Supplementary methods and materials

Bioinformatic analysis

TCGA_GBM RNA-seq (HTSeq counts with Verhaak subtype labels) and two GEO microarray datasets (GSE4290, GSE7696) were analyzed. In TCGA, differential expression between mesenchymal (ME) and proneural (PN) tumors was computed with DESeq2 on raw counts after low-expression filtering; genes were called ME-enriched at FDR < 0.05 (Benjamini–Hochberg) and |log2FC| ≥ 1. In GEO, tumor vs non-tumor contrasts were analyzed with limma after RMA normalization; GBM-high genes were defined at FDR < 0.05 and |log2FC| ≥ 1. The intersection of TCGA ME-enriched genes with GBM-high genes present in both GEO cohorts defined the candidate set. Overall survival associations were tested within the TCGA mesenchymal subset using Kaplan–Meier (median split, log-rank test) and Cox proportional hazards models (hazard ratio and 95% CI), with FDR correction for multiple genes.

Cell culture and transfection

Human glioma cell lines LN229, U251, and U87, along with lentivirus packaging HEK293T cells, were obtained from the American Type Culture Collection (ATCC). Cells were cultured in Dulbecco's Modified Eagle Medium (DMEM; ThermoFisher Scientific, USA), supplemented with 10% fetal bovine serum (FBS; Gibco, USA) at 37°C in a humidified atmosphere containing 5% CO2. Authentication of all cell lines was performed using short tandem repeat (STR) profiling, and routine mycoplasma detection was conducted using a mycoplasma PCR detection kit (Sigma, USA).

Small interfering RNAs (siRNAs) were synthesized by Gene Pharma Co. (Shanghai, China) and transfected into cells using Lipofectamine RNAiMAX

transfection reagent (Invitrogen, USA) according to the manufacturer's instructions. Plasmids were extracted using the HiPure Plasmid EF Mini Kit (Magen, Guangzhou, China) and subsequently transfected into cells with the Lipo8000 transfection reagent (Beyotime, China). The sequences of the siRNAs utilized in this study are provided in Table. S2.

Plasmid construction and stable cell line establishment

To generate CRISPR/Cas9-mediated knockout cell lines, single-guide RNAs (sgRNAs) targeting CSRP2 were cloned into the lentiCRISPRv2 backbone (Addgene #52961). For BMI1 and RNF2 knockdown, sgRNAs targeting these genes were cloned into the pLV-sgRNA-dCas9-KRAB-T2A-PuroR plasmid. Full-length cDNAs encoding CSRP2, PDGFRA, BMI1, and RNF2 were amplified from cDNA derived from human U251 cells by PCR and subsequently cloned into either the pCDH-blasticidin lentiviral plasmid or pcDNA3.1, incorporating the appropriate tags to generate overexpression constructs.

For lentivirus production, each lentiviral plasmid was co-transfected into HEK293T cells alongside the packaging plasmids psPAX2 (Addgene #12260) and pMD2.G (Addgene #12259) using Lipo8000 transfection reagent (Beyotime, China). Supernatants containing viral particles were collected every 24 hours for 3 days post-transfection and filtered before use in cell infections. Cell lines were infected with the designated lentiviruses in the presence of 8 μg/ml hexadimethrine bromide (Polybrene; Millipore Sigma, USA). At 24 hours post-infection, puromycin (Invitrogen) or blasticidin (Invitrogen) was added for 3-5 days to select for stably transduced cell lines. To stably knockout CSRP2 in LN229, U251, and U87 cell lines, cells were treated with CRISPR/Cas9 lentivirus targeting human CSRP2. Following the selection of stably infected cells, single cells were plated into 96-well plates to produce single-clonal knockout cell lines. Cells were incubated for 2 weeks, and single-clonal cells were selected and transferred to 12-well plates for expansion. Knockout

efficiency was confirmed by western blotting. Cells with complete knockout of CSRP2 were selected for subsequent experiments. The sequences of the sgRNAs used in this study are listed in Table. S3-4.

CRISPR-resistant CSRP2 rescue and transduction

A CSRP2 cDNA was made resistant to the sg#1 guide (5′-GGCACGCTTAACATGGACCG-3′, targets CDS nt 231–250; PAM TGG) by introducing four synonymous substitutions within the protospacer to disrupt Cas9 binding while preserving the protein sequence. The changes were ACG→ACA (Thr79), CTT→CTC (Leu80), AAC→AAT (Asn81), and GAC→GAT (Asp83). The modified CDS was synthesized and cloned into a lentiviral expression vector with a blasticidin marker. Inserts and junctions were verified by Sanger sequencing.

Lentivirus was produced and sgCSRP2#1 cells were transduced at MOI ~0.3–1 in the presence of polybrene (8μg/mL). Blasticidin selection was applied for 3–5 days to establish stable pools. Rescue expression was confirmed by Western blot for CSRP2 and by sequencing across the edited region. The rescued line is referred to as sg1+CSRP2.

RNA sequencing and CUT&Tag

RNA sequencing was performed to compare differentially expressed genes following the knockout of CSRP2 in U251 cells. Three single-clonal CSRP2 knockout cells and CRISPR/Cas9-sgNC cells were lysed using TRIzol Reagent and sent to BGI for RNA sequencing.

A Hyperactive Universal CUT&Tag Assay Kit for Illumina Pro (Vazyme, TD904-1, China) was utilized for the CUT&Tag assay, following the manufacturer's protocol. Briefly, 3 × 10^4 cells per test were collected, washed with Wash Buffer, and incubated with ConA Beads Pro for 10 minutes at room temperature. The cells were then mixed with Antibody Buffer and the indicated primary antibodies,

and incubated overnight at 4°C.

