Supporting Information

HIF-1α–SHMT-driven serine catabolism protects glioblastoma from ferroptosis in the serine/glycine-restricted brain microenvironment

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Supplementary Figures

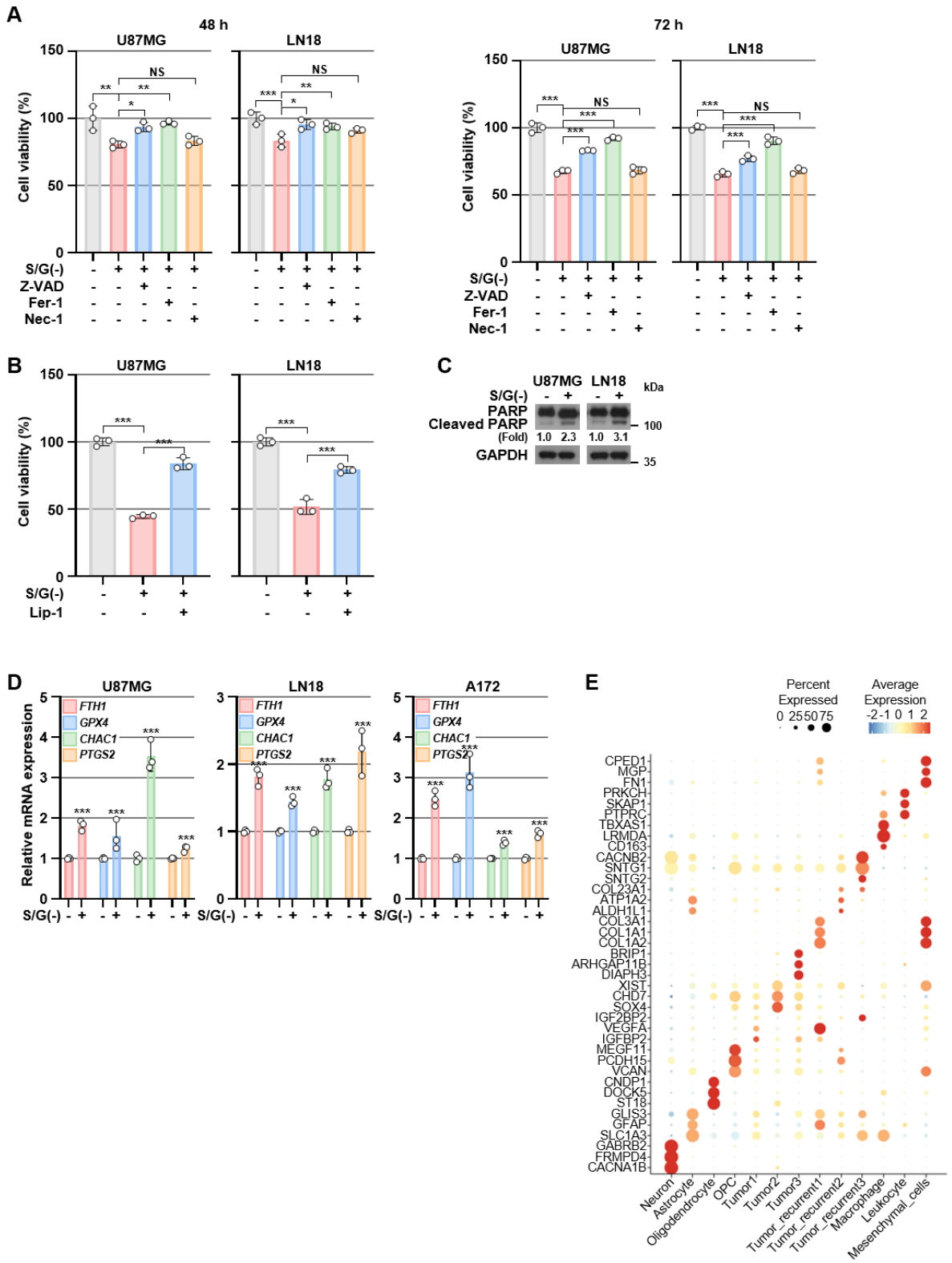


Figure S1 (related to Figure 1). Serine/glycine deprivation induces ferroptotic stress and cell death in GBM cells.

(A) Cell viability was measured after 48 or 72 h of concurrent S/G deprivation and inhibitor treatment. Cells were cultured in control or S/G(−) media, with or without Z-VAD-FMK (Z-VAD, 10 μM), Ferrostatin-1 (Fer-1, 5 μM), or Necrostatin-1 (Nec-1, 25 μM). **(B)** Cells were cultured in control or S/G(−) media for 96 h in the presence or absence of Liproxstatin-1 (Lip-1, 2 μM), and cell viability was measured. **(C)** The specified cell lines were maintained in S/G(−) or control media for 24 h. IB analyses were performed using the indicated antibodies. **(D)** The specified cell lines were maintained in S/G(−) or control media for 24 h. qPCR analyses were performed using the indicated primers. **(E)** Dot plots showing the marker genes of each cell type presented in Figure 1H. Data are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (A, B, D). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$. NS, not significant.

