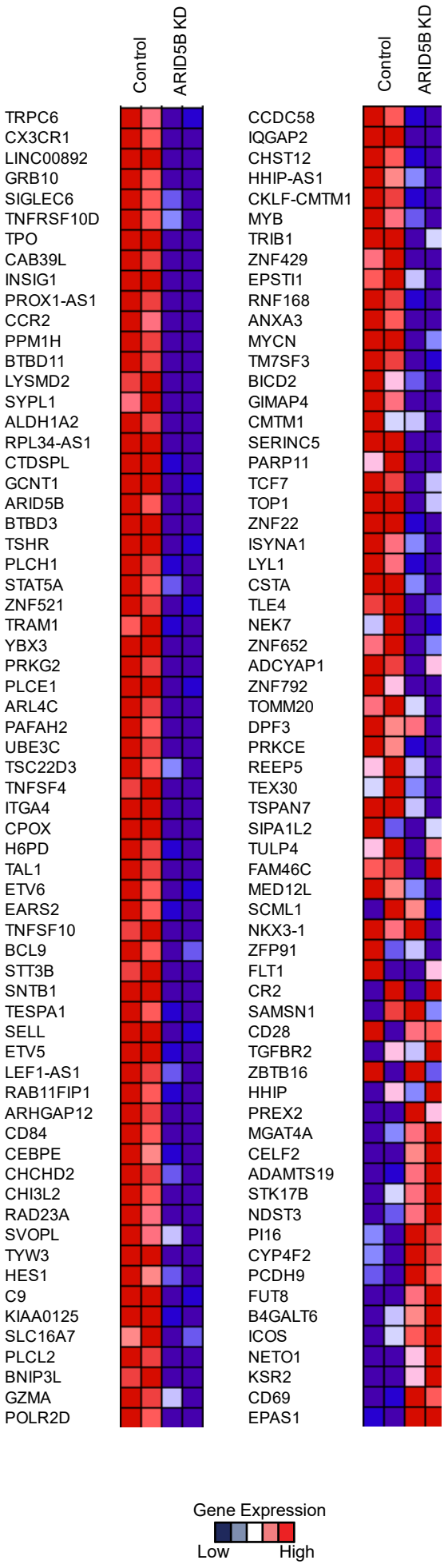
A



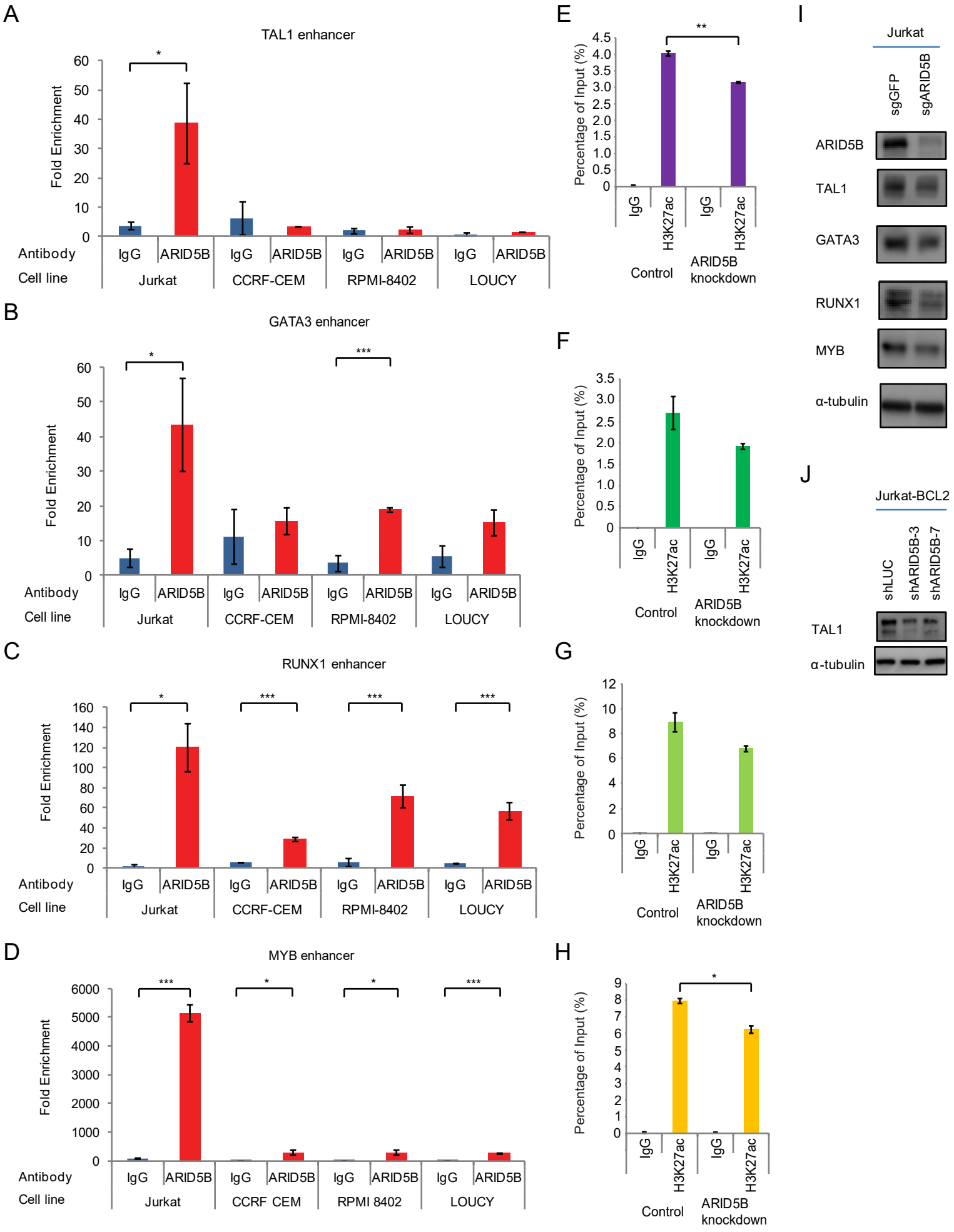
Supplemental Figure 4



Supplemental Figure 4



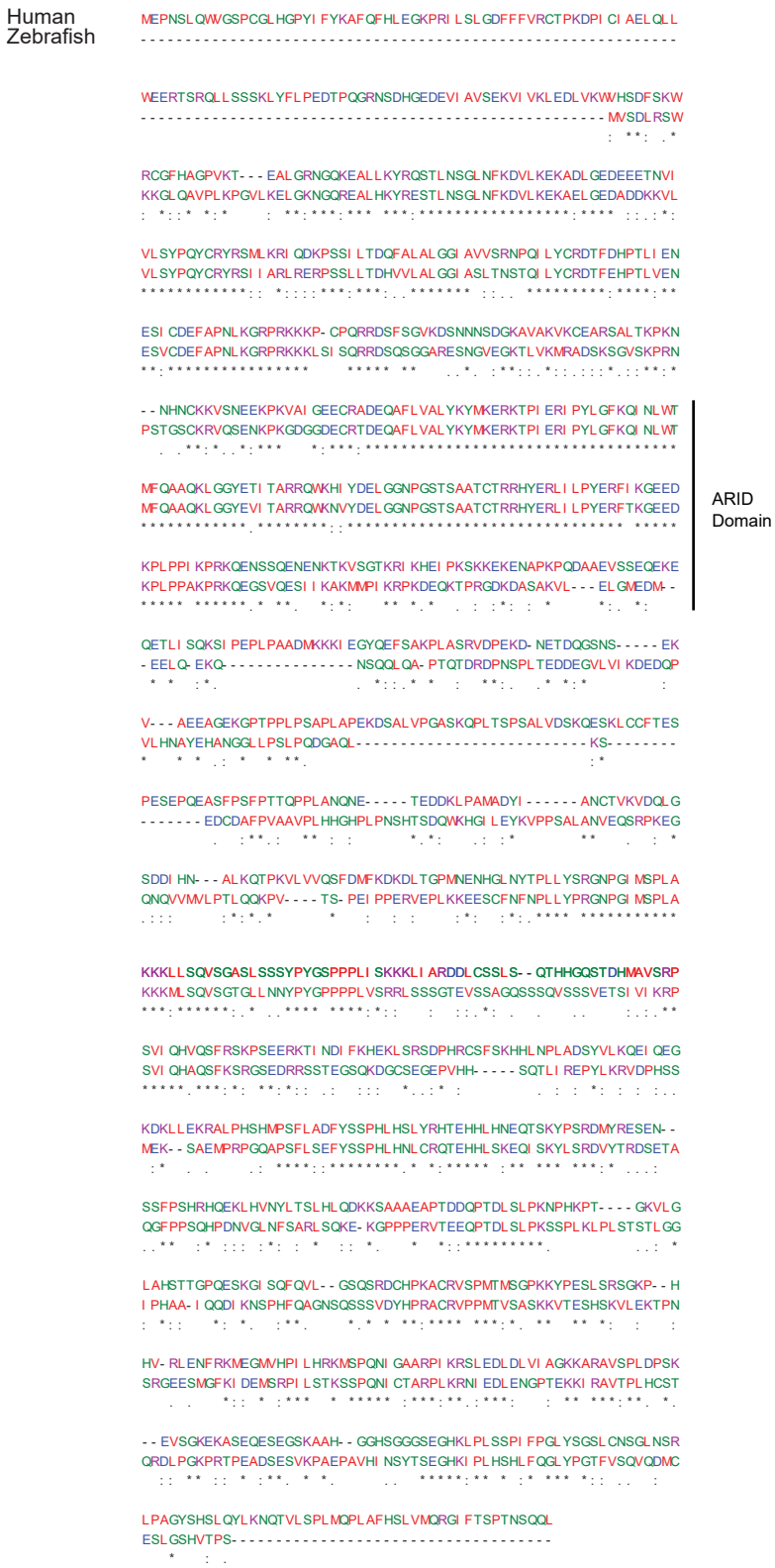