The next day, a secondary antibody was added, and the mixture was rotated for 1 hour at room temperature before being washed with Dig Wash Buffer. Next, 2μL of pA/G-Tnp pro was added to 98μL of Dig-300 Buffer and rotated for 1 hour at room temperature, followed by three washes with Dig-300 Buffer. The cells were then resuspended in 50μL TTBL and incubated at 37°C for 1 hour for fragmentation. To halt fragmentation, 2μL of 10% SDS was added, and the mixture was incubated at 55°C for 10 minutes. For DNA extraction, 25μL of DNA Extract Beads Pro was added to each tube, gently spun, and held for 20 minutes. Tubes were placed on a magnet stand to clear, washed with B&W Buffer, and kept on the magnet stand until dry. The beads were resuspended in 15μL ddH2O for subsequent amplification. To amplify libraries, 15μL of DNA was mixed with5μL of i5 and i7 primers in different combinations for each sample. A volume of 25μL 2x CAM was added, and PCR was performed following the recommended cycling conditions. Post-PCR clean-up involved adding 100μL of VAHTS DNA Clean Beads (Vazyme, #411), incubating the PCR products with the beads for 5 minutes at room temperature, washing twice with 80% ethanol, and eluting in 2μL ddH2O. The samples were then sent to Haplox Genomic Center (Nanjing, China) for sequencing.

Protein stability assay

To analyze protein stability, an equivalent number of cells were seeded in 12-well plates and incubated overnight. The following day, cells were washed with PBS and treated with fresh medium containing 50μg/ml cycloheximide (CHX, Sigma). Cells were harvested at the indicated time points and lysed with RIPA buffer to detect c-MYC levels by western blotting. To evaluate c-MYC proteasomal degradation, an equivalent number of cells were seeded in 12-well plates and incubated overnight. The next day, cells were washed with PBS and

incubated with fresh medium containing 20μM MG132 for 4-6 hours. Cells were then harvested, and protein levels were assessed by western blotting.

Hypoxia and TGF-β stimulation

LN229 and U251 cells were plated as described above. For hypoxia, cells were cultured in a chamber at 1% O_2, 5% CO_2, balance N_2 for 24 h. YC-1 (5μM) was added 1h before hypoxia and maintained during exposure. HIF-1α, LDHA, and CSRP2 were analyzed by Western blot. LDHA and CSRP2 mRNA were quantified by qRT-PCR.

For TGF-β stimulation, cells were treated with recombinant human TGF-β1 at 2, 4, 8 (ng/mL) for 24h. Where indicated, SB431542 (10μM) was added 1 h before TGF-β1. CSRP2 and SMAD2/3 were assessed by Western blot.

Sphere formation assay

LN229 and U251 cells were tested in four groups per line: sgNC, sgCSRP2#1, sgCSRP2#2, and sg1+CSRP2. Cells were dissociated to single-cell suspensions and passed through a 40-μm strainer. Cells were seeded into ultra-low-attachment plates at ~1×10^3 cells per well in serum-free neurosphere medium (DMEM/F12 with B27 and N2 supplements, EGF and bFGF) and cultured without serum. Medium and growth factors were refreshed every 2–3 days. After 7-10 days, spheroids with smooth borders and diameter ≥50 μm were counted as spheres. Sphere number per well and median sphere diameter were quantified in Fiji.

Immunohistochemistry (IHC) and hematoxylin and eosin (H&E) staining

Immunohistochemistry (IHC) staining was performed using an Immunohistochemistry Kit (ZSGB-BIO, PV-9000). Specimens from glioma

patients and xenograft mice were embedded in paraffin and sectioned into 4μm slides. Sections were deparaffinized in xylene, rehydrated in ethanol, and subjected to antigen retrieval by microwaving in citrate buffer (pH 6.0). Endogenous peroxidase activity was blocked with a blocking solution. Sections were then incubated with the indicated primary antibodies at 37°C for 60 minutes, followed by incubation with secondary antibodies at 37°C for 20 minutes. The sections were stained with DAB for 3-5 minutes and counterstained with hematoxylin for imaging. For H&E staining, sections were first treated with 10% hematoxylin solution for 5 minutes and subsequently with 1% eosin solution for 2 minutes. All images were captured using a slide scanner and analyzed with Imagescope.

Temozolomide IC50 assay

U251 and LN229 were tested in four groups per line: sgNC, sgCSRP2#1, sgCSRP2#2, and sg1+CSRP2. Cells were seeded at ~8×10^3 cells per well in 96-well plates. After 18–24 h, temozolomide was added at 0–2000μM and incubated for 48 h. Viability was measured by CCK-8 and OD450. Values were blank-subtracted and normalized to vehicle. Each condition used 6 technical wells averaged to one value per biological replicate. IC50 was estimated by a four-parameter logistic fit in GraphPad Prism. Group differences were tested by the extra-sum-of-squares F test.

Xenograft model

U87 cells (5 × 10^6 cells in 100μL DMEM containing 0.5 v/v Matrigel; Corning, USA), either stably knocking out CSRP2 or control CRISPR/Cas9-sgNC, were injected subcutaneously into the flanks of 5-week-old female BALB/c nude mice (Gempharmatech, Nanjing, China). Tumor volumes were measured every 3-4

days and calculated using the formula v=length×width^2×1/2. Three weeks post-injection, the mice were euthanized, and tumors were excised. Tumor weights were measured after resection, and samples were fixed in 4% paraformaldehyde for further immunohistochemical analysis or lysed for further western blotting.

Seahorse extracellular flux assays (ECAR and OCR)

Assays were performed on an Agilent Seahorse XFe96. U251 cells were used and included four groups: sgNC, sgCSRP2#1, sgCSRP2#2, and sg1+CSRP2 (rescue in the sg#1 background). Cartridges were hydrated overnight at 37 °C without CO_2. Cells were seeded at ~1×10^4 cells per well 18–24 h before measurement to reach ~80–90% confluence at assay time. Replicate numbers followed the study design described above.

Glycolysis stress test (ECAR): One hour before measurement, medium was replaced with Seahorse XF DMEM containing 2 mM L-glutamine, pH 7.4. Plates equilibrated for 45–60 min at 37 °C without CO_2. Injections were glucose to 10 mM, oligomycin to 1μM, and 2-deoxy-D-glucose to 50mM. ECAR was recorded for three baseline cycles and at least three cycles after each injection. Basal glycolysis, glycolytic capacity, and glycolytic reserve were calculated in Wave software per manufacturer definitions.

