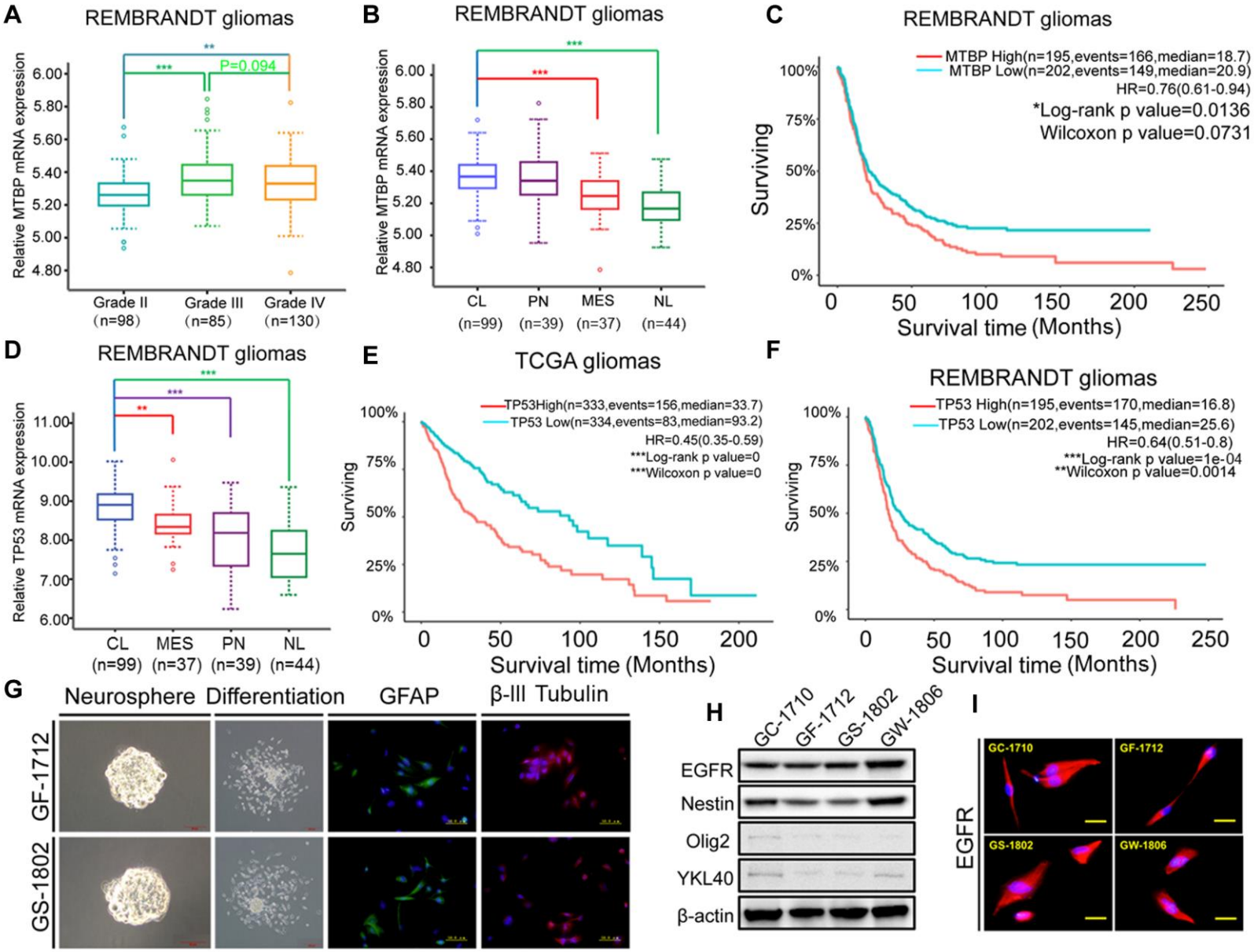
