Supplementary files

Dynamic chromosomal tuning of a novel *GAU1* lncing driver at chr12p13.32 accelerates tumorigenesis

Peiwei Chai*, Ruobing Jia*, Renbing Jia*, Hui Pan*, Shaoyun Wang*, Hongyan Ni, Huixue Wang, Chuandi Zhou, Yingyun Shi, Shengfang Ge, He Zhang#, Xianqun Fan#

Department of Ophthalmology, Ninth People's Hospital, Shanghai JiaoTong University School of Medicine, Shanghai, P.R. China

*These authors are co-first authors of this report.

#These authors are co-corresponding authors of this report.

Requests for reprints:

Xianqun Fan, Ph.D, Ninth People's Hospital, Shanghai JiaoTong University School of Medicine, Shanghai, 200025, P.R. China, E-mail: fanxq@sjtu.edu.cn;

Present address: He Zhang, Ph.D, School of Life Science and Technology, TongJi University, Shanghai, 200092, P.R. China, E-mail: zhanghe@sjtu.edu.cn;

MATERIALS AND METHODS

Tryptic digestion and peptide desalting for LC-MS/MS

First, 5 mM TCEP was added to each cell sample, which was mixed and incubated for 10 min. After the samples cooled to RT, 10 mM IAA was added followed by incubation in the dark for 15 min. Trypsin was resuspended with resuspension buffer to a concentration of 0.5 µg/µL and incubated at RT for 5 min. A 1-µL volume of trypsin solution was then added to each sample, which was mixed well, centrifuged, and incubated with a Thermo mixer for 8 h. Then, the reaction was quenched with 1% TFA. C18 columns were equilibrated with 200 µL of acetonitrile (ACN). The ACN was then washed out with 200 µL of 0.1% FA 2 times, and the wash-out was discarded. The peptide solution was loaded into the tip of the C18 column and allowed to flow-through the column slowly; the flow-through was collected. The peptide loading step was repeated. Then, the column was washed with 200 µL of 0.1% FA, and the wash-out was discarded. The peptides were eluted with 50 µL of 70% ACN, and the eluate (B) was collected in a new tube. The desalting procedure was repeated (up to the 6th step) using the flow-through (A). The 2 eluates (B) were combined and vacuum dried. The peptides were resuspended with 10 µL of 0.1% FA for LC-MS/MS analysis or stored as a peptide powder at -80°C.

LC-MS/MS

Half of each peptide sample was separated and analyzed with a Nano-HPLC coupled to a Q-Exactive mass spectrometer (Thermo Finnigan). Separation was performed using a reversed-phase column (100 µm, ID × 15 cm, Reprosil-Pur 120 C18-AQ, 1.9 µm, Dr. Math). The mobile phases were H_2O with 0.1% FA and 2% ACN (phase A) and 80% ACN and 0.1% FA (phase B). Separation of the sample was executed with a 120-min gradient at a 300 nL/min flow rate. Gradient B was as follows: 8–35% for 92 min, 35–45% for 20 min, 45-100% for 2 min, 100% for 2 min, 100–2% for 2 min and 2% for 2 min. Data-dependent acquisition was performed in the profile and positive mode with the Orbitrap analyzer at a resolution of 70,000 (200 m/z) and m/z range of 350–1400 for MS1; for MS2, the resolution was set to 17,500 (200 m/z). The automatic gain control (AGC) target was set to 1.0e+06 for MS1 and 1.0e+05 for MS2. The top 10 most intense ions were fragmented by higher energy collisional dissociation (HCD) with a normalized collision energy (NCE) of 28% and isolation window of 2 m/z. The dynamic exclusion time window was 30 s.

MaxQuant database search

Raw MS files were processed with MaxQuant (Version 1.5.6.0). The human protein sequence database (Uniprot_HUMAN_2016_09) was downloaded from UNIPROT. This database and its reverse decoy were then compared by MaxQuant software. The quantification type was MS1; trypsin was set as the specific enzyme with up to 2 miss-cleavages; oxidation [M] and acetyl [protein N-term] were considered as variable modifications, whereas carbamidomethyl [C] was set as a fixed modification; and both peptide and protein FDR should be less than 0.01. Only unmodified unique peptides were used for quantification. iBAQ label-free quantification was also performed with log fit checked.

Figure legend

Supplemental information includes 13 figures and 4 tables.

Supplementary Tables

Supplementary Table 1 Primers, oligos, shRNA, siRNA and sgRNA used in the experiment.

Supplementary Table 2 The clinical characteristics of retinoblastoma patient cohorts.

Supplementary Table 3 The clinical characteristics of neuroblastoma patients.

Supplementary Table 4 The list of lncRNA *GAU1* binding proteins.

Supplementary Figures

Supplementary Figure 1 A novel transcript found at chr12p13.32. (**A**) RT-PCR was performed to measure *GAU1* expression in uveal melanoma, conjunctival melanoma and blastoma cell lines. (**B**) The 5’ and 3’ RACE results in Y79, WERI-Rb1 and SK-N-AS cells. (**C**) The full sequence of *GAU1*. (**D**) Schematic of the RACE result for

GAU1. The first exon is expanded by 57 bp at the 5’ terminus and by 4 bp and a poly-A tail at the 3’ terminus.

Supplementary Figure 2 The epigenetic modifications in the *GAU1/GALNT8* cluster. (**A**) Schematic of the ChIP site of *GAU1/GALNT8* cluster. (**B-D**) ChIP assay was performed to estimate histone methylation (H3K4me1, H3K4me3, H3K9me3, H3K36me3), acetylation (H3K9Ac, H3K27Ac) in *GAU1/GALNT8* cluster. Epigenetic screening of histone modification and chromatin organization was performed in *GALNT8* (**B**), *GAU1* (**C**) and distal region (D). All the experiments were performed in triplicate and are presented as mean ± SEM. *p < 0.05, **p < 0.01.

Supplementary Figure 3 *GAU1* is a long non-coding RNA. (**A**) Predicted ORFs of GAU1, the blue and gray box shows the predicted ORFs. **(B)** Predicted coding potential of GAU1. **(C)** GALNT8, GAU1(predicted ORF-1 and ORF-2), and lnc-HOTAIR (predicted ORF) were cloned, respectively, into pcDNA3.1(+) with C-terminal EGFP-tag. **(D)** IF analysis of the EGFP expression in SK-N-AS cells transfected with the indicated constructs. Scale bars,100μm. **(E)** Western blot analysis of the recombinant protein by anti-EGFP tag. (**F**) Diagram of the GFP fusion constructs used for transfection. The start codon ATGGTG of the GFP (GFPwt) gene is mutated to ATTGTT (GFPmut). (**G-H**) The indicated constructs were transfected into SK-N-AS cells for 24 hr, (**G**) GFP fluorescence and the GFP fusion protein levels were determined by western blotting with (**H**) anti-GFP antibodies.

Supplementary Figure 4 The promoter region of *GAU1* and *GALNT8*. (**A**)

Schematic of the luciferase assay of the *GAU1/GALNT8* cluster. (**B**) The first exon of *GAU1* and *GALNT8* presents luciferase activity in opposite directions. The full-length 5' to 3' sequence and its opposite sequence were used as a positive control (line a, b). The untreated group and empty vector group served as negative control groups. The first exon of *GLANT8* presented the same transcription direction as *GAU1* (line g, h), and the first exon of *GAU1* exhibited the same transcription direction as *GLANT8* (line c, d). The gap presented neither forward nor reverse transcriptional activity (line e, f). (**C**) Schematic of the dCas9-KRAB target of the *GAU1*/*GALNT8* cluster. (**D**) Real-time PCR was performed to validate the knockdown efficiency in Y79, WERI-Rb1 and SK-N-AS cells. (**E**) Western blot assay was used to examine *GALNT8* knockdown efficiency by two different shRNA.

Supplementary Figure 5 The ***GAU1* or *GALNT8* overexpression in normal cells.** (**A**) Realtime-PCR was performed to test GAU1 expression after transfecting pcDNA3.1-*GAU1* in RPE1 and ARPE19. (**B**) Western blot was performed to examine GALNT8 expression after transfecting pcDNA3.1-*GALNT8* in RPE1 and ARPE19. (C) CCK8 assay was performed to measure proliferative rate after *GAU1* or *GALNT8* overexpression in normal cells. (**D**) A colony formation assay was performed to assess normal cell (ARPE19 and RPE1) growth with *GAU1* and *GALNT8* over-expressing. Quantification of visible colonies. The colony number of the pcDNA3.1 group was set to 1. (**E, F**) A CCK8 assay was performed to assess normal cell (ARPE19 and RPE1) growth with *GAU1* and *GALNT8* over-expressing. All the experiments were performed in triplicate, and the relative colony formation rates are shown as mean ± SEM. *p <

0.05. pcDNA3.1: negative control group transfected with empty pcDNA3.1 vector.