Mitochondrial stress test (OCR): Assay medium was Seahorse XF DMEM containing 10mM D-glucose, 1 mM sodium pyruvate, and 2mM L-glutamine, pH 7.4. Plates equilibrated for 45–60 min at 37 °C without CO_2. Injections were oligomycin to 1μM, FCCP to 1μM, and rotenone/antimycin A to 0.5μM each. Basal respiration, ATP-linked respiration, maximal respiration, and spare respiratory capacity were computed in Wave.

Normalization and QC: After assays, wells were rinsed and lysed for BCA protein quantification. Flux values were normalized to total protein per well.

Wells with detachment, air bubbles, or outlier normalization were excluded by pre-specified QC rules. Group differences were tested by one-way ANOVA with Tukey multiple comparisons.

Alkaline comet assay

Cells carrying sgNC, sgCSRP2#1, sgCSRP2#2, and sg1+CSRP2 rescue were irradiated at 5 Gy and collected at 0, 1, and 12 h. Cells were embedded in 0.5% low-melting agarose on comet slides, lysed at 4 °C, and alkaline-unwound in pH > 13 buffer. Electrophoresis ran at ~1 V/cm for ~25 min at 4 °C, followed by neutralization and DNA staining. At least 100 nucleoids per condition per biological replicate were scored with Fiji. The endpoint was percent DNA in tail.

Supplementary Figures

Supplementary Fig.1

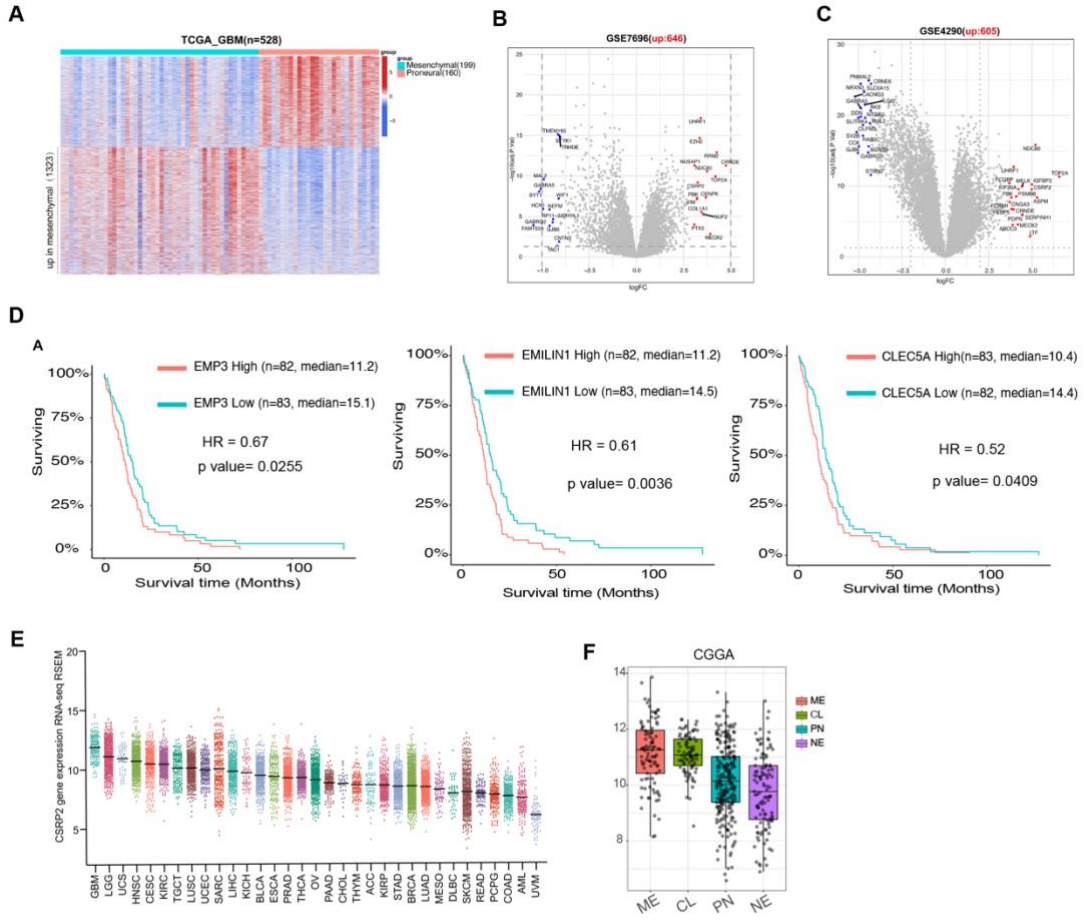

Fig. S1 Cross-cohort screen and expression patterns related to CSRP2. (A) TCGA_GBM heatmap contrasting mesenchymal (ME) and proneural (PN) tumors, highlighting genes upregulated in ME. (B–C) Volcano plots from GSE7696 and GSE4290 showing ME-enriched genes. (D) Kaplan–Meier analyses in the ME subgroup identify EMP3, EMILIN1, and CLEC5A as candidates associated with poorer survival (log-rank *p* values shown on plots). (E) Pan-cancer CSRP2 mRNA expression across TCGA cohorts, with highest levels in glioma. (F) CSRP2 expression across GBM subtypes in CGGA (ME,

CL, PN, NE).