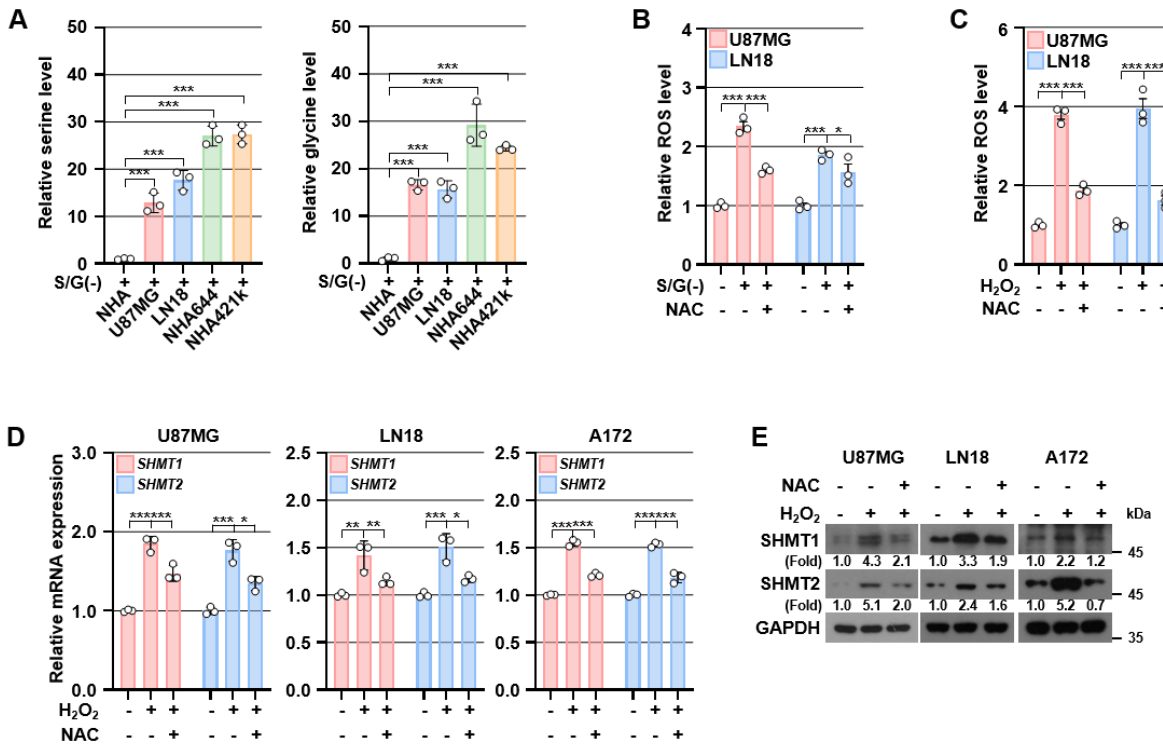
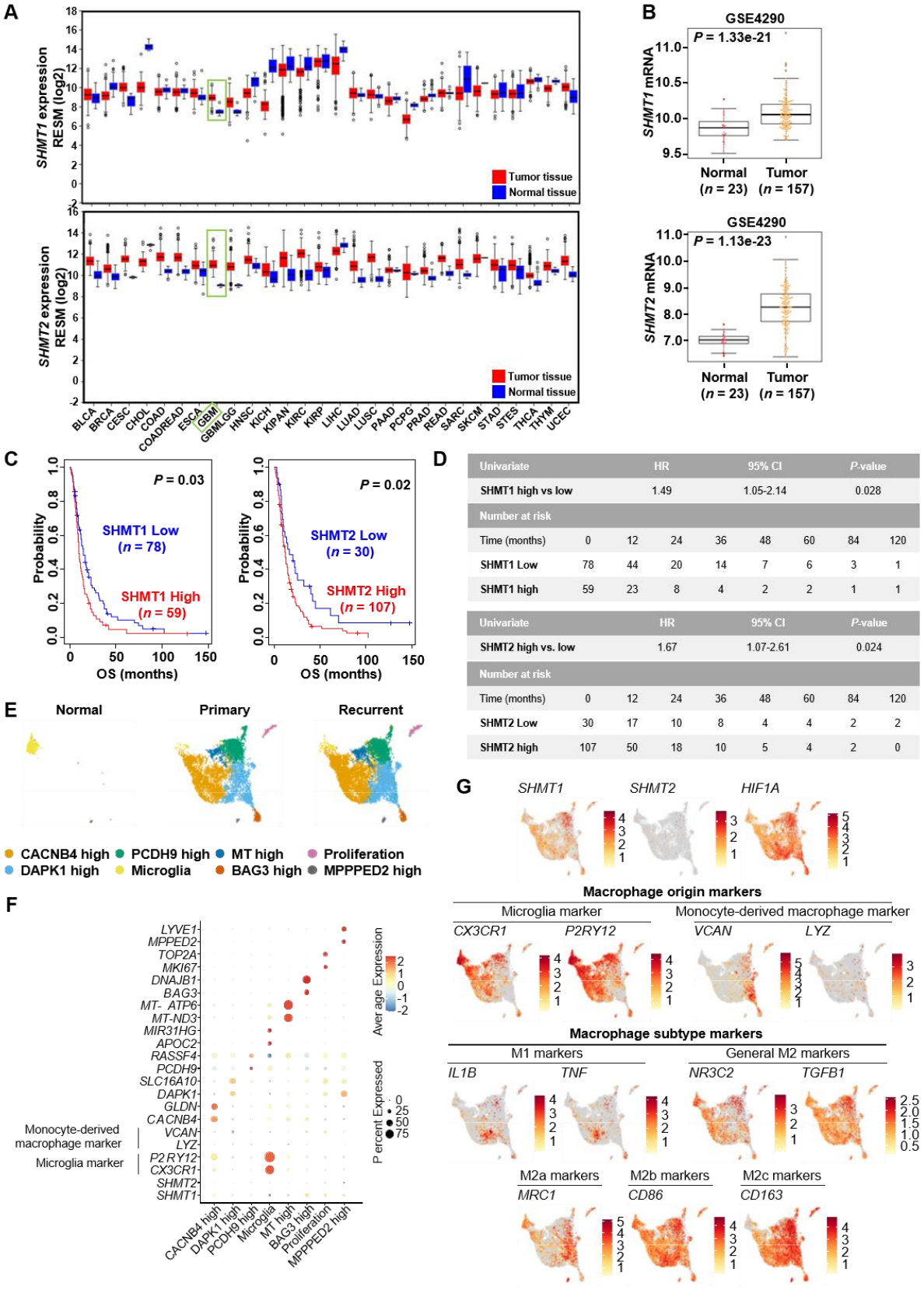


Figure S2 (related to Figure 2). S/G deprivation induces SHMT1/2 expression in GBM cells in association with oxidative-stress responses.

(A) The specified cell lines were maintained in S/G(−) media for 24 h. Serine (left panel) and glycine (right panel) levels were measured. **(B)** The specified cell lines were maintained in S/G(−) or control medium for 24 h in the presence or absence of NAC (2 mM). Intracellular ROS levels were measured. **(C)** The specified cell lines were maintained in complete media with or without H_2O_2 (100 μM) for 24 h in the presence or absence of NAC (2 mM). ROS levels were measured. **(D** and **E)** The specified cell lines were maintained in complete media with or without H_2O_2 (100 μM) for 24 h in the presence or absence of NAC (2 mM). qPCR (D) and IB (E) analyses were performed using the indicated primers and antibodies, respectively. Data are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (A-D). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.



Univariate	HR	95% CI	P-value
SHMT1 high vs low	1.49	1.05-2.14	0.028

Number at risk								
Time (months)	0	12	24	36	48	60	84	120
SHMT1 Low	78	44	20	14	7	6	3	1
SHMT1 high	59	23	8	4	2	2	1	1

Univariate	HR	95% CI	P-value
SHMT2 high vs. low	1.67	1.07-2.61	0.024

Number at risk								
Time (months)	0	12	24	36	48	60	84	120
SHMT2 Low	30	17	10	8	4	4	2	2
SHMT2 high	107	50	18	10	5	4	2	0

Figure S3 (related to Figure 3). Elevated SHMT1/2 expression is associated with

malignant features and poor prognosis in GBM.