Supplementary Figure 6 The *GAU1* or *GALNT8* overexpression in Y79, WERI-RB1 and SK-N-AS. (**A**) Realtime-PCR was performed to test *GAU1* expression after transfecting pcDNA3.1-*GAU1* in Y79, WERI-RB1, and SK-N-AS. (**B**) Western blot was performed to examine GALNT8 expression after transfecting pcDNA3.1-*GALNT8* in RPE1 and ARPE19. **(C)** CCK8 assay was performed to measure proliferative rate after *GAU1* or *GALNT8* overexpression in tumor cells. (**D**) A soft-agar tumor formation assay was performed to determine the colony formation ability of *GAU1* or *GALNT8* overexpressed tumor cells (Y79, WERI-Rb1 and SK-N-AS). (**E**) Quantification of visible colonies. The colony number in the empty vector group was set as 100%. All the experiments were performed in triplicate, and the relative colony formation rates are shown as the mean ± SEM. *P<0.05. pcDNA3.1: negative control group transfected with empty pcDNA3.1 vector.

Supplementary Figure 7 *GAU1* siRNA interference efficiency by the three siRNA sequences in tumor cells. (A-C) Real-time PCR was performed to validate the siRNA knockdown efficiency in Y79, WERI-Rb1 and SK-N-AS cells. The control group was set to 1. The 2nd and the 3rd siRNA sequence presented significant knockdown efficiency. Con: no siRNA was transfected. NC siRNA: transfected with controlled scramble siRNA. *P<0.05.

Supplementary Figure 8 Localization of *GAU1* expression. (A) RT-PCR analysis of the nuclear fraction. *GAU1* was mainly expressed in the nucleus. *GAPDH* and *U2* RNA served as positive controls for the cytoplasmic and nuclear fractions, respectively. (**B, C**) RT-PCR analysis of *GAU1* and *GALNT8* expression after knocking down *GAU1* in tumor cell lines by sgRNAs (**B**) or siRNAs (**C**). Empty vector: negative control group transfected with the empty dCas9-KRAB vector. sgRNA1 and sgRNA2: the first and second sequences used with the dCas9-KRAB system to knock out *GAU1*. *P<0.05. (**D**) RT-PCR analysis of *GAU1* and *GALNT8* expression after overexpression *GAU1* in normal cell lines. *P<0.05, **P<0.01.

Supplementary Figure 9 Overexpression of *GAU1* promoted GALNT8 in tumor cells. (**A, B**) RT-PCR (**A**) and real-time PCR (**B**) confirmed that *GAU1* was overexpressed after transfecting with pcDNA3.1-*GAU1* plasmid, while *GAU1* remained unchanged after transfecting with pcDNA3.1-*GALNT8* in tumor cells. (**C**) Western blot showed that GALNT8 was upregulated after overexpression of *GAU1* in tumor cells. pcDNA3.1-*GALNT8* was conducted as positive control. (**D**) RT-PCR analysis of *GAU1* and *GALNT8* expression after overexpression *GAU1* in tumor cell lines. *P<0.05.

Supplementary Figure 10 ChIRP analysis of *GAU1*-interacting proteins. (**A**) The protein peptides isolated by ChIRP. The lncRNA *U2* was selected as the normal control, and scrambled oligos were selected as negative controls. (**B**) RNA-ChIP analysis of the binding of *GAU1* with MCM2, TCEA1, RBMX, and CBX3. IgG antibody and *U2* RNA was used as negative controls.

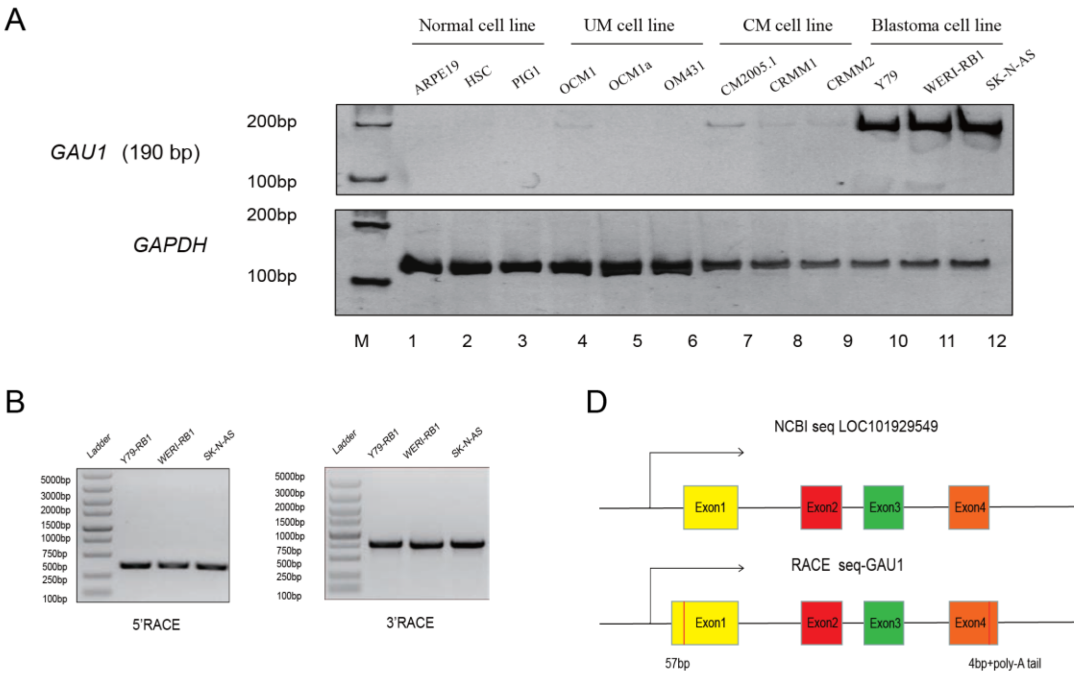
Supplementary Figure 11 TCEA1 is recruited to the *GALNT8* promoter. (**A**)

Schematic of sites in the *GALNT8* promoter as detected using the ChIP assay. (**B, C**) RT-PCR examination of the binding of *TCEA1* to the *GALNT 8* promoter using samples from the ChIP assay.

Supplementary Figure 12 TCEA1 binds to *GAU1* promoter in tumor cells. (A) Schematic of sites in the *GALNT8* and *GAU1* promoter in the ChIP assay. **(B-E)** TCEA1 binds to *GAU1* promoter in tumor. **(B)** *GAU1* promoter was selected as target loci and a distal site was choosing as negative site. Non-selective antibody IgG was selected as negative control and the total cellular DNA (input) was referred to positive control. RT-PCR examination of the interaction between TCEA1 and *GAU1* promoter in the ChIP assay. **(C)** Quantification of the binding of TCEA1 to the *GAU1/GALNT8* cluster in ChIP assay. **(D-E)** Negative site presented no TCEA1 binding. The value obtained for IgG group cells was set to 1. All the experiments were performed in triplicate and are presented as mean ± SEM. *P<0.05, **p < 0.01.

Supplementary Figure 13 Global TCEA1 expression analysis. (**A**) Global expression levels of TCEA1 in tumor cell lines (Y79, WERI-Rb1, SK-N-AS) and normal cell lines (HSC, ARPE19) were measured by Western blot. (**B**) Global expression levels of TCEA1 were measured by Western blot in *GAU1* knockdown tumor cells.