Supplementary Fig.2

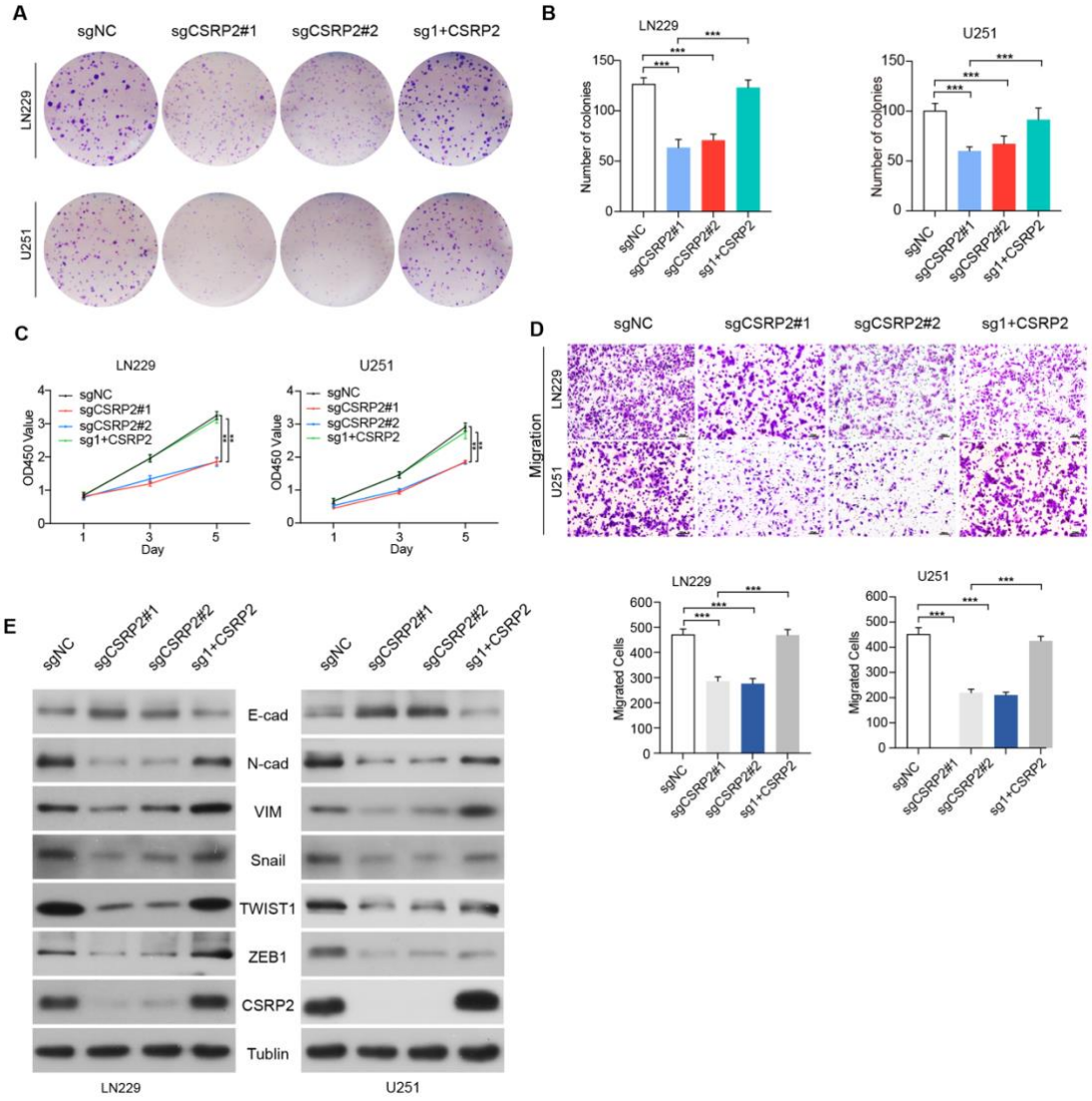

Fig. S2 CSRP2 rescue restores proliferation and migration and partially reverses EMT changes. (A) Representative colony-formation images of LN229 and U251 cells (sgNC, sgCSRP2#1, sgCSRP2#2, and rescue with CRISPR-resistant CSRP2, sg1+CSRP2). (B) Quantification of colony numbers. (C) CCK-8 growth curves. (D) Transwell migration images and quantification; scale bar, 100μm. (E) Western blotting of EMT markers showing increased E-

cadherin and decreased N-cadherin, Vimentin, Snail, Twist1, and ZEB1 after CSRP2 knockout, with partial restoration upon CSRP2 re-expression. Data are represented as mean ± SD. Significance: ns, not significant, * p<0.05, ** p<0.01, *** p<0.001, **** p<0.0001.