(A) Differential expression analysis of *SHMT1* (top panel) and *SHMT2* (bottom panel) across 28 human cancer types using data from FireBrowse (www.firebrowse.org). Expression levels of *SHMT1* and *SHMT2* were analyzed. Tumor tissues are shown in red and corresponding normal tissues are shown in blue. TCGA study abbreviations: BLCA, bladder urothelial carcinoma; BRCA, breast invasive carcinoma; CESC, cervical squamous cell carcinoma and endocervical adenocarcinoma; CHOL, cholangiocarcinoma; COAD, colon adenocarcinoma; COADREAD, colorectal adenocarcinoma; ESCA, esophageal carcinoma; GBM, glioblastoma multiform; GBM LGG, glioblastoma multiform and brain lower grade glioma; HNSC, head and neck squamous cell carcinoma; KICH, kidney chromophobe; KIPAN, pan-kidney cohort; KIRC, kidney renal clear cell carcinoma; KIRP, kidney renal papillary cell carcinoma; LIHC, liver hepatocellular carcinoma; LUAD, lung adenocarcinoma; LUSC, lung squamous cell carcinoma; PAAD, pancreatic adenocarcinoma; PCPG, pheochromocytoma and paraganglioma; PRAD, prostate adenocarcinoma; READ, rectum adenocarcinoma; SARC, sarcoma; SKCM, skin cutaneous melanoma; STAD, stomach adenocarcinoma; STES, stomach and esophageal carcinoma; THCA, thyroid carcinoma; THYM, thymoma; UCEC, uterine corpus endometrial carcinoma. **(B)** Analysis of *SHMT1* and *SHMT2* mRNA expression in the GEO dataset (GSE4290) in normal (n = 23) and brain tumor (n = 157) tissues. **(C)** Kaplan–Meier analysis of overall survival (OS) according to SHMT1 (left) or SHMT2 (right) expression in the CGGA cohort. Patients were stratified into high- and low-expression groups using the optimal cutoff values described in the Methods. Survival differences were assessed using the log-rank test. **(D)** Univariate Cox proportional hazards regression analyses of overall survival according to SHMT1 (top) or SHMT2 (bottom) expression. Hazard ratios (HRs), 95% confidence intervals (CIs), and *P*-values are shown. The numbers of patients at risk at the

indicated time points are also presented. **(E)** UMAP visualization showing cells colored according to cell type identity. **(F)** Dot plots showing the expression of *SHMT1*, *SHMT2*, microglial marker genes (*CX3CR1* and *P2RY12*), monocyte-derived macrophage marker genes (*LYZ* and *VCAN*), and the other cluster marker genes indicated in the figure. **(G)** Feature plots displaying the expression of *SHMT1*, *SHMT2*, *HIF1A*, macrophage origin markers (microglia: *CX3CR1* and *P2RY12*; monocyte-derived macrophages: *VCAN* and *LYZ*), and macrophage subtype markers (M1: *IL1B* and *TNF*; general M2: *NR3C2* and *TGFB1*; M2a: *MRC1*; M2b: *CD86*; M2c: *CD163*).