Supplementary Figure 1

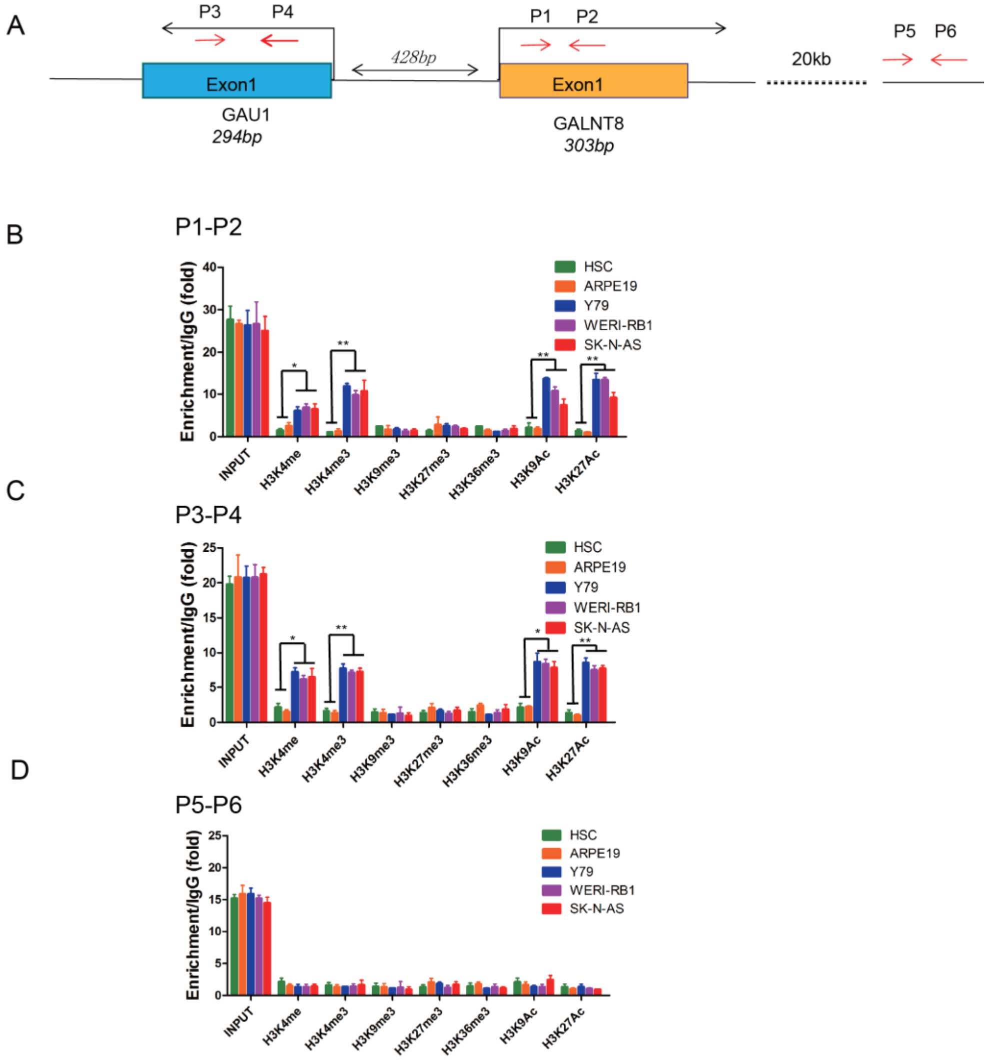


C

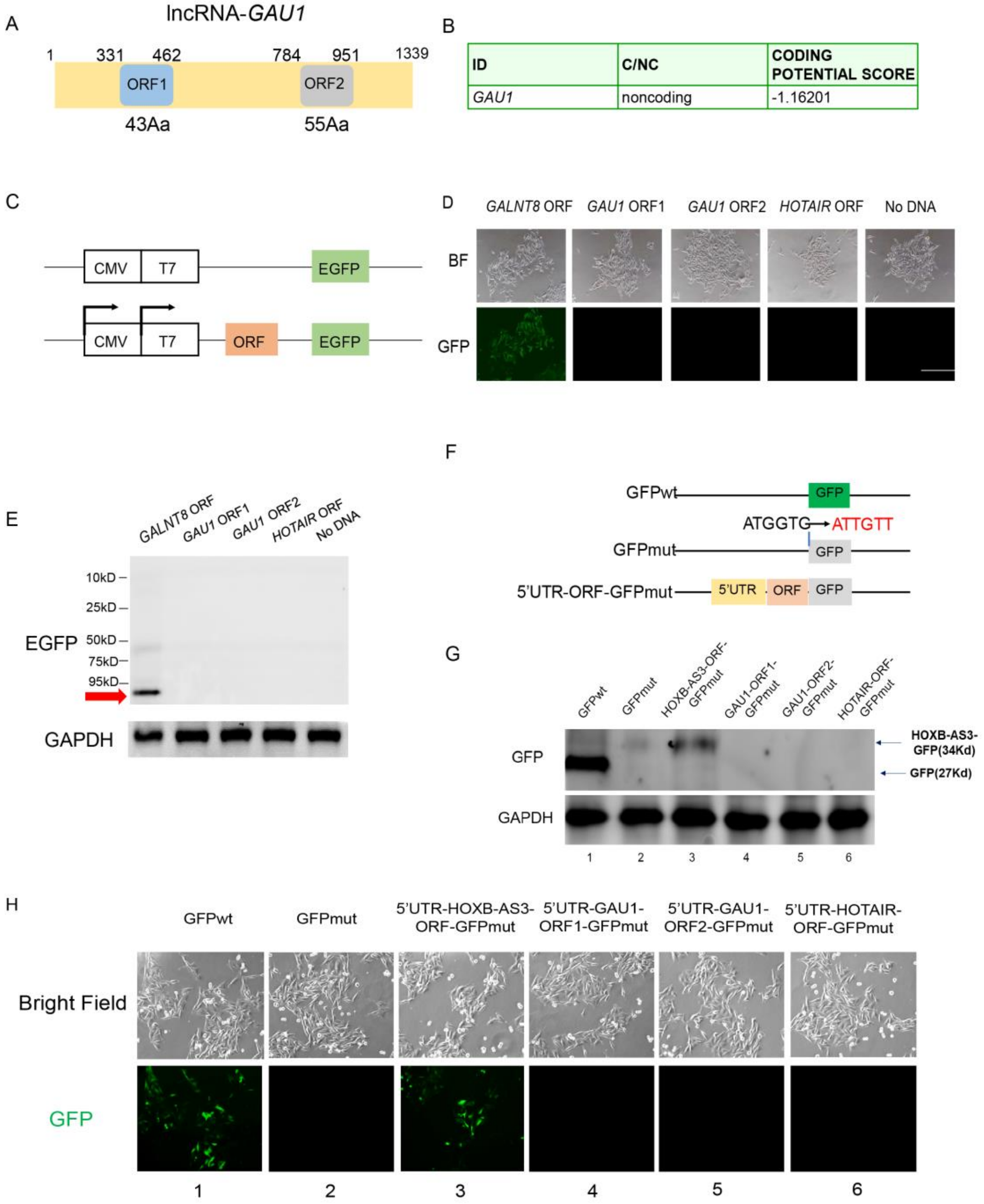
GAU1 Full length Sequence:

GTGGAGCCCAGAGCTCTGGGCCGTGAGCAAGCCTCCAACGCTCTGGTCCCCTGG
ACCTGCCTTTTAAACGCTTGTTCTGTCTCTTTCTAACTCCTTTGTCTCCGCCGGACT
CGGGGTATCCGCTGGGTGGTGTGGGGCTGGTTTCCCCAACAGCTTTCTCTCCACC
CAGCTTCATAGATGACACCCTGCCTGCTAGGCCAGATGTTCTCCGGCCAAGAACA
CTATTATAGCTGCCTGGCTTCTCCCTGAGCAGCGTCAGGTGTGAGGAGTGCGCAT
GGCAGCTGCCCAGTGACAGATGGAATCTCCCTCTCTCACCCAGGCTGGAGTGCAA
TGGCACAATCAGCTCACTGCAACCTCCACCTCCCGGGTTCAAGTGATTCTTCTGCC
TCAGCCTCCCGAGTAGCTGGGACTACAGCCTTCATTTCAGTCAACAACCACATAAC
AGATCATCAAAATACATAAAGCAAAACTGACAGAACTAAAGTTAAATATACATATCT
ACAATTATGGAGAGAATAACAAACAAAAAAATAGAACAAAAATGTTCCACGCCTTG
GTCAATGGTACCACCAGCCAGCGAGGTTCCCAAGCCAAGAACTTCGGAAGCATCCG
TGATTCTTCATCTTCCACATCCACTTTATCACCGGAAGCACGCATTCAACTTTCTAAA
CATCCCTCAGACTGACCCACATCTTTCTATCCACTTTTCACATCTTGCTTGAATGACT
TCCCCAGGCCCAGCCAGTCTCCCGCCTCCAGCTCCGCCCCCTTCTGACTCCCTTTAT
GTGGTACAATCAGAGAGCCCTTCCCAAAGCACAAATCTGCTTTCACTGCTTTGCTTC
AAGCCCTTTCGTGGCTCCTATTTCCAGCTGGATGAGGTCCAAGCCTCTTAACGTGCT
GTACGTGTACATGCCTGACCCCCGCTACTTGACCAGCCAAGTTTCTTCATAGCGCTG
TGTCTTTGTTCCTCCTGCCTAGAATGTCCTTCCCCTTTTCTCTCGCTTCATGGTGTGC
CAAACTCCTATCCTGTGAGATCCAGCTTACACGTCAGCTTCCCCGTGATGCTTTTCC
TAACTCCCTATTCAGCCAGTGAAGTGCTCCCTGCGTTCCCATGTGGCTTGTATCTCT
GTAAAAGCTCCACAGAGCTGTTTTAATTTATCCGTTAATGTACATGTCTTCCCCACTA
GACTTTTGCTCCTTAAAGTATCTCCTTCATCTCTGCTTTGCATAACCTAACAGAGTT
CAACACATAGGAGTGTTTTAATAAATGCTGATTAAAATAATTAATAAACAAATGAAA
CAATACAGCAAAAAAAAAAAAAATAAAAAAAAAAAAAAAA