Supplementary Fig.3

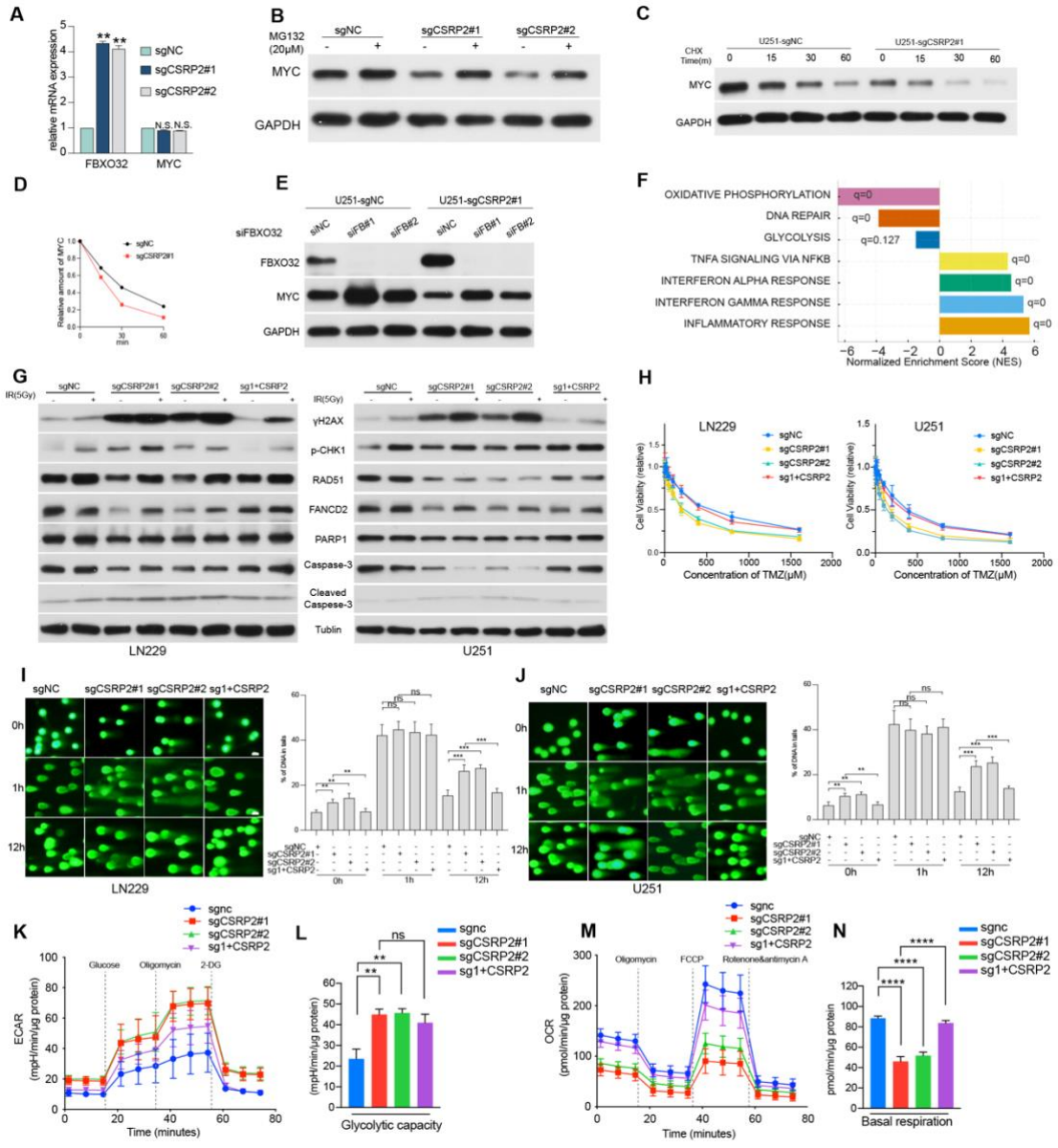

Fig. S3 CSRP2 loss destabilizes MYC, heightens DNA-damage responses,

increases TMZ sensitivity, and shifts metabolism. (A) RT-qPCR of FBXO32 and MYC after CSRP2 knockout. (B) Proteasome inhibition with MG132 (20μM, 4h) restores MYC protein in sgCSRP2 cells (Western blotting). (C) Cycloheximide chase (50μg/mL; 15–60min) shows reduced MYC stability after CSRP2 loss (Western blotting). (D) Quantification of MYC decay from CHX chases. (E) siFBXO32 partially rescues MYC protein in CSRP2-knockout cells (Western blotting). (F) Hallmark GSEA: inflammatory/interferon and TNFα/NF-κB pathways positively enriched; oxidative phosphorylation and DNA repair negatively enriched; glycolysis shows a modest negative trend. (G) DNA-damage/repair markers after 5Gy IR: γH2AX, p-CHK1, RAD51, and FANCD2 change consistently with increased damage; CSRP2 re-expression partially restores. (H) Temozolomide dose–response curves (LN229, U251) showing sensitization upon CSRP2 knockout and partial rescue by CSRP2 re-expression. (I–J) Comet assays at 0, 1, and 12h indicate delayed DNA-break repair after CSRP2 loss with partial rescue. (K–L) Glycolysis stress test: ECAR time course and glycolytic capacity. (M–N) Mito stress test: OCR time course and basal respiration. Data are represented as mean ± SD. Significance: ns, not significant, * $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$.

Supplementary Fig.4

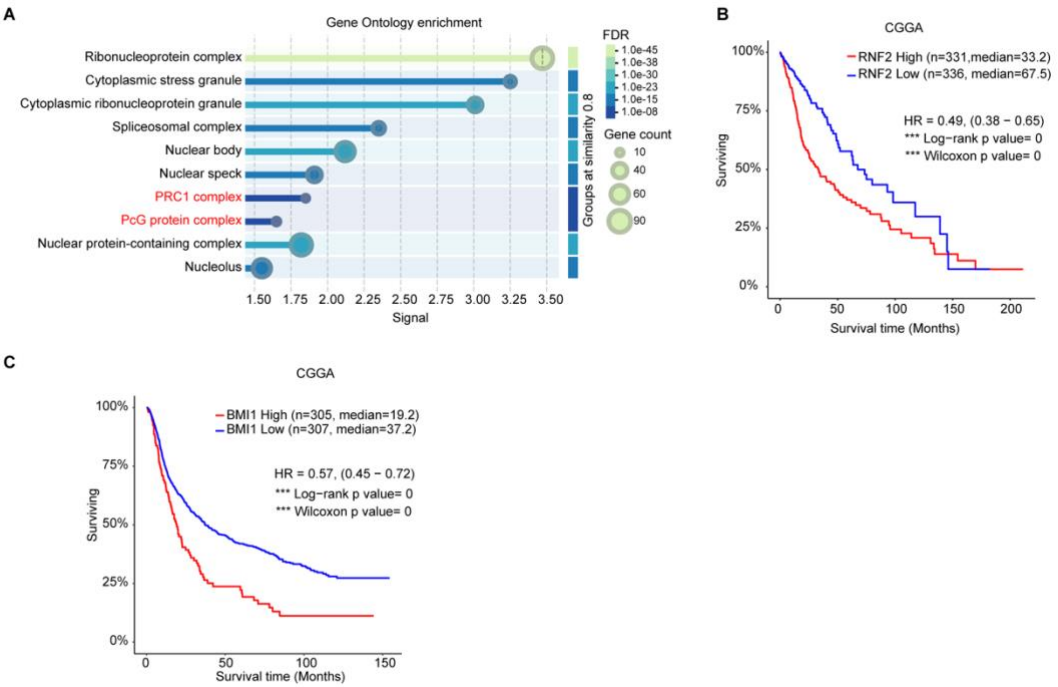

Fig. S4 CSRP2 interactome enrichment and clinical relevance of PRC1 components. (A) Gene Ontology enrichment of proteins from CSRP2 IP–MS, highlighting PcG/PRC1 complexes. (B–C) Kaplan–Meier analyses in the CGGA cohort showing shorter overall survival with higher RNF2 (B) or BMI1 (C); log-rank $p<0.0001$.