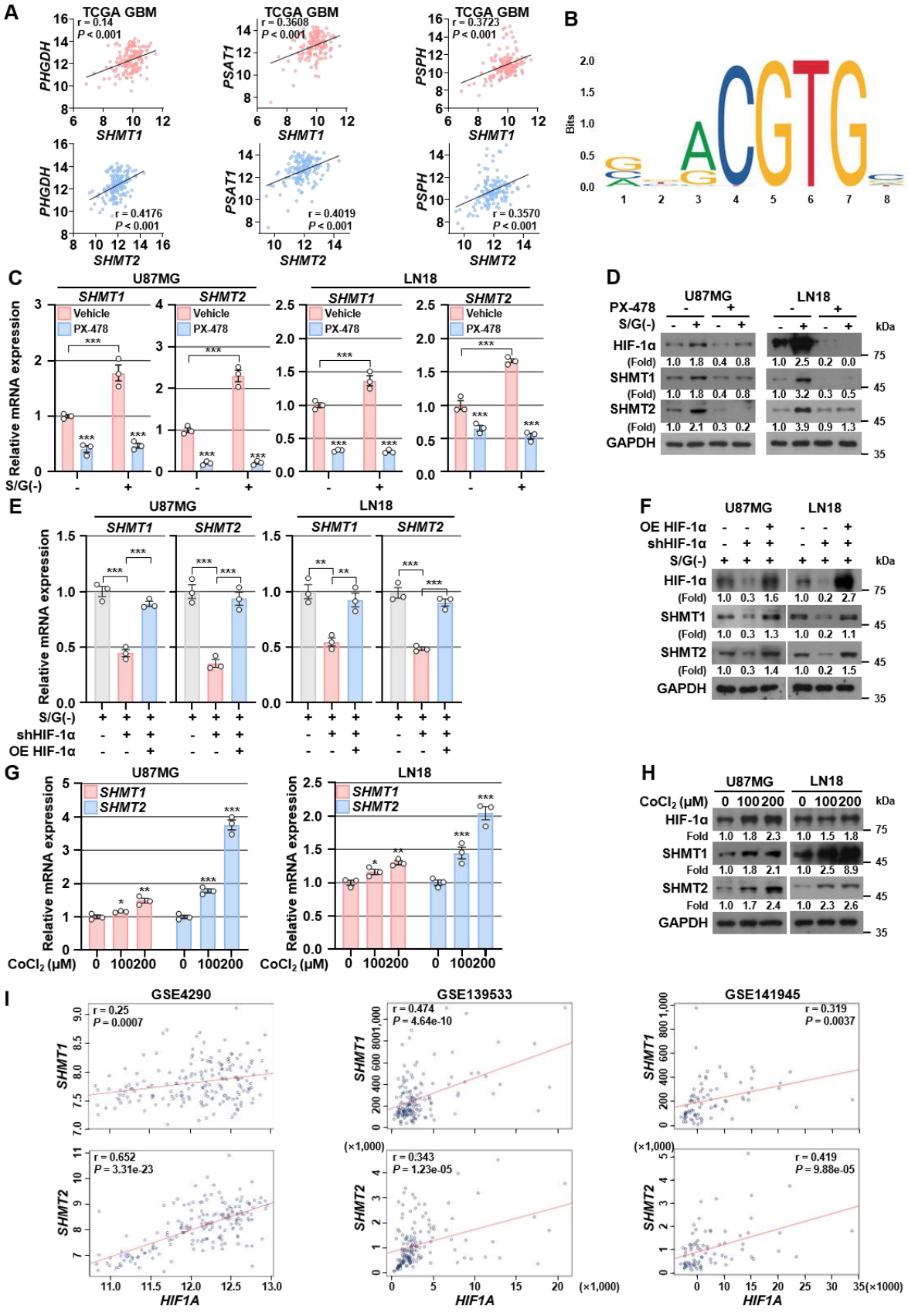

A
TCGA GBM
r = 0.14
P < 0.001
PHGDH
SHMT1
r = 0.4176
P < 0.001
SHMT2
r = 0.3608
P < 0.001
PSAT1
r = 0.4019
P < 0.001
r = 0.3723
P < 0.001
PSPH
r = 0.3570
P < 0.001
B
Bits
C
U87MG
LN18
SHMT1
SHMT2
Vehicle
PX-478
Relative mRNA expression
S/G(-)
D
PX-478
S/G(-)
kDa
HIF-1α
(Fold) 1.0 1.8 0.4 0.8 1.0 2.5 0.2 0.0
SHMT1
(Fold) 1.0 1.8 0.4 0.8 1.0 3.2 0.3 0.5
SHMT2
(Fold) 1.0 2.1 0.3 0.2 1.0 3.9 0.9 1.3
GAPDH
75
45
45
35
E
U87MG
LN18
SHMT1
SHMT2
Relative mRNA expression
S/G(-)
shHIF-1α
OE HIF-1α
F
U87MG
LN18
OE HIF-1α
shHIF-1α
S/G(-)
kDa
HIF-1α
(Fold) 1.0 0.3 1.6 1.0 0.2 2.7
SHMT1
(Fold) 1.0 0.3 1.3 1.0 0.2 1.1
SHMT2
(Fold) 1.0 0.3 1.4 1.0 0.2 1.5
GAPDH
75
45
45
35
G
U87MG
LN18
SHMT1
SHMT2
Relative mRNA expression
CoCl2 (μM) 0 100200 0 100200
H
U87MG
LN18
CoCl2 (μM) 0 100200 0 100200
kDa
HIF-1α
Fold 1.0 1.8 2.3 1.0 1.5 1.8
SHMT1
Fold 1.0 1.8 2.1 1.0 2.5 8.9
SHMT2
Fold 1.0 1.7 2.4 1.0 2.3 2.6
GAPDH
75
45
45
35
I
GSE4290
r = 0.25
P = 0.0007
SHMT1
r = 0.652
P = 3.31e-23
SHMT2
HIF1A
GSE139533
r = 0.474
P = 4.64e-10
SHMT1
r = 0.343
P = 1.23e-05
SHMT2
(×1,000)
HIF1A
GSE141945
r = 0.319
P = 0.0037
SHMT1
r = 0.419
P = 9.88e-05
SHMT2
(×1,000)
HIF1A

Figure S4 (related to Figure 4). HIF-1α directly transactivates SHMT1/2 under serine/glycine deprivation and correlates with SHMT1/2 expression in human GBM.

(A) TCGA analysis of the correlations between *PHGDH, PSAT1* or *PSPH* mRNA expression and *SHMT1* or *SHMT2* mRNA expression in the TCGA-GBM dataset (n = 141). **(B)** Predicted HIF-1α-binding sites within the *SHMT1* and *SHMT2* promoter regions and the corresponding regions analyzed by ChIP-qPCR and promoter luciferase assays. The predicted HIF-1α-binding motif is shown as a sequence logo. **(C** and **D)** The specified cell lines were maintained in S/G(−) or control media for 24 h in the presence or absence of PX-478 (10 μM). qPCR (C) and IB (D) analyses were performed using the indicated primers and antibodies, respectively. **(E** and **F)** The specified cell lines with or without HIF-1α depletion and reconstituted HIF-1α expression were maintained in S/G(−) for 24 h. mRNA and protein levels were examined by qPCR (E) and IB (F), respectively. **(G** and **H)** The specified cell lines were maintained in complete media with or without $CoCl_2$ for 24 h. qPCR (G) and IB (H) analyses were performed using the indicated primers and antibodies, respectively. **(I)** Correlation analysis between *SHMT1* or *SHMT2* mRNA expression and *HIF1A* mRNA expression in GSE4290 (n = 180), GSE139533 (n = 155), and GSE141945 (n = 81). Data are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (C, E, G). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

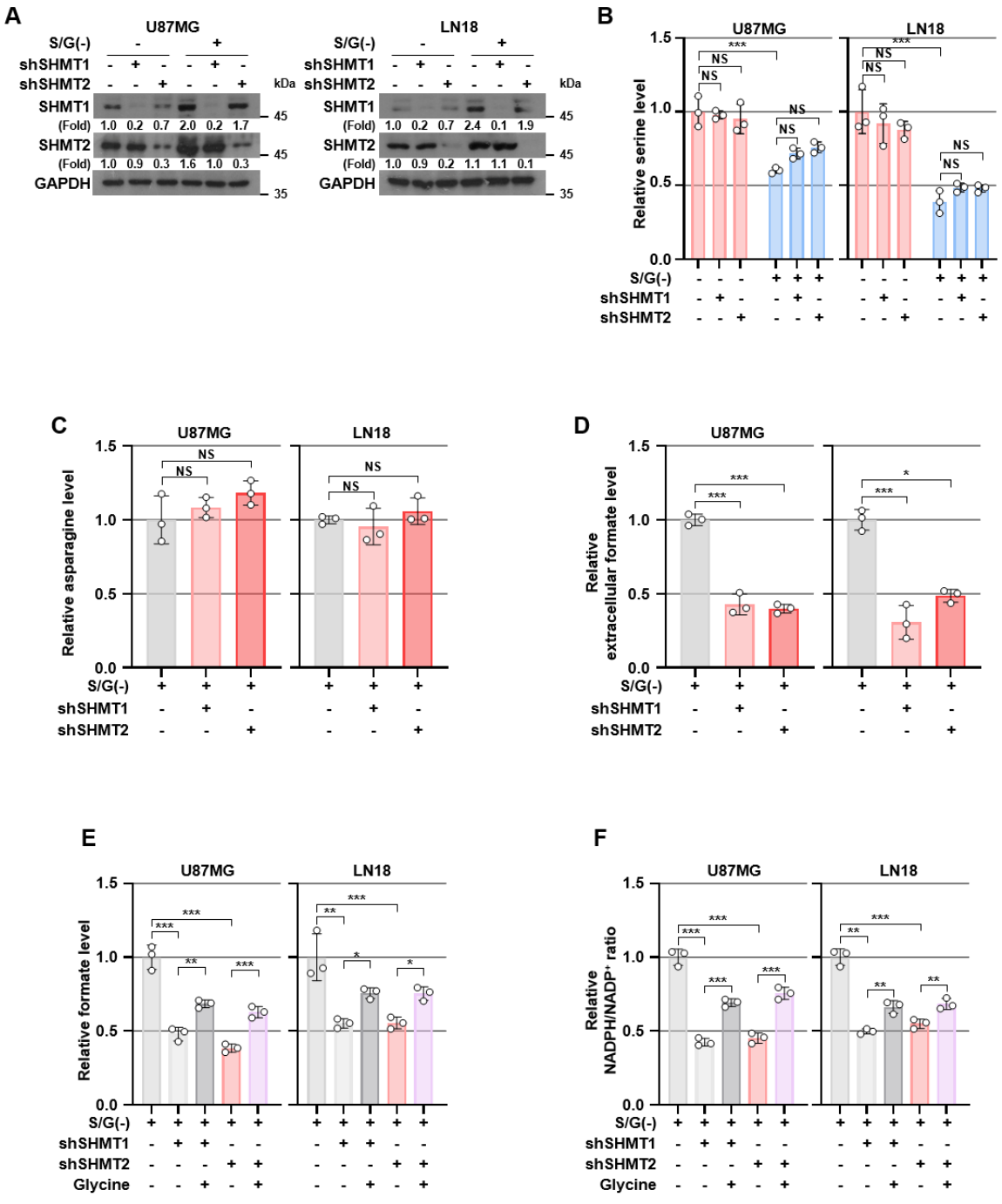


Figure S5 (related to Figure 5). HIF-1α–SHMT1/2-driven serine catabolism sustains redox homeostasis under S/G deprivation.