Supplementary Figure 2

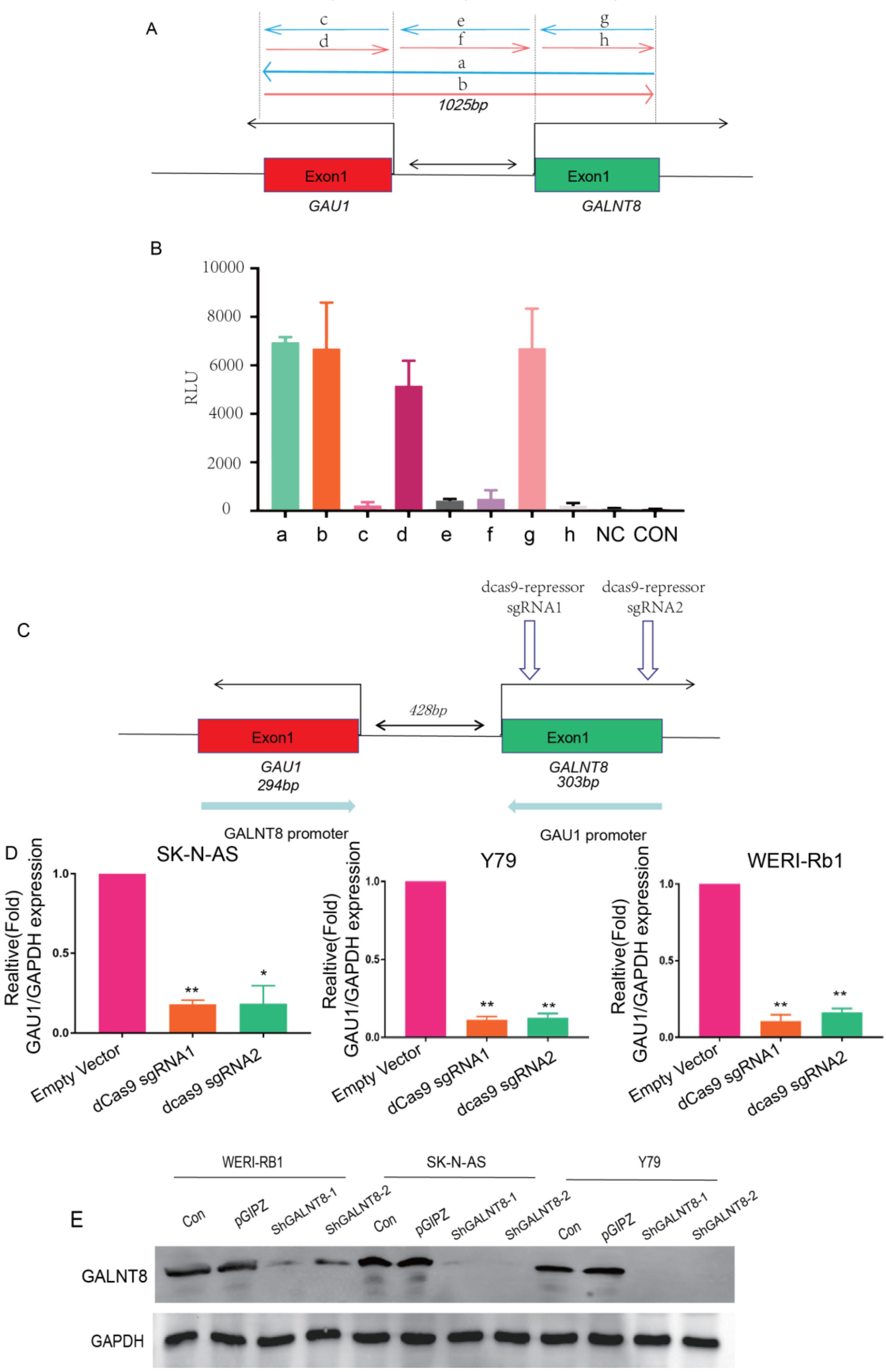


Supplementary Figure 3

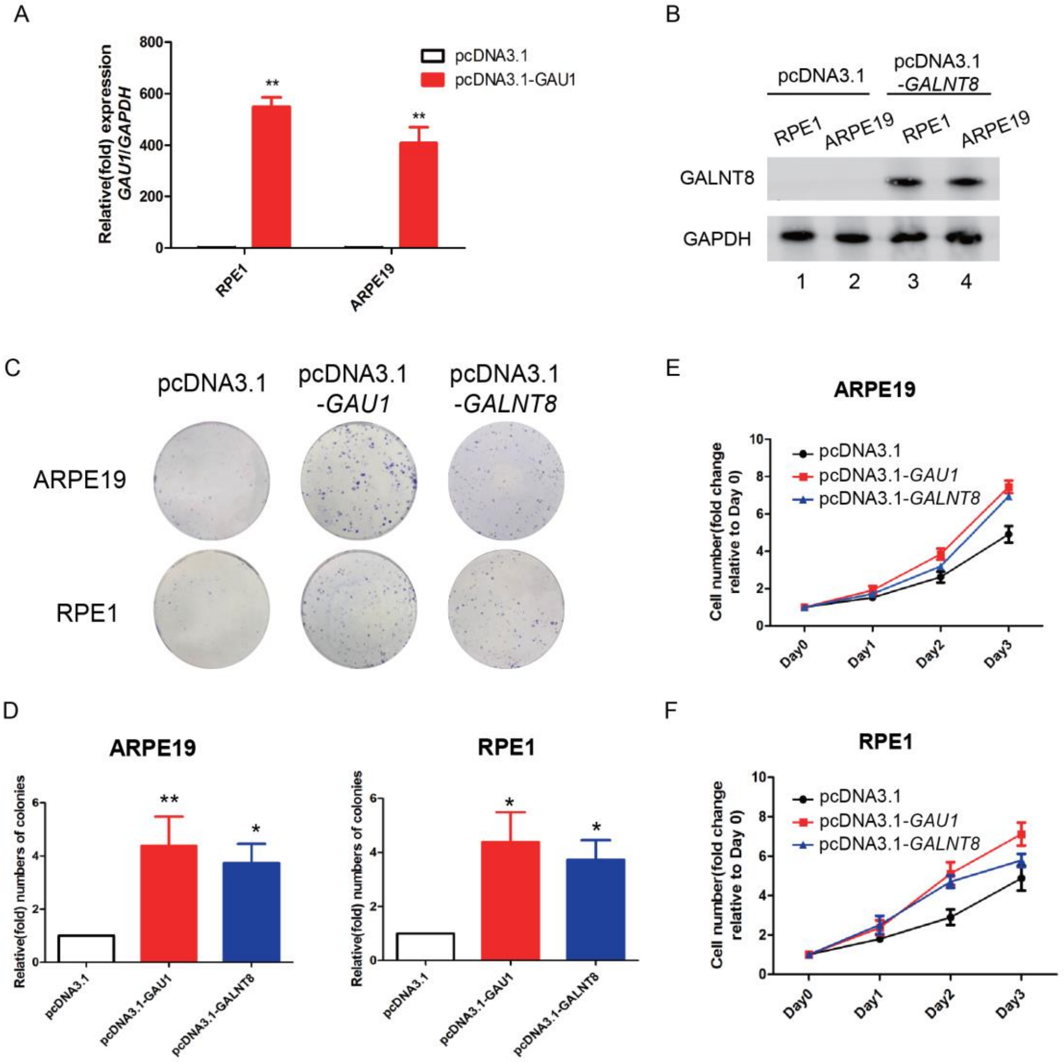


ID	C/NC	CODING POTENTIAL SCORE
GAU1	noncoding	-1.16201

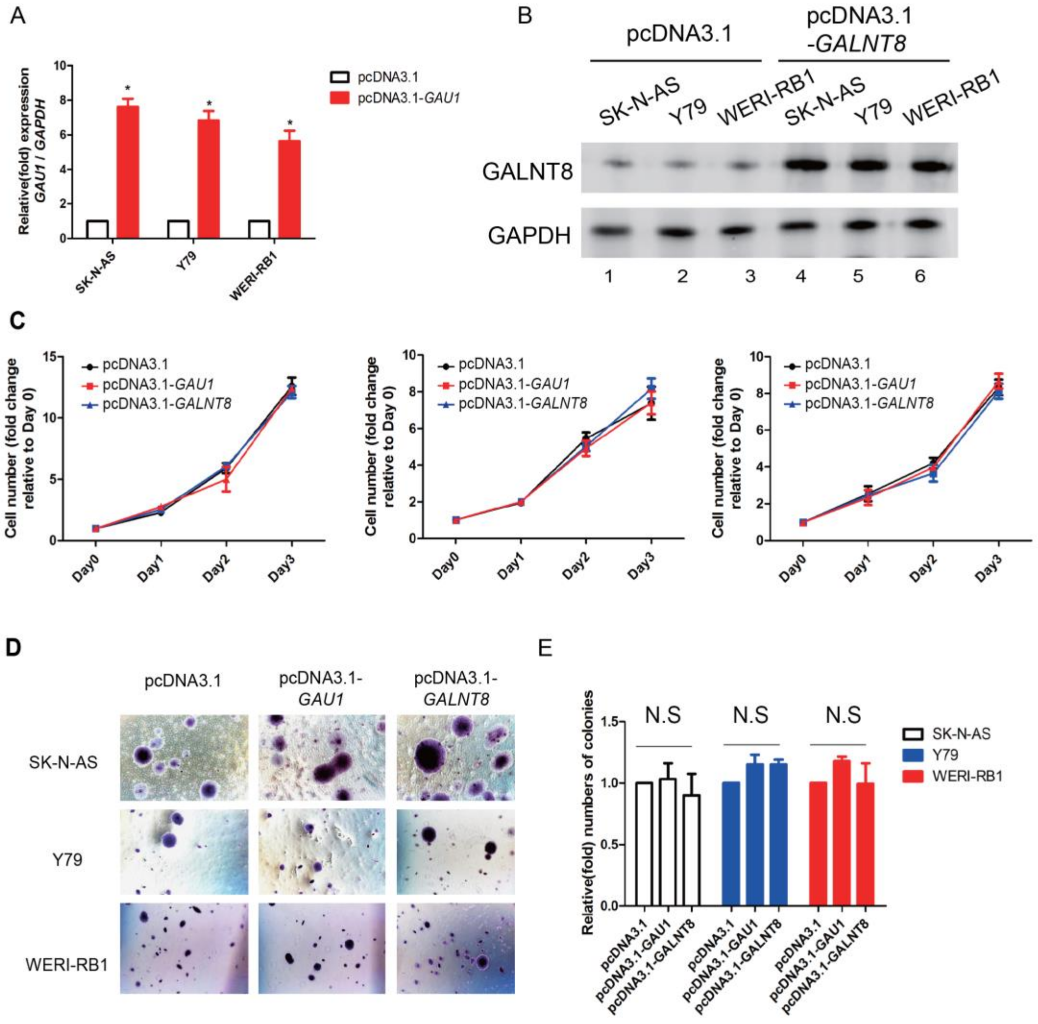