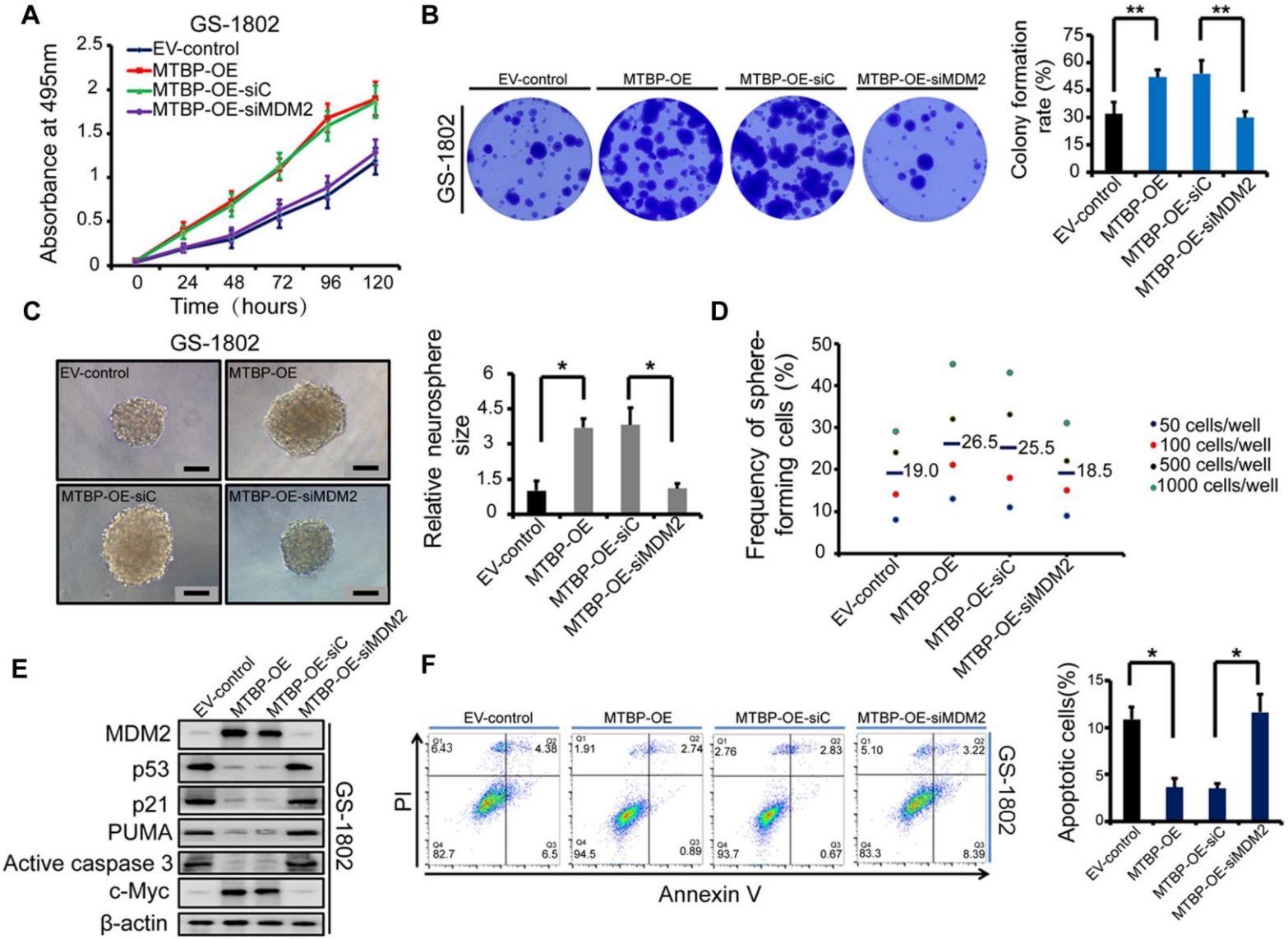