Supplemental Figure 5



Supplemental Figure 6

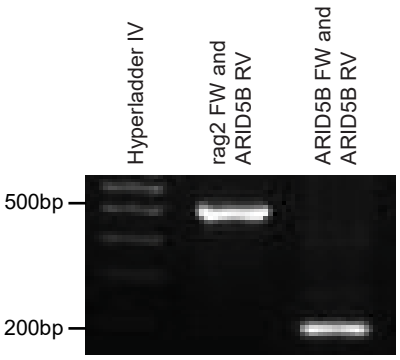
A

CLUSTAL O(1.2.4) multiple sequence alignment



Percent Identity Matrix - created by Clustal2.1
ARID5B protein: 48.69
ARID domain: 88.28

C



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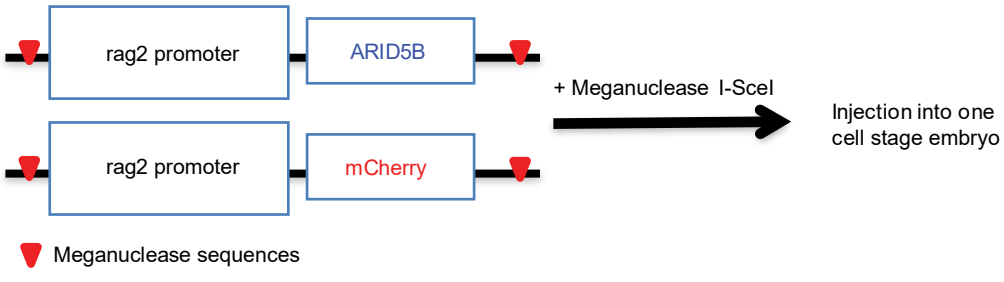

Supplemental Figure 7