Supplementary Figure 4



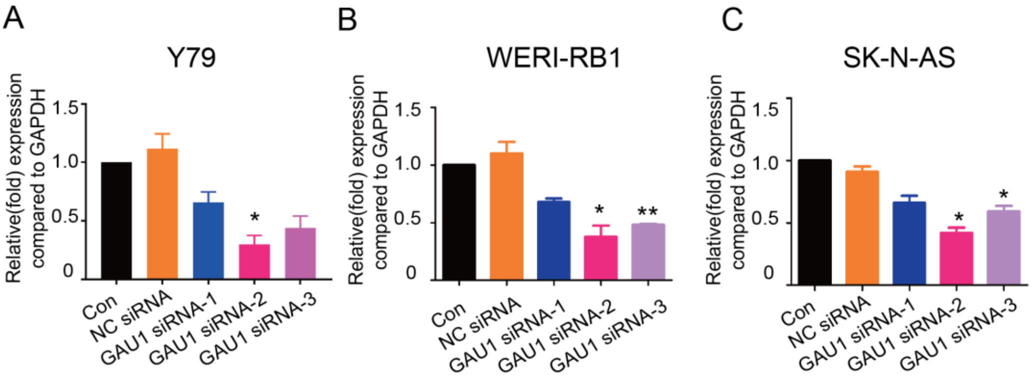
Supplementary Figure 5



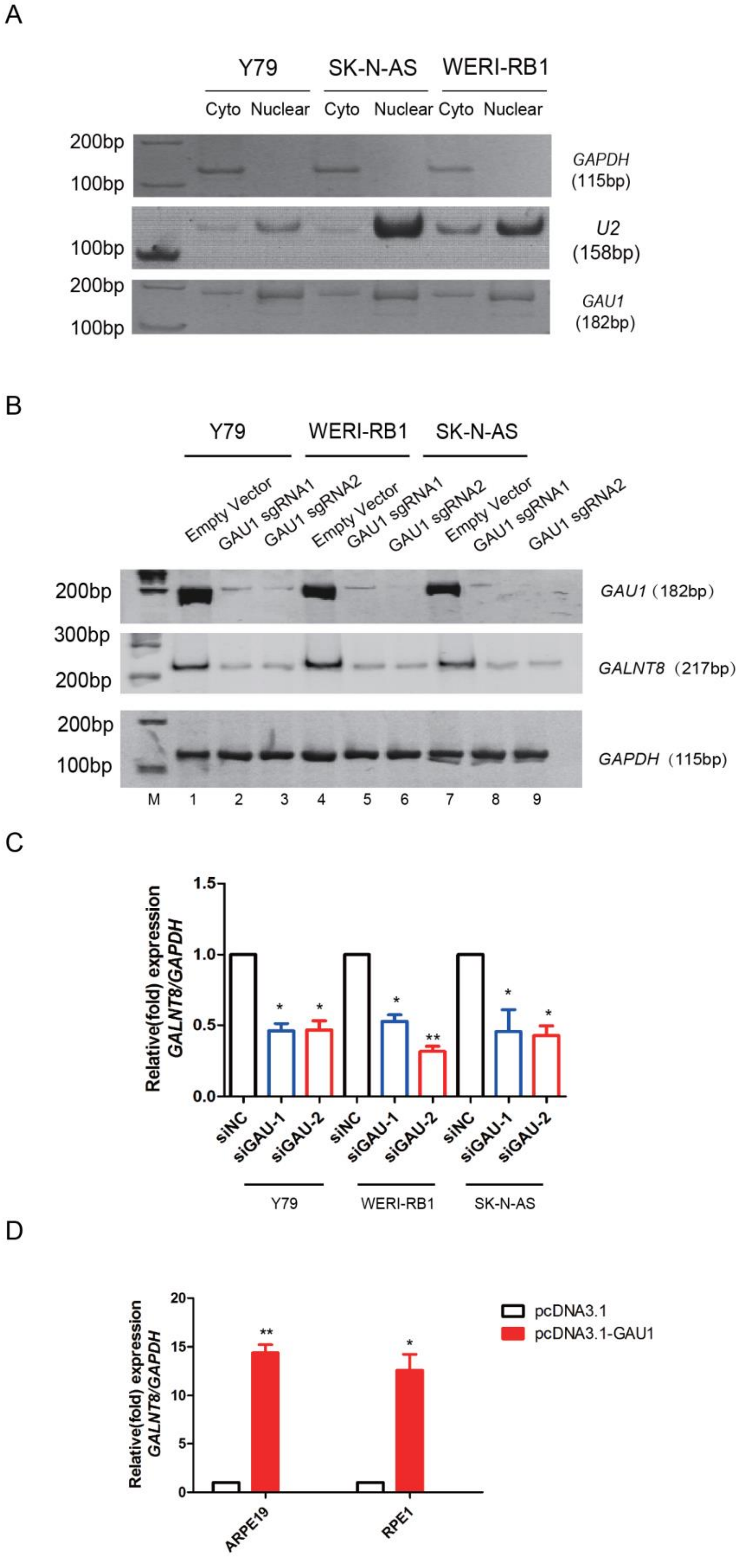
Supplementary Figure 6



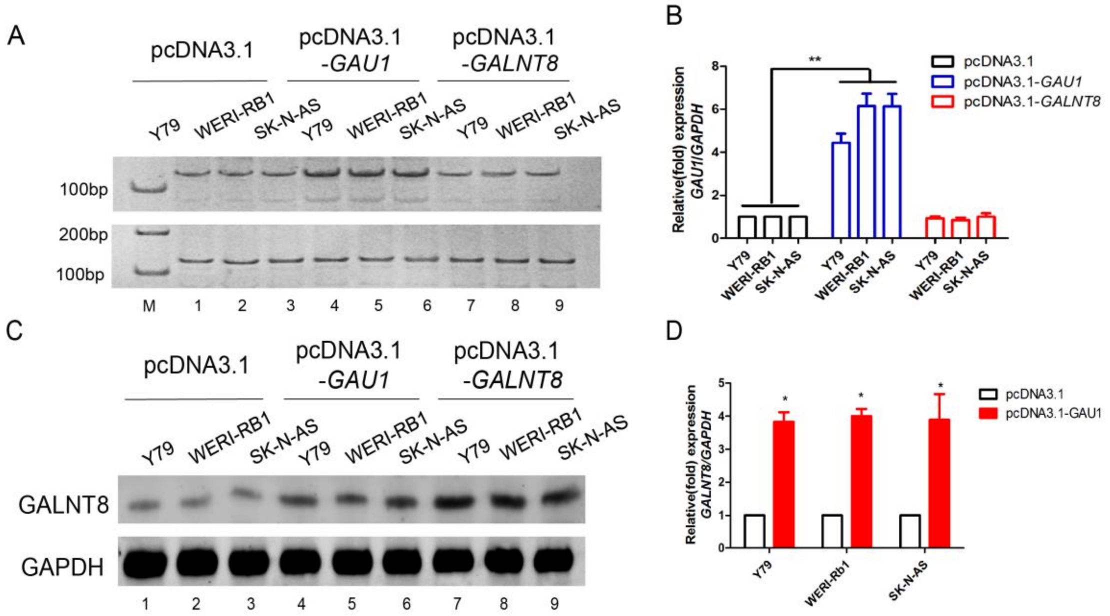
Supplementary Figure 7