(A) The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in S/G(−) or control media for 24 h. IB analyses were performed using the indicated antibodies.

(B) The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in S/G(−) or control media for 24 h. Serine levels were measured. **(C and D)** The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in S/G(−) media for 24 h. Asparagine levels (C) and extracellular formate levels (D) were measured. **(E** and **F)** The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in S/G(−) for 24 h in the presence or absence of glycine (0.4 mM). Formate levels (E) and the $NADPH/NADP^+$ ratio (F) were measured. Data are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (B-F). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$. NS, not significant.

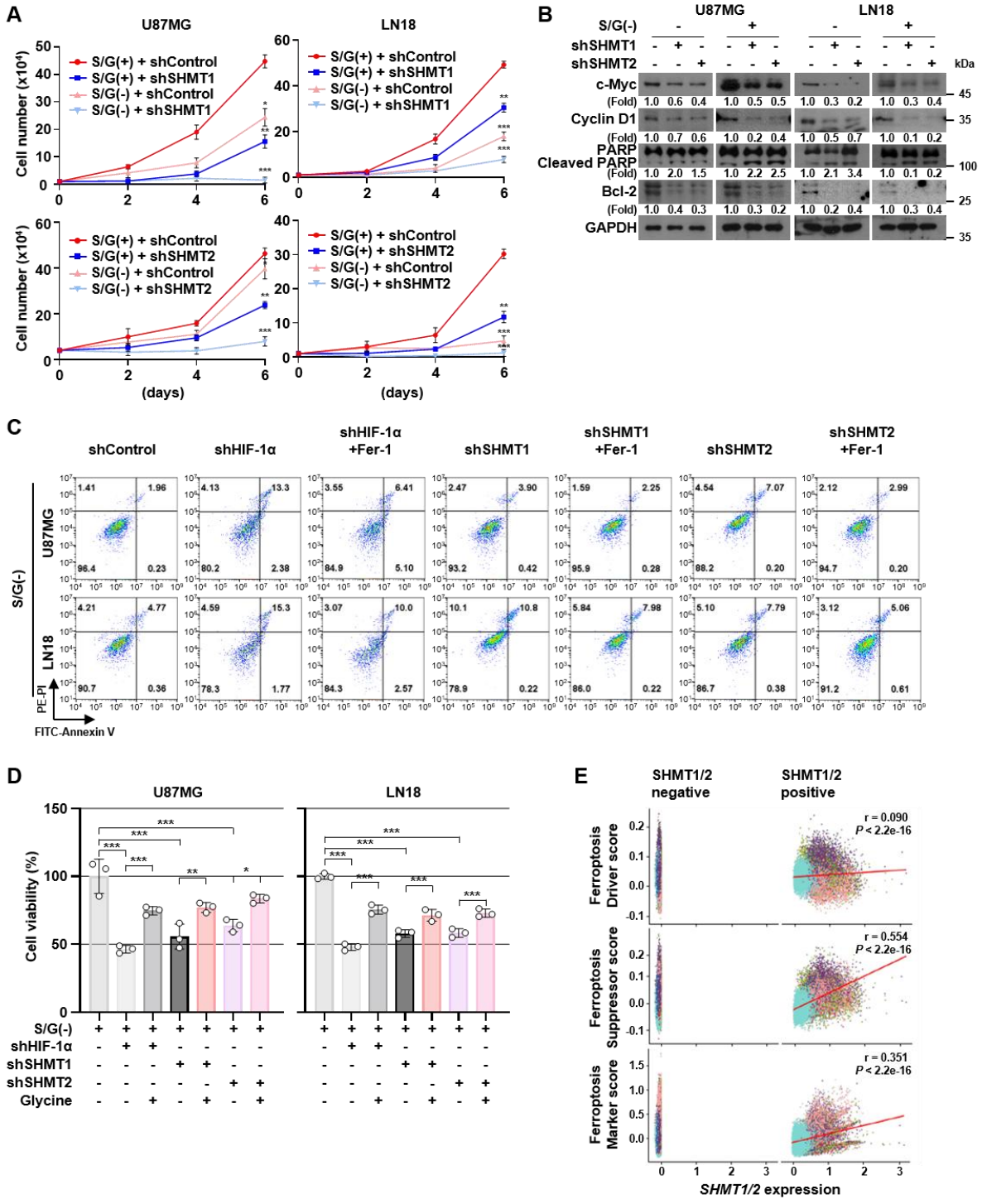
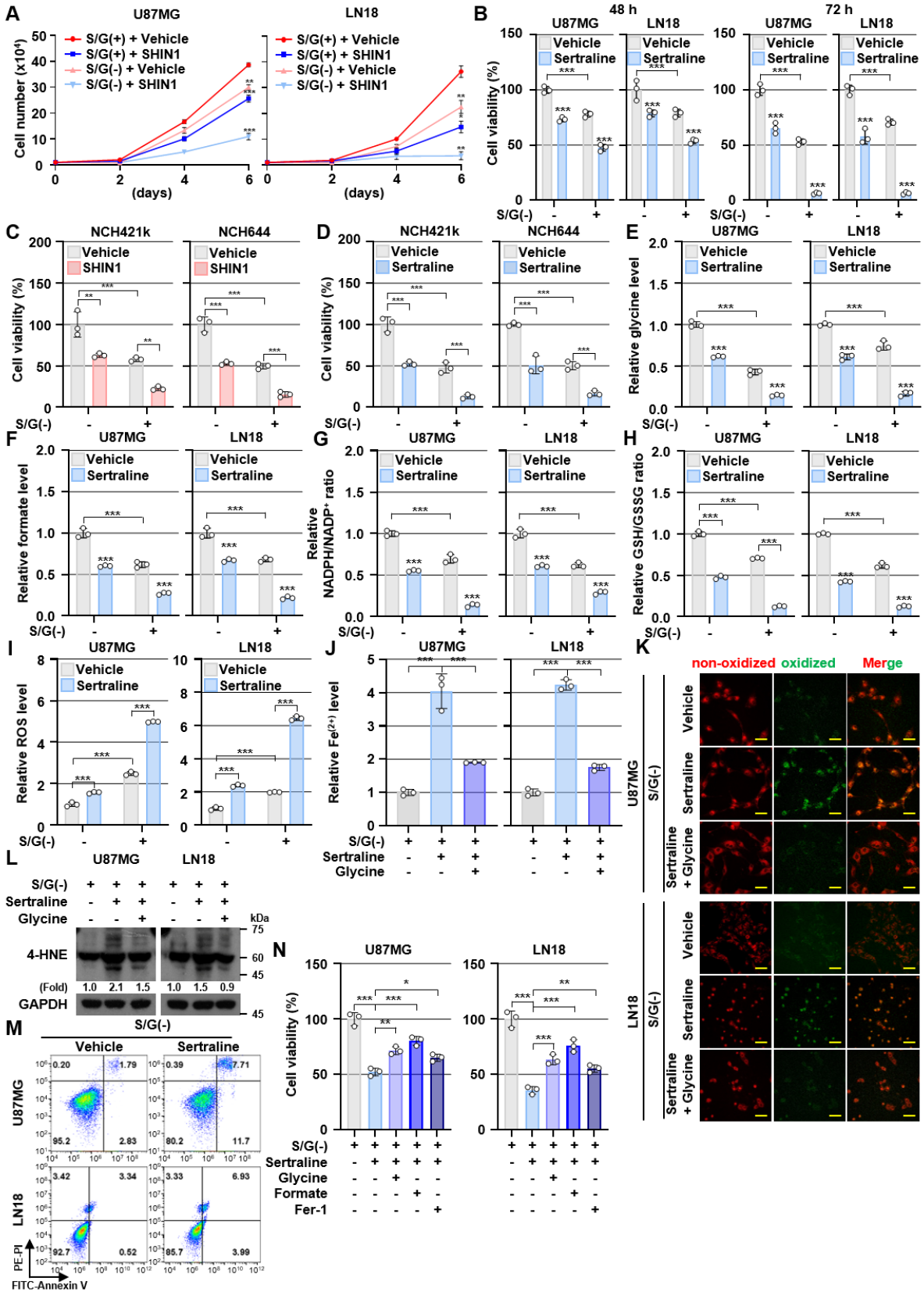


