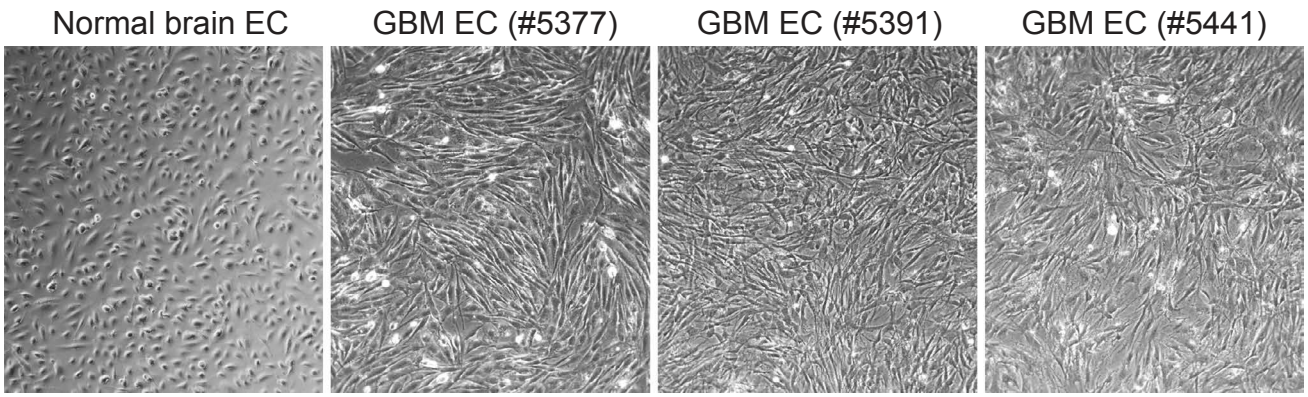
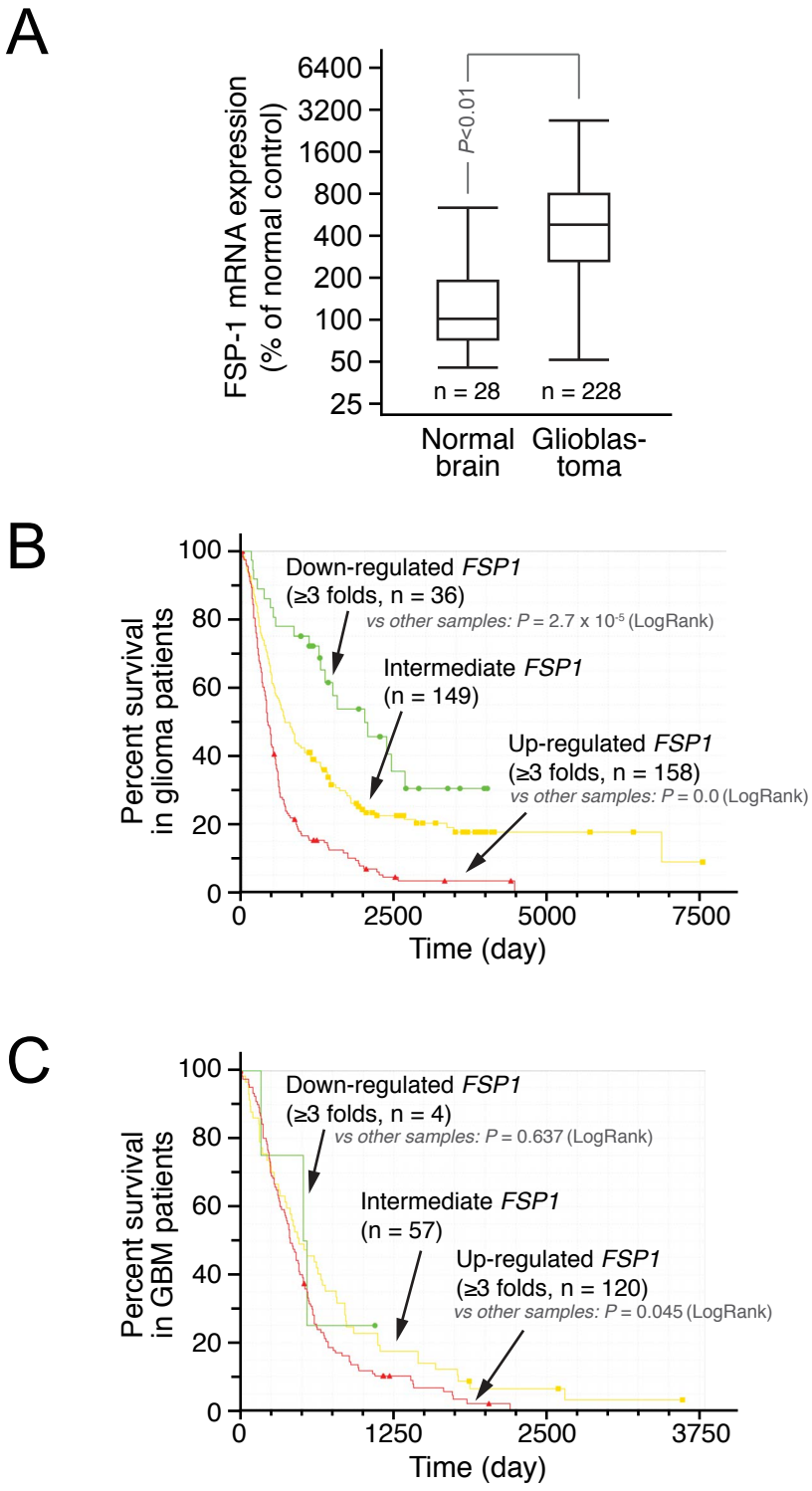
Patient #	Specimen Tumor Type (histopathology)	Tumor Location (Laterality)	Recurrence	Gender	Ethnicity	Age
5377	Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Right)		Male	Caucasian	70
5391	Glioblastoma, WHO Grade IV	Cerebrum-Parietal (Left)		Female	Caucasian	51
5433	Residual Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Right)		Male	Caucasian	71
5441	Multicentric Glioblastoma, WHO Grade IV	Cerebrum-Temporal (Right)		Female	Caucasian	56