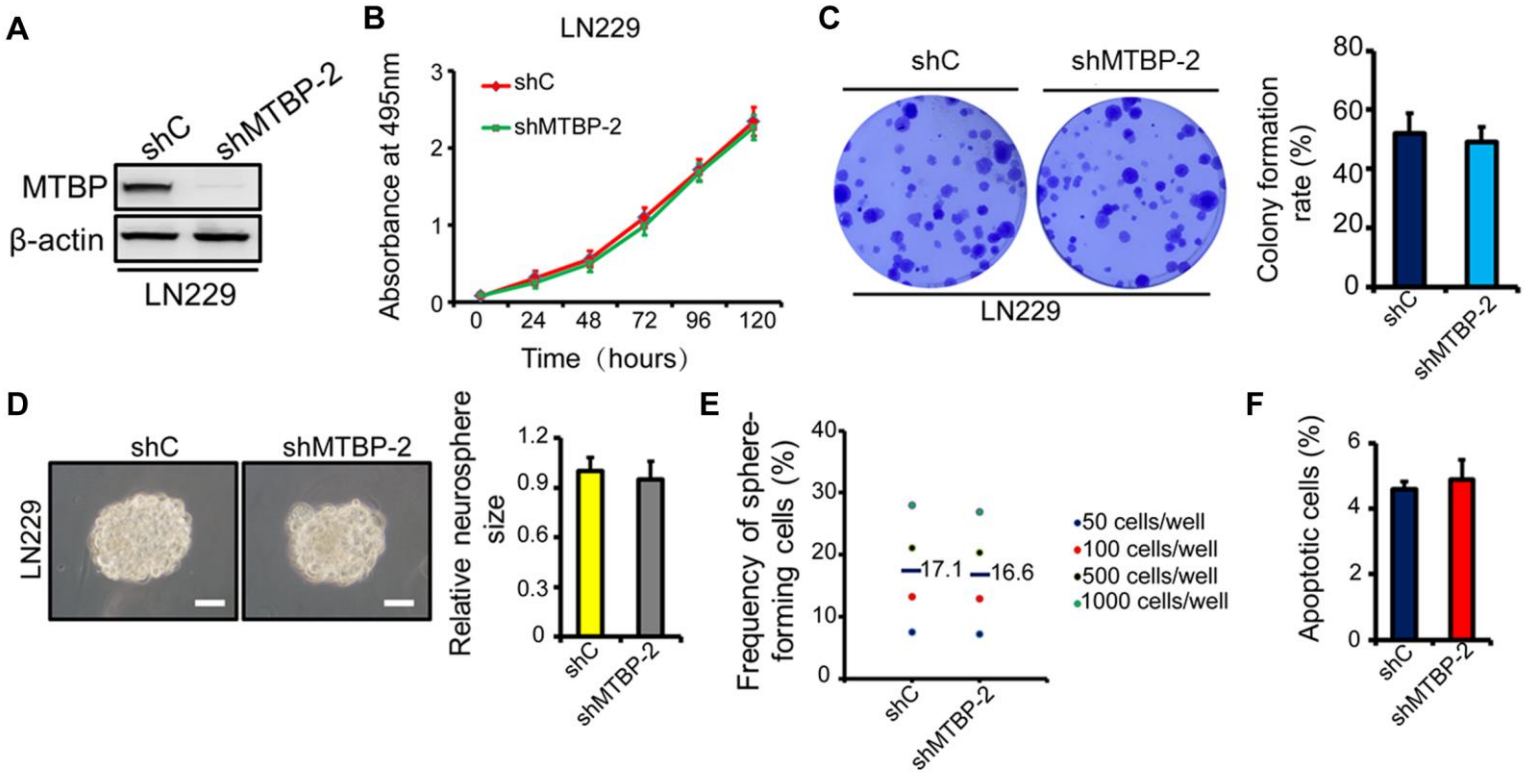