Figure S6 (related to Figure 6). HIF-1α–SHMT1/2-mediated serine catabolism supports ferroptosis resistance under S/G deprivation.

(A) The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in

S/G(−) or control media for specified time intervals and then harvested for cell counting. **(B)** The specified cell lines with or without SHMT1 or SHMT2 depletion were maintained in S/G(−) or control media for 48 h. IB analyses were performed using the indicated antibodies. **(C)** The specified cell lines with or without HIF-1α, SHMT1, or SHMT2 depletion were maintained in S/G(−) media for 48 h in the presence or absence of Fer-1 (5 μM). Apoptosis was analyzed by flow cytometry. **(D)** The specified cell lines with or without HIF-1α, SHMT1, or SHMT2 depletion were maintained in S/G(−) media for 96 h in the presence or absence of glycine (0.4 mM). Cell viability was assessed using the WST-8 assay. **(E)** Scatter plots showing the relationship between *SHMT1/2* expression and transcriptional module scores for ferroptosis-driver (top row), ferroptosis-suppressor (middle row), and ferroptosis-marker (bottom row) gene sets in SHMT1/2-negative and SHMT1/2-positive cells. Correlations were assessed using Pearson's correlation coefficient. Data are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (A, D). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.



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154 **Figure S7 (related to Figure 7). Pharmacological inhibition of SHMT disrupts redox**

homeostasis and sensitizes GBM cells to ferroptotic stress.

(A) The specified cell lines were maintained in S/G(−) or control media in the presence or absence of SHIN1 (500 nM) for the specified time intervals and then harvested for cell counting. **(B)** The specified cell lines were maintained in S/G(−) or control media for 48 or 72 h in the presence or absence of sertraline (3 μM), and cell viability was assessed. **(C** and **D)** The specified cell lines were cultured under S/G-deprived or control conditions for 72 h, with or without SHIN1 (500 nM) (C) or sertraline (3 μM) (D). Cell viability was assessed using the WST-8 assay. **(E-I)** The specified cell lines were cultured in S/G-deprived or control conditions for 24 h, with or without sertraline (3 μM). Glycine levels (E), formate levels (F), the NADPH/NADP+ ratio (G), the GSH/GSSG ratio (H), and ROS levels (I) were measured. **(J)** The specified cell lines were maintained in S/G(−) media in the presence or absence of sertraline (3 μM) or glycine (1 mM) for 24 h. Intracellular Fe^{2+} levels were measured. **(K)** The specified cell lines were maintained in S/G(−) media in the presence or absence of sertraline (3 μM) or glycine (1 mM) for 24 h, and stained with BODIPY C11. Representative fluorescence images showing changes in lipid ROS accumulation are shown. Scale bar, 60 μm. **(L)** The specified cell lines were maintained in S/G(−) media in the presence or absence of sertraline (3 μM) or glycine (1 mM) for 24 h. IB analyses were performed using the indicated antibodies. **(M)** The specified cell lines were maintained in S/G(−) media in the presence or absence of sertraline (3 μM) for 48 h. Apoptosis was analyzed by Annexin V/PI flow cytometry. Representative flow cytometry plots are shown. **(N)** The specified cell lines were cultured under S/G-deprived conditions for 96 h, with or without sertraline (3 μM), glycine (0.4 mM), formate (0.5 mM), or Fer-1 (5 μM). Cell viability was assessed using the WST-8 assay. Data

are presented relative to the untreated S/G(+) control group. Values represent mean ± s.d. of three independent experiments (A-J, N). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

Figure. 1

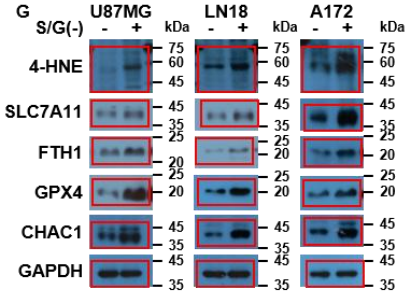
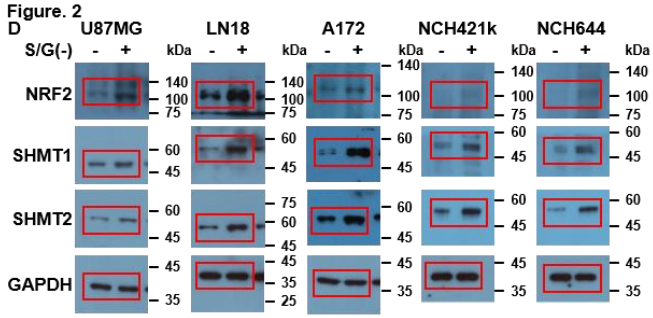