5453	Recurrent Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Left)	3rd	Male	Caucasian	48
5460	Recurrent Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Left)	3rd	Male	Caucasian	61
5465	Oligodendroglioma, WHO grade II	Cerebrum-Temporal (Left)		Female	Caucasian	23
5467	Recurrent Glioblastoma, WHO Grade IV	Cerebrum-Temporal (Left)	1st	Male	Caucasian	76
5573	Glioblastoma, WHO Grade IV	Cerebrum-Temporal (right)		Male	Unknown	75
5581	Glioblastoma, WHO Grade IV with sarcomatous features	Cerebrum-Temporal (Right)		Male	Caucasian	53
5584	Recurrent Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Left)	1st	Female	Caucasian	49
5597	Recurrent Glioblastoma, WHO Grade IV	Cerebrum-Parietal (Right)	3rd	Male	Caucasian	57
5654	Glioblastoma, WHO Grade IV	Cerebrum-Frontal (Right)		Female	Caucasian	49
5659	Glioblastoma, WHO Grade IV	Cerebrum-Temporal (Left)		Female	Caucasian	83
5785	Glioblastoma, WHO Grade IV	Cerebrum-Left(Parietal)		Male	Caucasian	71

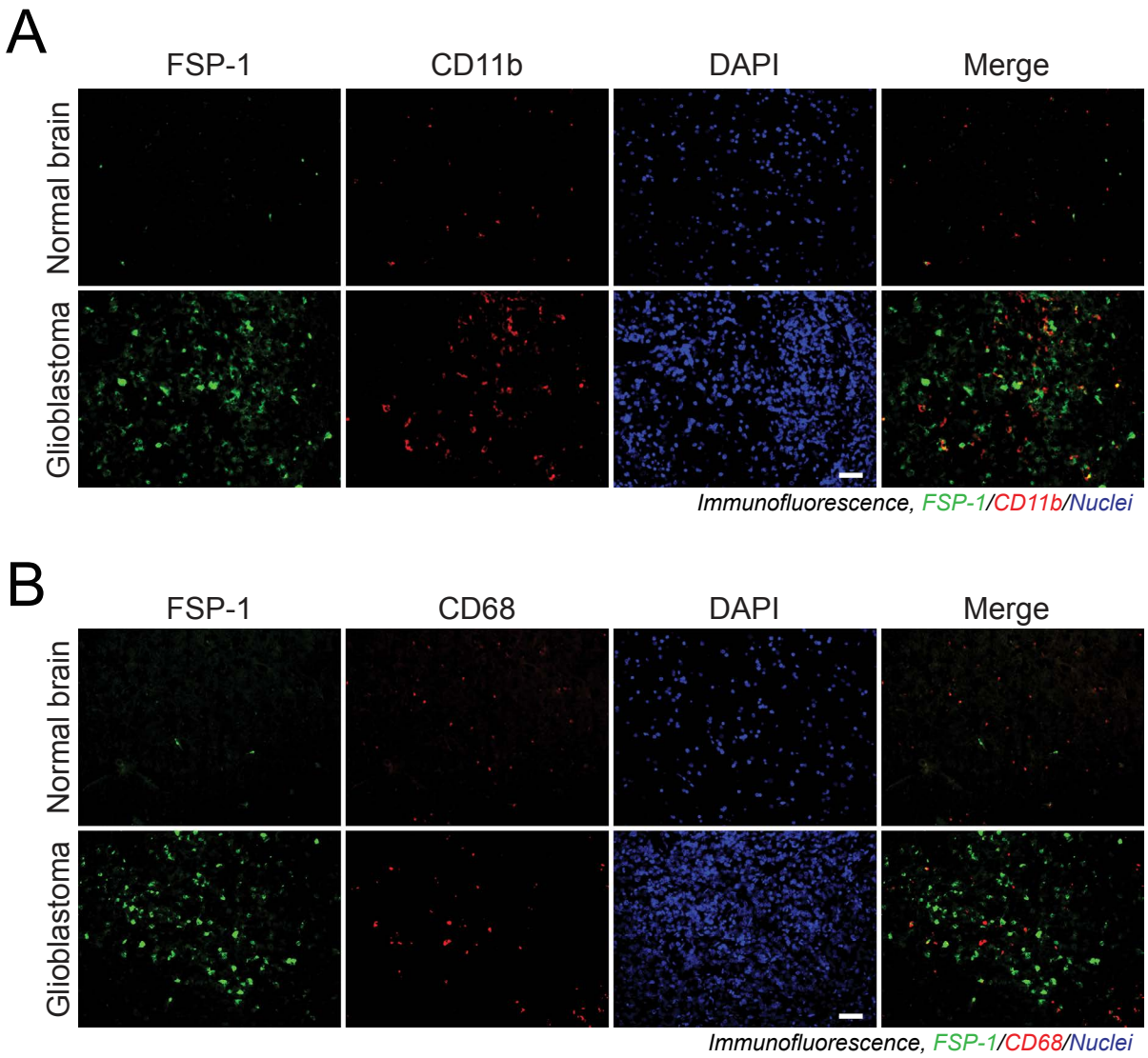
Supplemental Table S1. Patients enrolled in this study. Patients were treated at the Department of Neurosurgery, University of Pennsylvania. The surgical tissue was collected by the tissue bank.