Supplementary Figure 8

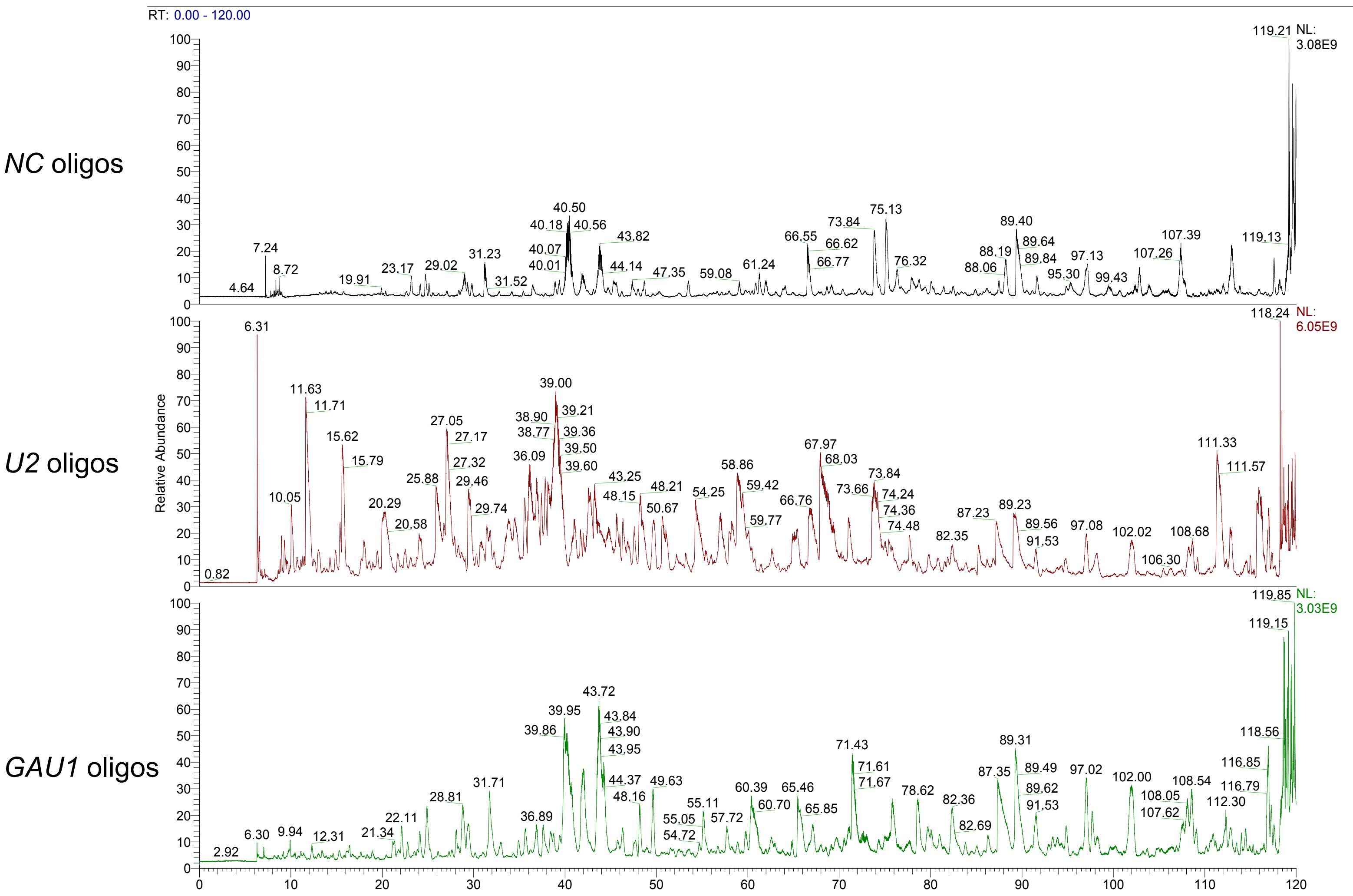


Supplementary Figure 9

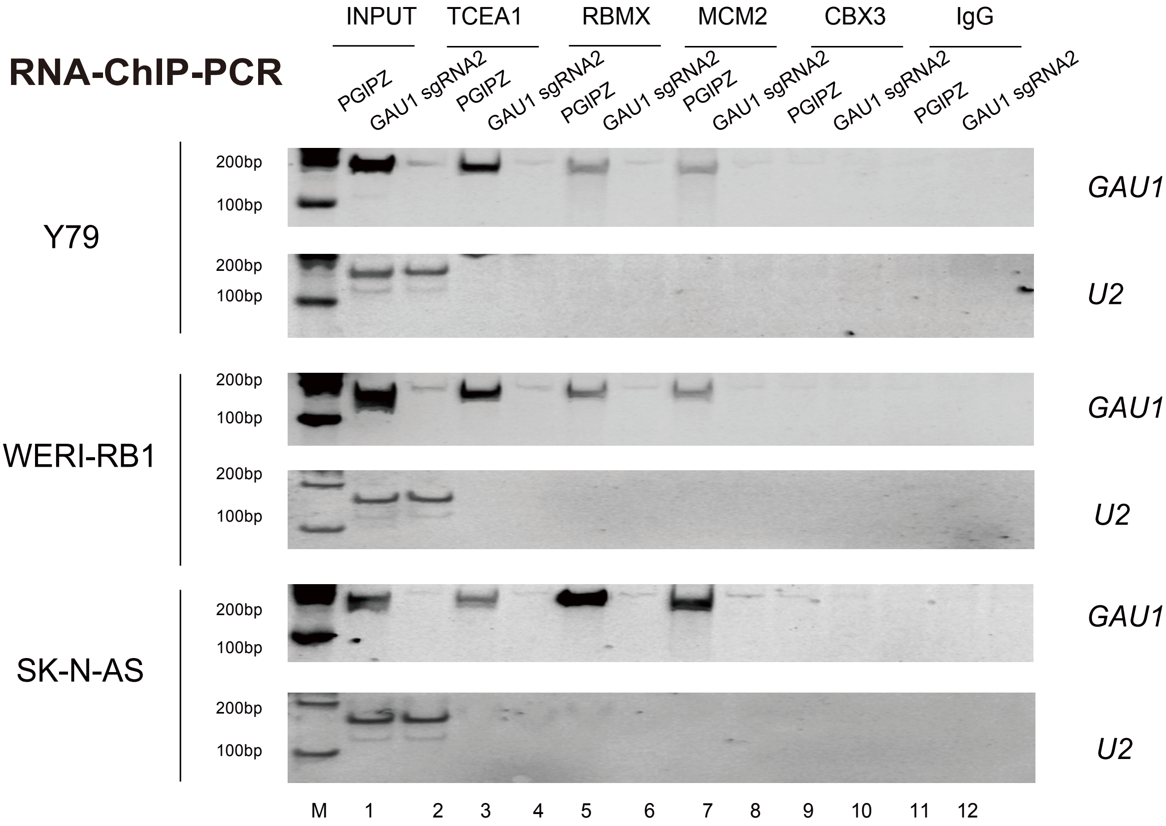


Supplementary Figure 10

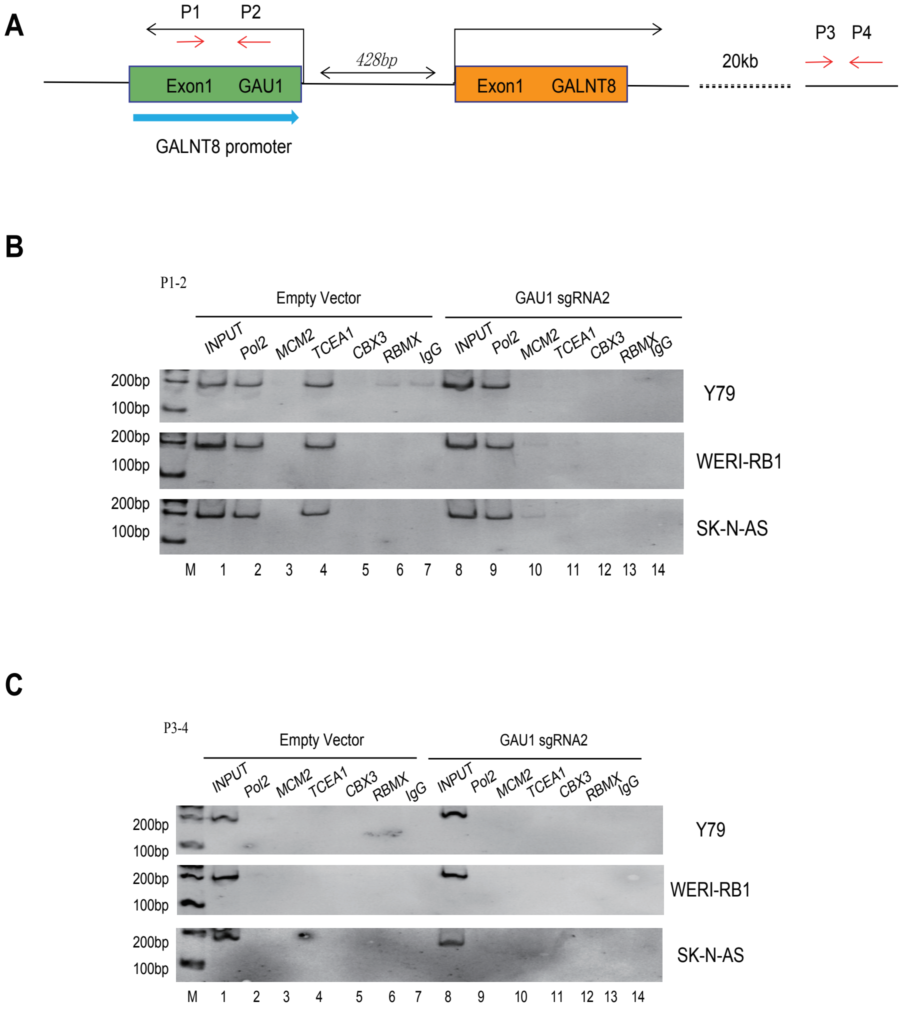
A



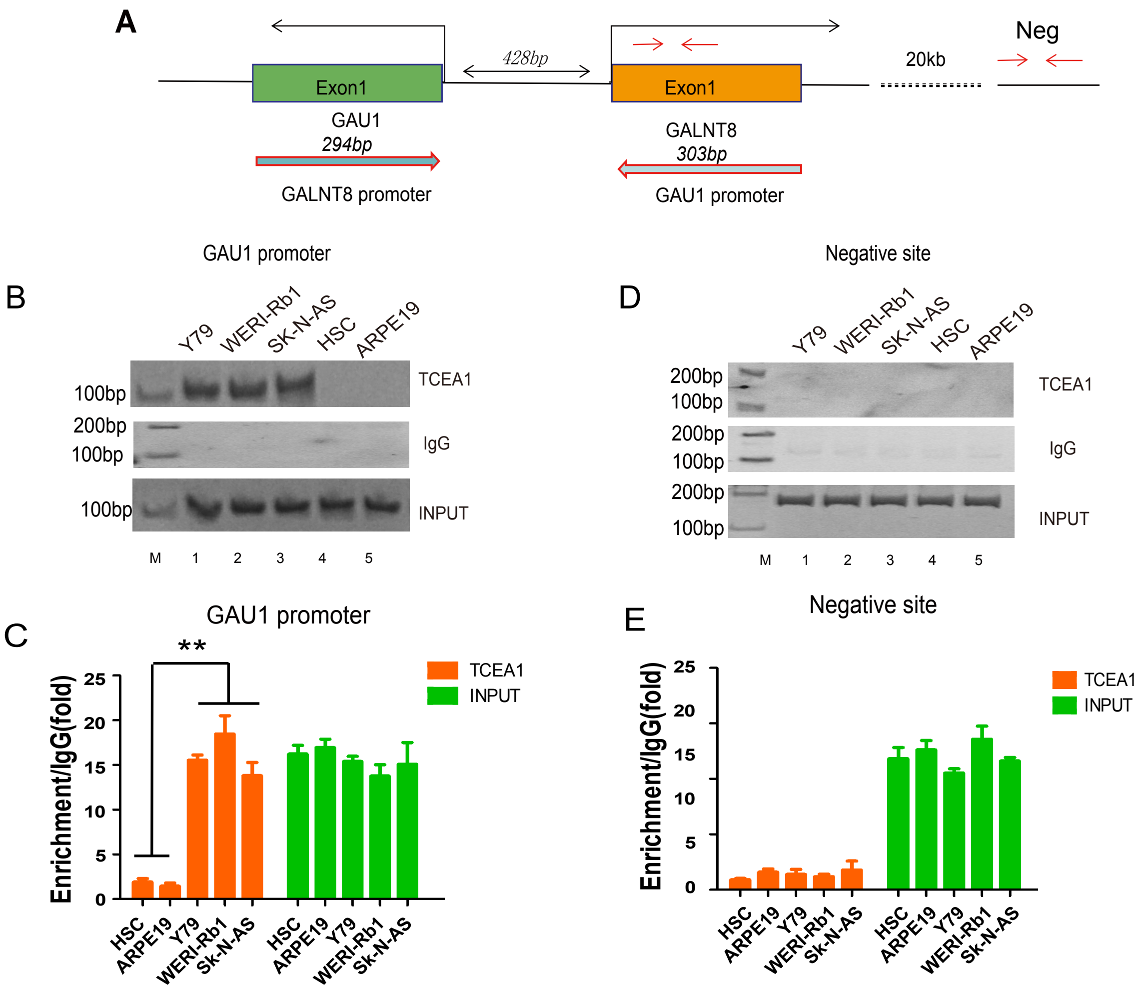