Figure. 2



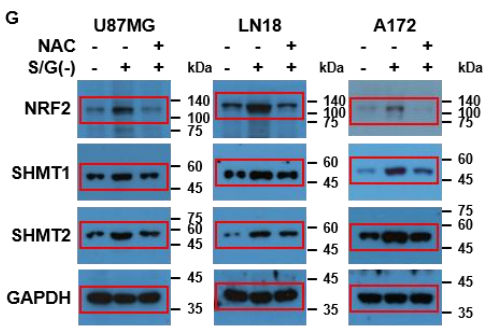


Figure. 3

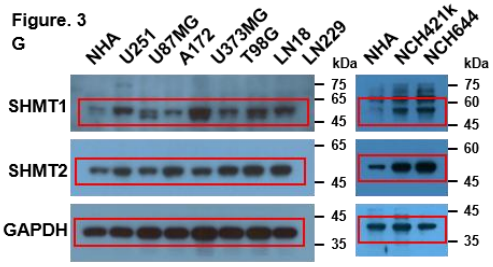


Figure. 4

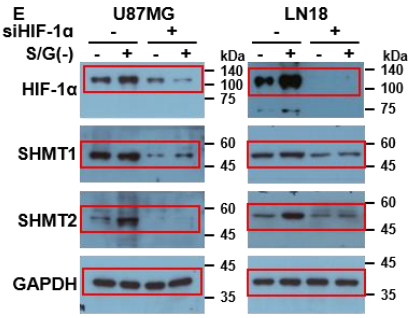


Figure. 6

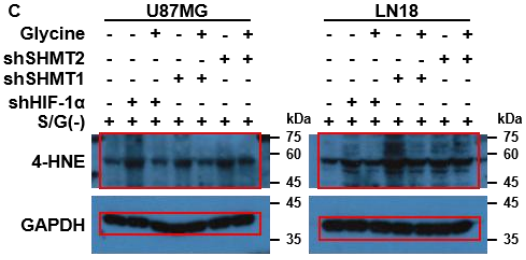


Figure. 7

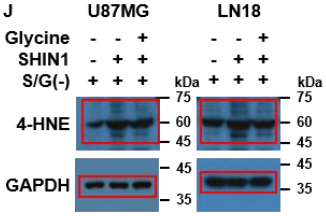


Figure. S1

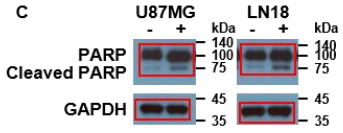


Figure. S2

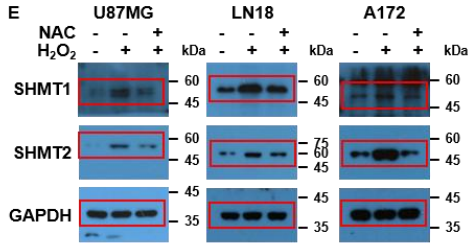


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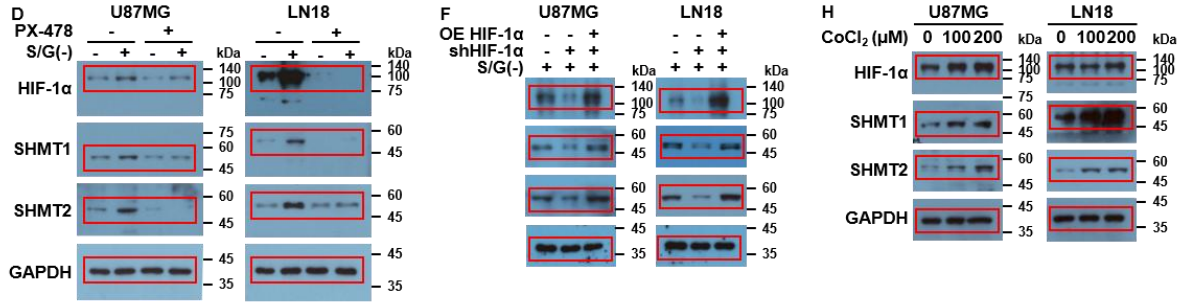


Figure. S5

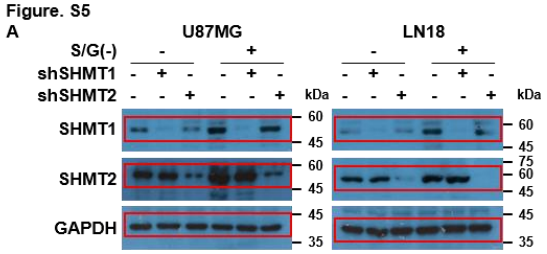


Figure. S6

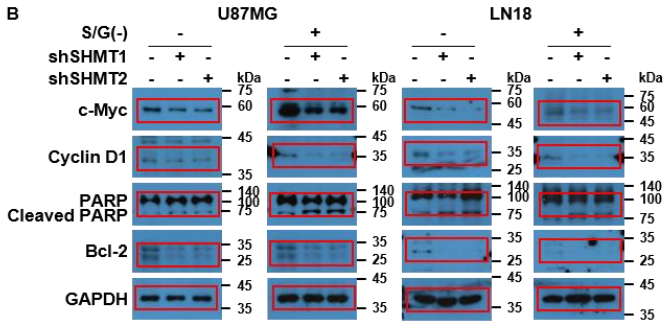
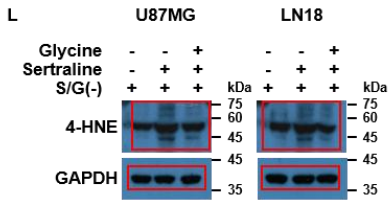


Figure. S7



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Figure S8. Uncropped immunoblots.

All uncropped immunoblot images are presented with lane identities and the regions corresponding to the cropped images shown in the main and supplementary figures clearly indicated. Red boxes denote the regions used for the corresponding cropped images. For each blot, target proteins and their corresponding loading controls were detected on the same membrane by sequential stripping and reprobing. The lane order is identical to that shown in the corresponding figure unless otherwise indicated.

Supplementary Tables

Table S1.

Antibodies used in this study.