Supplementary Figure 1



Supplementary Figure 2



Supplementary Figure 3

Supplementary Figure Legends

Supplementary Figure 1. Influence of pathological and molecular glioma features on MTBP and TP53 expressions. A: Influence of REMBRANDT glioma histopathologic grades on MTBP mRNA expression. B: Influence of REMBRANDT glioma molecular subtypes on MTBP mRNA expression. C: Prognostic significance of MTBP in REMBRANDT gliomas. D: Influence of REMBRANDT glioma molecular subtypes on TP53 mRNA expression. E and F: The prognostic role of TP53 mRNA expression in TCGA (E) and REMBRANDT (F) gliomas. G: Multi-lineage differentiation capacity of GSCs. H and I: The expression of classical markers (EGFR and Nestin), proneural marker (Olig2) and mesenchymal marker (YKL40) was examined in GC-1710, GF-1712, GS-1802, and GW-1806 cells using western blot (H) or immunofluorescence (I).***P < 0.001, **P < 0.01. Classical, CL; neural, NL; proneural, PN; and mesenchymal, MES.

Supplementary Figure 2. Dependence of the MTBP pro-survival effect on the expression of MDM2 in TP53wt GBM cells. A: Effect of MDM2 silencing on MTBP-induced proliferation of GS-1802 cells. B: Effect of MDM2 knockdown on MTBP-induced colony formation in GS-1802 cells as determined by soft agar colony assays. C: Representative images of GS-1802 neurospheres transduced with indicated plasmids (left) and quantification of relative neurosphere sizes of indicated GSCs (right). Scale bar: 50 μm. D: Effect of MDM2 silencing on MTBP-induced *in vitro* clonogenicity of GS-1802 GSCs. E: Effect of MDM2 silencing on the MTBP-induced downregulation of p53, p21, PUMA, and active caspase3 and upregulation of c-myc in GS-1802 cells as determined by western blot analyses. F: Effect of MDM2 knockdown on the apoptosis of MTBP-overexpressing GS-1802 cells. Results are presented as mean ± SEM of triplicate samples from three independent experiments. *P < 0.05, **P < 0.01.

Supplementary Figure 3. Effect of MTBP silencing on the growth of TP53mut GBM cells. A: Western blotting analysis of TP53mut LN229 cells transduced with shRNA targeting MTBP (shMTBP-2) or a control shRNA (shC). B: Effect of MTBP knockdown by shRNA on cell viability in LN229 cells. C: Effect of MTBP knockdown on colony formation in LN229 cells, as assessed by soft agar colony assays. D: Representative images of LN229 neurospheres transduced with shMTBP-2 or shC (left) and quantification of relative neurosphere sizes of indicated GSCs (right). Scale bar: 50 μm. E: Effect of MTBP silencing on the clonogenicity of LN229 GSCs, as determined by limiting dilution neurosphere formation assays. F: Effect of MTBP silencing on the apoptosis of LN229 cells. Results are presented as mean ± SEM of triplicate samples from three independent experiments.

Supplementary Table 1. Clinical information of the primary glioma cells

	GC-1710	GF-1712	GS-1802	GW-1806
Gender	Male	Female	Male	Female
Age	68 years old	72 years old	67 years old	48 years old
Location	Right temporal lobe	Right occipital lobe	Left temporal lobe	Left frontal lobe
Pathological diagnosis	Glioblastoma	Glioblastoma	Glioblastoma	Glioblastoma
WHO grade	IV	IV	IV	IV
Ki-67	15% (+)	30% (+)	30% (+)	40% (+)
TP53	Wildtype	Wildtype	Wildtype	Wildtype
1p/19q	Non-codeletion	Non-codeletion	Non-codeletion	Non-codeletion
IDH1	Wildtype	Wildtype	Wildtype	Wildtype
IDH2	Wildtype	Wildtype	Wildtype	Wildtype
TERT	Mutant	Mutant	Mutant	Mutant
MGMT promoter	methylated	unmethylated	unmethylated	unmethylated