B



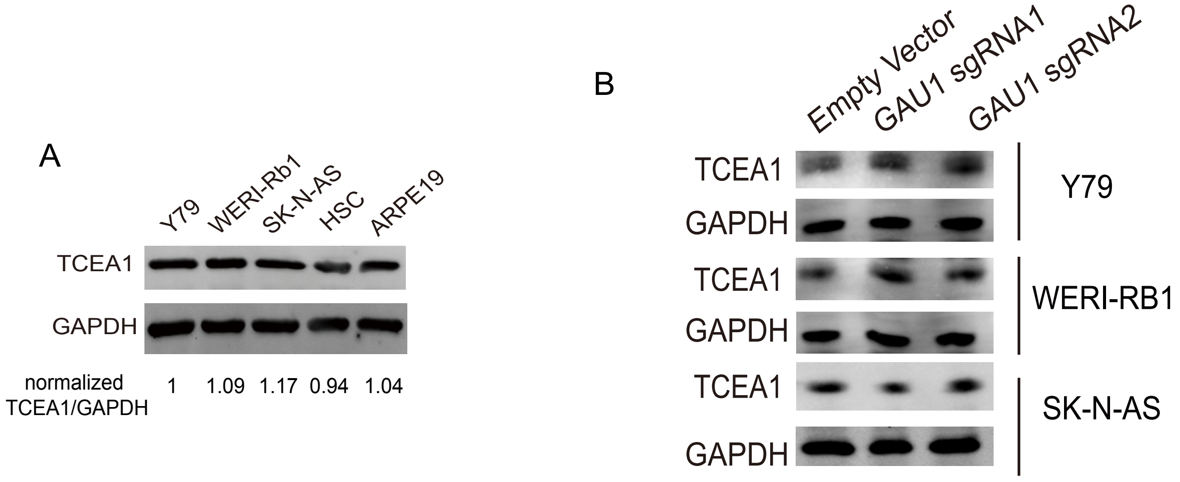
Supplementary Figure 11



Supplementary Figure 12



Supplementary Figure 13



Supplemental Table 1: Primers, oligos, shRNAs, siRNAs and sgRNAs used in the experiment.