Supplementary Fig.5

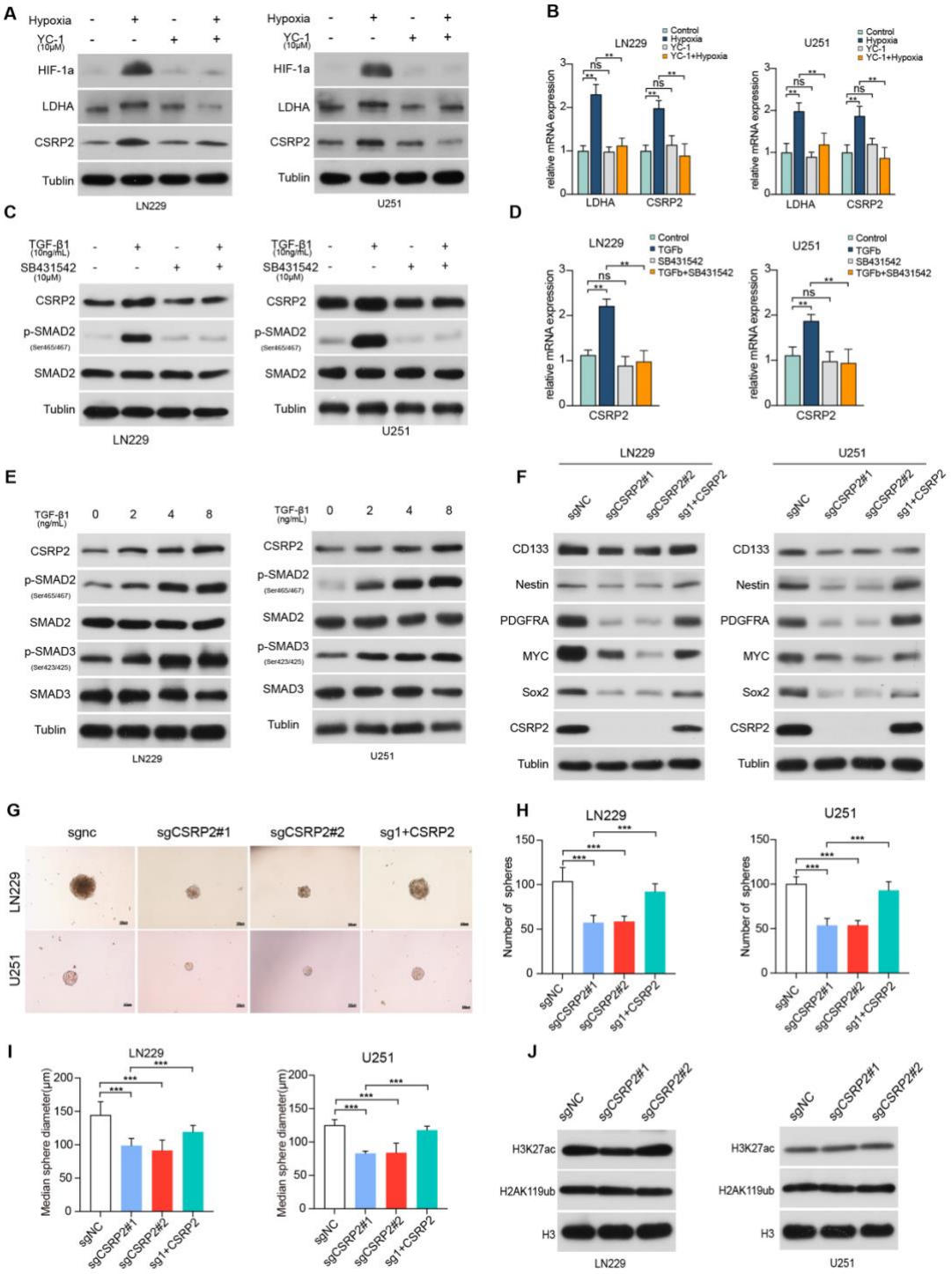

Fig. S5 Hypoxia and TGF-β1 induce CSRP2, and CSRP2 knockout reduces glioma cell stemness. (A) Western blotting under normoxia, hypoxia, and

hypoxia+YC-1 (5μM) in LN229 and U251; HIF-1α/LDHA induction confirms hypoxia and CSRP2 increases with hypoxia but is blocked by YC-1. (B) RT-qPCR for LDHA and CSRP2 under the same conditions. (C) Western blotting with TGF-β1 (10 ng/mL) ± SB431542 (10μM) shows CSRP2 induction with p-SMAD2 increase; SB431542 blocks both. (D) RT-qPCR confirms TGF-β1–induced CSRP2 and its inhibition by SB431542. (E) Dose response to TGF-β1 (0, 2, 4, 8 ng/mL) shows graded increases in CSRP2 with p-SMAD2/p-SMAD3 activation. (F) Western blotting of stemness-associated markers after CSRP2 knockout and rescue in LN229 and U251: CD133, Nestin, PDGFRA, MYC, and SOX2 decrease with CSRP2 loss and are partially restored by re-expression. (G) Tumorsphere images for sgNC, sgCSRP2#1, sgCSRP2#2, and rescue (sg1+CSRP2). (H) Quantification of sphere number. (I) Median sphere diameter. (J) Acid-extracted histone immunoblots of LN229 and U251 showing H3K27ac and H2AK119ub1 with H3 as loading control; global levels are largely unchanged after CSRP2 knockout, consistent with locus-specific remodeling rather than global shifts. Data are represented as mean ± SD. Significance: ns, not significant, * $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$.