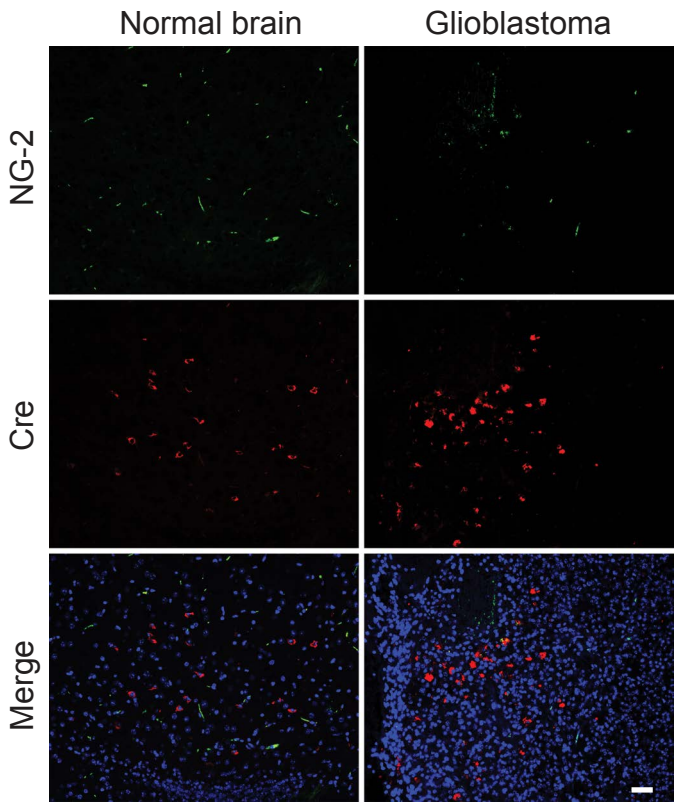
Supplemental Figure 1. Patient GBM tumor-derived ECs exhibit altered morphology.
ECs were isolated from single-cell tumor suspensions by CD31 antibody-based magnetic-activating cell sorting (MACS). Cells were cultured and imaged. Representative data are shown from 2 independent experiments.



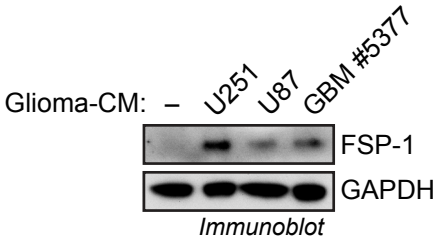
Supplemental Figure 2. FSP-1 expression is increased in glioblastoma and correlates with poor patient survival in humans.
Rembrandt database of the National Cancer Institute was analyzed (http://caintegrator.nci.nih.gov/rembrandt). (**A**) FSP-1 (S100A4) mRNA expression analysis in normal brain and glioblastoma tissue. Results are shown as box plots representing median, 25th and 75th percentiles as boxes, and the range of data as bars (total n = 256 patients). (**B**,**C**) Analysis of survival rate in glioma and GBM patients with different FSP-1 mRNA expression levels, intermdiate and upregulated (total n = 343 patients with glioma and 181 patients with GBM).