PCR	
GAU1 primer set1	TATCACCGGAAGCACGCATT TTTGTGCTTTGGGAAGGGCT
GAU1-2 primer set2	AGCCCTTCCCAAAGCACAAA AGAGGCTTGGACCTCATCCA
GALNT8	TCCCACAAAAGCTAGGCTGG GTGGAGACCACCCGTAAACA
GALNT8-FAIRE	TCCCACAAAAGCTAGGCTGG CATCTTCTTCCTGGGGGTCG
GAU1-FAIRE	CTGACCCCCGCTACTTGAC TACAAGCCACATGGGAACGC
KCNA6-FAIRE	GGCTCCTGTCATCCTCTTCG ACACTGCGGACTCTGTTCCA
DYRK4-FAIRE	TACGACGTGGCCATTGACAT GCTGCTCCACCTCATTCTCC
AKAP3-FAIRE	AGGAGGTTCATTGGCAGGAA CGAGGACCTGATCGTGTCTG
GLANT8 promoter-ChIP	TAGATGACACCCTGCCTGCTA CAAGCATTCTTCTGACTTCTGAGAC
GAU1 promoter-ChIP	CCACAAAAGCTAGGCTGGTTC TCTTCTTCCTGGGGGTCGTC
Negative probes-ChIP	GGAGCCATTGTATGTCGTGC ACCCCAAAGTCCTACCCAGT
Intergenic region-ChIP	TTCTTAGGGCAGATGGGAAGG GGAGATAACACTCCCTTGAAGCAG
GAPDH-promoter-ChIRP	TCGGAGTCAACGGGTGAGTT CTACCCTGCCCCCATACGA
GAPDH primer set1	TCCAAAATCAAGTGGGGCGA TGATGACCCTTTTGGCTCCC
GAPDH primer set2	GGGAGCCAAAAGGGTCATCA TGGTCATGAGTCCTTCCACG
U2	TCGGCACCGAGAAAGACAAA CGTCTGTGCACTGTTTTGGG
siRNA	
siGALNT8-1	CUCGAUUGUUGAAGGAAAUdTdT
siGALNT8-2	GCUCACAGAAUGUCUACUAdTdT
siGAU1-1	CAGACAUGCACUUCCAUGUdTdT

siGAU1-2	GACACAGAAAAGUCACACAdTdT
siGAU1-3	CAGCAUUGAAAUUGAUCAAdTdT
dCas9 SgRNA	
GAU1 SgRNA1	ACATCTGAATAAACGCTACG
GAU1 SgRNA2	TGTGGAGACCACCCGTAAAC
RNA-FISH	
GAU1 oligos	GATGTGGGTCAGTCTGAGGGATGTTTAGAAAGTTGAATGCGTGC
ChIRP-probes	
GAU1 probes	ATCTATGAAGCTGGGTGGAG GTGAGAGAGGGAGATTCCAT GTTGTTGACTGAAATGAAGG GCGCTATGAAGAAACTTGGC AGAGATACAAGCCACATGGG

Supplementary Table 2. The clinical characteristics of retinoblastoma patient cohorts.

Features	Retinoblastoma	Unaffected
Numbers	10	18
Sex		
Male	6	8
Female	4	10
Age	4.7±2.09	9.28±8.80
ICRB group		
A stage	0	/
B stage	0	/
C stage	0	/
D stage	0	/
E stage	10	/
Laterality		
Unilateral	3	/
Bilateral	7	/

Supplementary Table 3. The clinical characteristics of neuroblastoma patient.

Features	Neuroblastoma
Numbers	8
Sex	
Male	5
Female	3
Age	2.57±3.59
INSS stage	
I stage	0
IIA stage	0
IIB stage	1
III stage	4
IV stage	2
IVS stage	1
Origins	
Adrenal gland	4
Chest	2
Abdominal	2
pelvic	0

Supplementary Table 4 ChIRP-MS identified lncRNA *GAU1* specifically binding proteins

Gene	Number.of. proteins	Score	iBAQ.NC	iBAQ.U2	iBAQ.GAU1	Unique. peptides .NC	Unique. Peptides .U2	Unique. peptides .GAU1	Rank.NC	Rank.U2	Rank.GAU1
RPL35	4	31.389	0	5898700	5136500	0	3	4	1126.5	297	63
CRABP2	2	40.98	0	0	4301100	0	0	5	1126.5	1486	80
RPLP2	3	17.782	0	334750	2414900	0	1	2	1126.5	1074	149
RPL9	9	30.655	0	230610	2297200	0	1	4	1126.5	1159	155
COX5A	4	11.418	0	0	2207800	0	0	2	1126.5	1486	160
RPL23	9	51.091	0	7457600	2062500	0	7	3	1126.5	250	174
SDHC	8	20.475	0	13505000	1001400	2	2	2	1126.5	146	259
RPL14	3	83.327	0	1708500	980580	0	1	2	1126.5	616	264
ILF3	29	323.31	63561	38552000	781660	2	37	7	541	65	293
CBX3	5	85.478	0	64116000	581100	0	5	2	1126.5	40	340
PSMA3	6	12.308	0	0	521320	0	0	2	1126.5	1486	352
GDI2	17	55.315	44407	2062600	500250	1	8	4	558	561	359
PSMD4	4	10.605	0	0	497850	0	0	2	1126.5	1486	361
RBMX	13	323.31	0	49578000	491700	0	14	4	1126.5	55	363
CAPRIN1	7	11.88	0	0	479360	0	0	2	1126.5	1486	365
PSME3	15	19.98	0	681560	466600	0	2	2	1126.5	883	371
ERH	3	304.83	0	28750000	414450	1	7	2	1126.5	83	396
TPM3	78	43.009	0	0	367110	1	0	4	1126.5	1486	410
SURF4	9	12.216	0	749910	313140	0	1	2	1126.5	854	440
WARS	6	17.012	0	0	297600	0	0	3	1126.5	1486	449
HSD17B10	5	49.127	0	2147100	294620	0	5	2	1126.5	552	452
ATP6V1B2	6	10.626	0	0	276270	0	0	2	1126.5	1486	467
PSMD12	3	11.546	0	0	270070	0	0	2	1126.5	1486	471
SF3A3	6	83.198	0	2927600	243540	0	6	2	1126.5	476	483
EIF2S3	5	57.762	0	1215700	207880	1	6	4	1126.5	710	502
PRPF19	5	261.49	0	26712000	206570	0	16	2	1126.5	92	503
ASNS	4	11.27	0	0	198020	0	0	2	1126.5	1486	508
NARS	9	16.416	0	0	190440	0	0	3	1126.5	1486	514
H1FX	1	221.17	0	64014000	188630	0	13	2	1126.5	41	516
EWSR1	17	173.22	0	11528000	184760	0	8	2	1126.5	175	519
HIST1H1C	1	45.429	0	0	182730	3	4	4	1126.5	1486	521
UQCRC1	2	11.407	0	0	182360	0	0	2	1126.5	1486	523
CDC2	8	33.745	0	1194800	181880	0	3	2	1126.5	717	525
RCC2	3	317.84	0	23684000	181290	0	21	3	1126.5	97	526
XRCC6	8	211.92	0	9596000	179710	0	21	3	1126.5	209	528
TRAP1	12	41.852	0	309870	161610	0	3	3	1126.5	1090	544
YWHAH	7	20.275	0	0	153100	0	1	3	1126.5	1486	556
IVD	5	12.096	0	0	137750	0	0	2	1126.5	1486	571
PPP2R1A	19	22.643	0	163090	136130	0	1	3	1126.5	1217	576
TCEA1	11	25.136	0	330570	130530	0	3	2	1126.5	1076	584
ALDOC	14	10.81	0	0	129940	0	0	2	1126.5	1486	589
ACAT1	4	11.127	0	0	117290	0	0	2	1126.5	1486	598
SF3B2	9	323.31	0	17072000	112070	0	26	2	1126.5	123	604
DLST	11	11.298	0	0	108520	0	0	2	1126.5	1486	606
9-Sep	14	12.388	0	0	107210	0	0	2	1126.5	1486	608
PYCR1	26	29.776	0	0	106940	1	0	2	1126.5	1486	609
MCM2	6	158.37	0	3664500	99237	0	17	3	1126.5	420	616
SLC25A11	4	15.164	0	0	99191	1	0	2	1126.5	1486	617
PKM2	14	13.475	0	0	93292	0	0	2	1126.5	1486	623
PTBP2	10	10.852	0	0	92592	0	0	2	1126.5	1486	625
PSIP1	5	285	0	22631000	92129	0	20	2	1126.5	99	626
LARS	16	16.221	0	0	65822	0	0	3	1126.5	1486	655
PSMD11	4	12.656	0	0	62709	1	0	2	1126.5	1486	659
NPEPPS	12	12.221	0	0	57453	0	0	2	1126.5	1486	665
MTHFD1	4	61.478	0	810100	56238	0	9	2	1126.5	826	669
NOP58	5	316.75	0	32196000	50186	0	22	2	1126.5	77	674
GART	12	30.236	0	167940	41956	0	3	2	1126.5	1211	679
SF3B3	11	323.31	0	9955500	25174	0	29	3	1126.5	207	694
SNRNP200	11	323.31	0	18515000	23019	0	72	3	1126.5	112	698
HDLBP	25	25.168	0	32666	13983	1	2	2	1126.5	1317	709
SPTAN1	10	48.021	0	120240	13980	0	5	2	1126.5	1257	710