Supplementary Fig.6

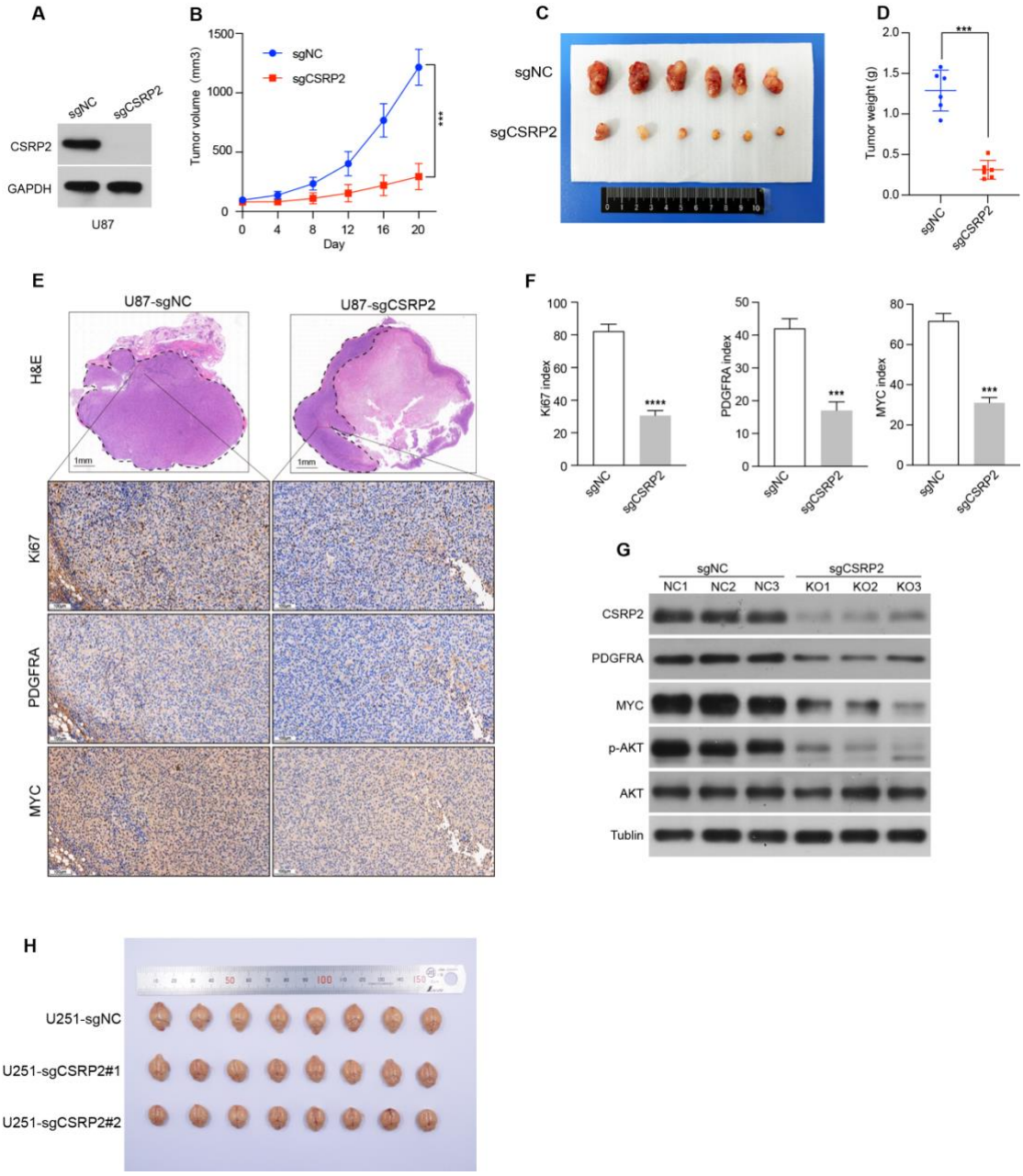

Fig. S6 Subcutaneous xenografts showing growth suppression after CSRP2 knockout. (A) Western blot confirming CSRP2 loss in U87 cells. (B) Tumor growth curves of U87 xenografts (sgNC vs sgCSRP2). (C) Representative excised tumors. (D) Tumor weights. (E) H&E and IHC for Ki-67, PDGFRA, and MYC in U87 tumors; scale bars, 1mm (whole section) and 100μm (IHC). (F) Quantification of Ki-67, PDGFRA, and MYC IHC indices. (G)

Western blots of xenograft lysates showing CSRP2, PDGFRA, MYC, p-AKT, and AKT. (H) Representative orthotopic intracranial tumors from U251 cohorts (sgNC, sgCSRP2#1, sgCSRP2#2). Data are represented as mean ± SD. Significance: ns, not significant, * $p<0.05$, ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$.

Supplementary Tables

Supplementary Table 1 primers used in the study

Genes	Sequences（5'-3'）
GAPDH-qF	GGAGCGAGATCCCTCCAAAAT
GAPDH-qR	GGCTGTTGTCATACTTCTCATGG
MYC-qF	CCCTCCACTCGGAAGGACTA
MYC-qR	GCTGGTGCATTTTCGGTTGT
PDGFRA-qF	TGGCAGTACCCCATGTCTGAA
PDGFRA-qR	CCAAGACCGTCACAAAAAGGC
FBXO32-qF	AAGTCTGTGCTGGTCGGGAA
FBXO32-qR	AGTGAAGGTGAGGCCTTTGAAG
CDKN2A-qF	CTCGTGCTGATGCTACTGAGGA
CDKN2A-qR	GGTCGGCGCAGTTGGGCTCC
CDKN1A-qF	TGTCCGTCAGAACCCATGC
CDKN1A-qR	AAAGTCGAAGTTCCATCGCTC
E2F1-qF	ACGCTATGAGACCTCACTGAA
E2F1-qR	TCCTGGGTCAACCCCTCAAG
BMP7-qF	TCGGCACCCATGTTCATGC
BMP7-qR	GAGGAAATGGCTATCTTGCAGG
BMI1-qF	AATCCCCACCTGATGTGTGT
BMI1-qR	GCTGGTCTCCAGGTAACGAA
RNF2-qF	GTCGTGATGAGTATGAAGCTC
RNF2-qR	TTGTTACTAGGGCCTGCTTC
FBXL7-qF	GTGACCTGCATCAGCTTGAC
FBXL7-qR	CAGTCCGTCATGTCCAGGTA
ATF3-qF	TGCTCAGAGAAGTCGGAAGAA
ATF3-qR	TGGCACAAAGTTCATAGGGCA
LDHA-qF	ATGGCAACTCTAAAGGATCAGC
LDHA-qR	CCAACCCCAACAACTGTAATCT
CSRP2-qF	TGGGAGGACCGTGTACCAC
CSRP2-qR	CCGTAGCCTTTTGGCCCATA