Antibody	Source	Catalog No.	Dilution factors
Anti-SHMT1	Cell Signaling Technology, Danvers, MA, USA	41107	1:1,000 (IB) 1:100 (IHC)
Anti-SHMT2	Cell Signaling Technology, Danvers, MA, USA	33443	1:1,000 (IB) 1:100 (IHC)
Anti-c-Myc	Cell Signaling Technology, Danvers, MA, USA	18583	1:1,000 (IB)
Anti-Bcl-2	Cell Signaling Technology, Danvers, MA, USA	4223	1:1,000 (IB)
Anti-SLC7A11	Cell Signaling Technology, Danvers, MA, USA	12691	1:1,000 (IB)
Anti-FTH1	Cell Signaling Technology, Danvers, MA, USA	4393	1:1,000 (IB)
Anti-GAPDH	Cell Signaling Technology, Danvers, MA, USA	2118	1:1,000 (IB)
Anti-PARP	Cell Signaling Technology, Danvers, MA, USA	9542	1:1,000 (IB)
Anti-HIF-1α	Abcam, Cambridge, UK	ab51608	1:1,000 (IB) 1:100 (IHC)
Anti-GPX4	Abcam, Cambridge, UK	ab125066	1:1,000 (IB)
Anti-Ki-67	Abcam, Cambridge, UK	ab16667	1:100 (IHC)
Anti-4-HNE	Abcam, Cambridge, UK	ab48506	1:1,000 (IB) 1:250 (IHC)
Anti-NRF2	Proteintech, IL, USA	80593-1-RR	1:2,000 (IB)
Anti-CHAC1	Proteintech, IL, USA	15207-1-AP	1:2,000 (IB)
Anti-cyclin D1	Santa Cruz Biotechnology, CA, USA	sc-8396	1:1,000 (IB)
Goat anti-rabbit IgG, HRP	BioActs, Incheon, Republic of Korea	RSA1221	1:3,000 (IB)
Goat anti-mouse IgG, HRP	BioActs, Incheon, Republic of Korea	RSA1122	1:3,000 (IB)

Table S2.

Primers used for qPCR.

Each qPCR assay was performed in triplicate, and gene expression levels were normalized to *GAPDH*.

Primer	Forward sequence (5'-3')	Reverse sequence (5'-3')
Human *SHMT1*	CTGGCACAACCCCTCAAAGA	AGGCAATCAGCTCCAATCCAA
Human *SHMT2*	CCCTTCTGCAACCTCACGAC	TGAGCTTATAGGGCATAGACTCG
Human *SLC7A11*	TCTCCAAAGGAGGTTACCTGC	AGACTCCCCTCAGTAAAGTGAC
Human *SOD2*	GGCCTACGTGAACAACCTGAA	CTGTAACATCTCCCTTGGCCA
Human *FTH1*	CCATCAACCGCCAGATCAACCTG	GTTTCTCAGCATGTTCCCTCTCCTC
Human *GPX4*	AGAGATCAAAGAGTTCGCCGC	TCTTCATCCACTTCCACAGCG
Human *CHAC1*	AAGATGCTCCTGACCAACCA	CCACAGAGCTGCATGAAGTC
Human *PTGS2*	CTGCGCCTTTTCAAGGATGG	CCCCACAGCAAACCGTAGAT
Human *GAPDH*	GCATCTTCTTTTGCGTCG	TGTAAACCATTGTAGTTGAGGT

Table S3.

Primers used for ChIP-qPCR.

Each qPCR assay was performed in triplicate.

Primer	Forward sequence (5'-3')	Reverse sequence (5'-3')
Human *SHMT1*	TTCAAGCGATTCTCTTGCCT	ATTTTTAGTAGAGACGGGGT
Human *SHMT2*	CCTTAGTTCCTAGTCACAAA	GTTAACTGCGAGCCTGTCCT

Table S4.

List of response to oxidative stress genes differentially regulated under S/G-restricted conditions.

Upregulated genes	Fold change	Downregulated genes	Fold change
SLC7A11	7.568	ECT2	0.761
STC2	4.407	CDK1	0.758
SESN2	3.626	SCARA3	0.752
ATF4	2.631	AXL	0.749
HMOX1	2.358	NUDT1	0.751
TP53INP1	1.384	NET1	0.747
MSRB2	1.379	PXN	0.712
HSPA1B	1.349	MAPK8	0.699
TXN	1.357	COL1A1	0.655
STAT1	1.341	PRR5L	0.545
TRPA1	2.152	HGF	0.491
CYP1B1	2.296		
IDH1	2.136		
NQO1	1.860		
PYCR1	1.759		
MGST1	1.341		
GPX4	1.331		
SIRPA	1.301		

SRXN1	1.768
NCOA7	1.733
LONP1	1.682
PTPRN	1.648
ANKZF1	1.598
MMP14	1.586
GCLC	1.553
SOD2	1.555
DDR2	1.494
MMP2	1.512
NAGLU	1.482
PRDX5	1.503
G6PD	1.479
MAP1LC3A	1.509
TRIM25	1.445

Gene symbol	Full name
SLC7A11	Solute Carrier Family 7 Member 11
STC2	Stanniocalcin 2
SESN2	Sestrin 2
ATF4	Activating Transcription Factor 4
HMOX1	Heme Oxygenase 1
TP53INP1	Tumor Protein p53 Inducible Nuclear Protein 1

MSRB2	Methionine Sulfoxide Reductase B2
HSPA1B	Heat Shock Protein Family A (Hsp70) Member 1B
TXN	Thioredoxin
STAT1	Signal Transducer and Activator of Transcription 1
TRPA1	Transient Receptor Potential cation channel subfamily A member 1
CYP1B1	Cytochrome P450 Family 1 Subfamily B Member 1
IDH1	Isocitrate Dehydrogenase (NADP(+)) 1
NQO1	NAD(P)H Quinone Dehydrogenase 1
PYCR1	Pyrroline-5-Carboxylate Reductase 1
MGST1	Microsomal Glutathione S-Transferase 1
GPX4	Glutathione Peroxidase 4
SIRPA	Signal Regulatory Protein Alpha
SRXN1	Sulfiredoxin 1
NCOA7	Nuclear Receptor Coactivator 7
LONP1	Lon Peptidase 1, Mitochondrial
PTPRN	Protein Tyrosine Phosphatase Receptor Type N
ANKZF1	Ankyrin Repeat and Zinc Finger Peptidyl tRNA hydrolase 1
MMP14	Matrix Metallopeptidase 14
GCLC	Glutamate-Cysteine Ligase Catalytic Subunit
SOD2	Superoxide Dismutase 2
DDR2	Discoidin Domain Receptor Tyrosine Kinase 2
MMP2	Matrix Metallopeptidase 2

NAGLU	N-Acetyl-Alpha-Glucosaminidase
PRDX5	Peroxiredoxin 5
G6PD	Glucose-6-Phosphate Dehydrogenase
MAP1LC3A	Microtubule Associated Protein 1 Light Chain 3 Alpha
TRIM25	Tripartite Motif Containing 25
ECT2	Epithelial Cell Transforming 2
CDK1	Cyclin Dependent Kinase 1
SCARA3	Scavenger Receptor Class A Member 3
AXL	AXL Receptor Tyrosine Kinase
NUDT1	Nudix Hydrolase 1
NET1	Neuroepithelial Cell Transforming 1
PXN	Paxillin
MAPK8	Mitogen-Activated Protein Kinase 8
COL1A1	Collagen Type I Alpha 1 Chain
PRR5L	Proline Rich 5 Like
HGF	Hepatocyte Growth Factor

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