ACTA1-qF	GGCATTCACGAGACCACCTAC
ACTA1-qR	CGACATGACGTTGTTGGCATAC

1 **Supplementary Table 2 siRNAs used in the study**

Genes	Sense（5’-3’）	Antisense
siFBXO32#1	CUGGAUUCCAGAAGAUUUA	UAAAUCUUCUGGAAUCCAG
siFBXO32#2	GCAGAUCCGCAAACGAUUATT	UAAUCGUUUGCGGAUCUGCTT
siPDGFRA#1	CCUCUAUCCUUCCAAAUGATT	UCAUUUGGAAGGAUAGAGGTT
siPDGFRA#4	GAGCUUCACCUAUCAAGUUTT	CAGGAAGCUAUCCCUAUUCTT

2 **Supplementary Table 3 sgRNAs for CRISPR knockout**

Genes	Forward（5’-3’）	Reverse
sgCSRP2#1	GGCACGCTTAACATGGACCG	CGGTCCATGTTAAGCGTGCC
sgCSRP2#2	GACCGTGTACCACGCAGAAG	CTTCTGCGTGGTACACGGTC
sgCSRP2#3	GTGGTACACGGTCCTCCCAC	GTGGGAGGACCGTGTACCAC
sgNC	CGCTTCCGCGGCCCGTTCAA	TTGAACGGGCCGCGGAAGCG

3 **Supplementary Table 4 sgRNAs for CRISPR knockdown**

Genes	Forward（5’-3’）	Reverse
sgBMI1#1	GTGTGTGTGCGTGCGAGCGG	CCGCTCGCACGCACACACAC
sgBMI1#2	CGTGTGTGTGCGTGCGAGCG	CGCTCGCACGCACACACACG
sgBMI1#3	CCCGCTCGCACGCACACACA	TGTGTGTGCGTGCGAGCGGG
sgRNF2#1	CTGATACCAGAGTCTTGCTC	GAGCAAGACTCTGGTATCAG
sgRNF2#2	CCGCGGCCGGAGCAAGACTC	GAGTCTTGCTCCGGCCGCGG
sgNC	GCGGGCAGAACGACCCTGAC	GTCAGGGTCGTTCTGCCCGC

4 **Supplementary Table 5 Antibodies used in the study**

Antibodies	sources	Identifier
Rabbit monoclonal anti- BMI1	CST	#6964
Rabbit monoclonal anti-p-Akt（Ser473）	CST	#4060
Rabbit monoclonal anti-P110α	CST	#4249
Rabbit polyclonal anti-p-PI3K	CST	#4228
Rabbit polyclonal anti-MYC	Proteintech	10828-1-AP
Mouse monoclonal anti-GAPDH	Proteintech	60004-1-Ig
Rabbit monoclonal anti-CDK4	CST	#12790
Rabbit polyclonal anti-CSRP2	Proteintech	10892-2-AP

Rabbit monoclonal anti- P21(CDKN1A)	CST	#2947
Rabbit monoclonal anti- P16(CDKN2A)	ABclonal	A23450
Rabbit monoclonal anti-FBXO32	Abcam	ab168372
Rabbit polyclonal anti-Akt	CST	#9272
Rabbit monoclonal anti-RNF2	CST	#5694
Rabbit monoclonal anti-PDGFRA	CST	#5241
Rabbit polyclonal anti-E2F1	CST	#3742
Rabbit polyclonal anti-H3K27ac	Abcam	ab4729
Rabbit monoclonal anti-H2AK119ub	PTMBIO	PTM-7449
Rabbit polyclonal anti-β-Tublin	Bioworld	AP0064
Goat Anti-Rabbit IgG (H+L)	Proteintech	SA400001-2
Goat Anti-Mouse IgG (H+L)	Proteintech	SA400001-1
Rabbit IgG control polyclonal antibody	Proteintech	30000-0-AP
Rabbit monoclonal anti-Flag	CST	#14793
Rabbit monoclonal anti-HA	CST	#3724
Mouse monoclonal anti-HIF1a	Novus	NB100-10S
Rabbit monoclonal anti-LDHA	Proteintech	84198-1-RR
Rabbit monoclonal anti-SMAD2	Abmart	T55090
Rabbit monoclonal anti-SMAD3	Abmart	T55013
Rabbit monoclonal anti-p-SMAD2	Immunoway	YM8539
Rabbit monoclonal anti-p-SMAD3	Immunoway	YM8682
Rabbit polyclonal anti-Ki67	Proteintech	27309-1-AP
Rabbit monoclonal anti-γH2AX	Abmart	TA3187
Rabbit monoclonal anti-p-CHK1(ser345)	CST	#2348
Rabbit monoclonal anti-RAD51	CST	#8875
Rabbit monoclonal anti-FANCD2	CST	#16323
Rabbit polyclonal anti-CD133	Novus	NB120
Rabbit monoclonal anti-SOX2	CST	#23064
Rabbit monoclonal anti-Nestin	CST	#89529
Rabbit monoclonal anti-E-Cad	CST	#3195
Rabbit monoclonal anti-N-Cad	CST	#13116
Rabbit monoclonal anti-Vimentin	CST	#5741
Rabbit monoclonal anti-Snail	CST	#3879

Rabbit monoclonal anti-ZEB1	CST	#3396
Rabbit monoclonal anti-Twist1	CST	#90445
Rabbit monoclonal anti-caspase3	CST	#9622
Rabbit monoclonal anti-PARP1	CST	#9542

Supplementary Table 6

Correlation of CSRP2 expression with clinicopathological features in glioma patients. p values were determined using Chi-square and Fisher's exact tests.

Variable		CSRP2 high	CSRP2 low	p value
Age	≥45	182	144	0.0035
	<45	125	161	
Gender	Male	186	171	0.2865
	Female	121	134	
WHO grade	2	91	137	<0.0001
	3	115	130	
	4	105	46	
Subtype	Neural	32	80	<0.0001
	Proneural	92	148	
	Classical	68	20	
	Mesenchymal	71	27	
IDH1 status	Mutant	174	256	<0.0001
	Wild-type	158	77	
1p/19q	Codel	38	133	<0.0001
	Non-codel	293	202	
MGMT promoter	Methylated	211	267	<0.0001
	Unmethylated	102	61	