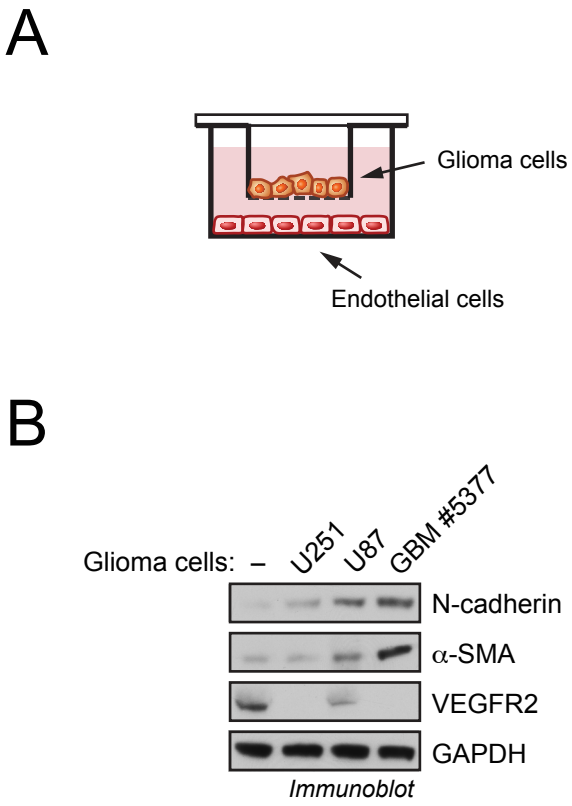
Supplemental Figure 3. FSP-1 is poorly colocalized with macrophage markers CD11b and CD68 in normal brain and glioblastoma tissues.
Sections from biopsy specimens from subjects with glioblastoma (n = 5 samples) and from normal brains (n = 4 samples) were probed with anti-FSP-1, (**A**) anti-CD11b, and (**B**) anti-CD68 antibodies. Representative images are shown. Representative data are shown from 2 independent experiments. Scale bar: 100 μm.



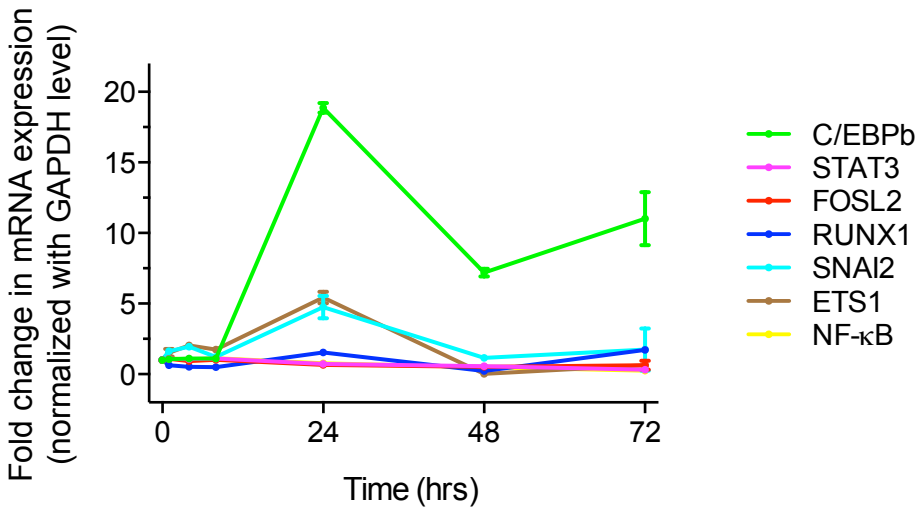
Supplemental Figure 4. Cre is poorly colocalized with pericyte marker NG-2 in Tie2-Cre mice bearing GBM tumor. GL26 glioma cells were orthotopically injected to Tie2-Cre mice (n = 5 mice). The brain sections were probed with anti-NG-2 and anti-Cre antibodies. Representative images are shown from 2 independent experiments. Scale bar: 100 μm.



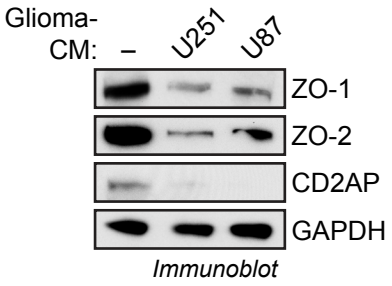
Supplemental Figure 5. Different glioma-CM induce FSP-1 expression in ECs.
Human brain microvascular ECs were treated with different glioma CM that were harvested from U251, U87 and GBM patient #5377 cells. Cells were lyzed and subjected to immunoblot analysis. Representative data are shown from 3 independent experiments.



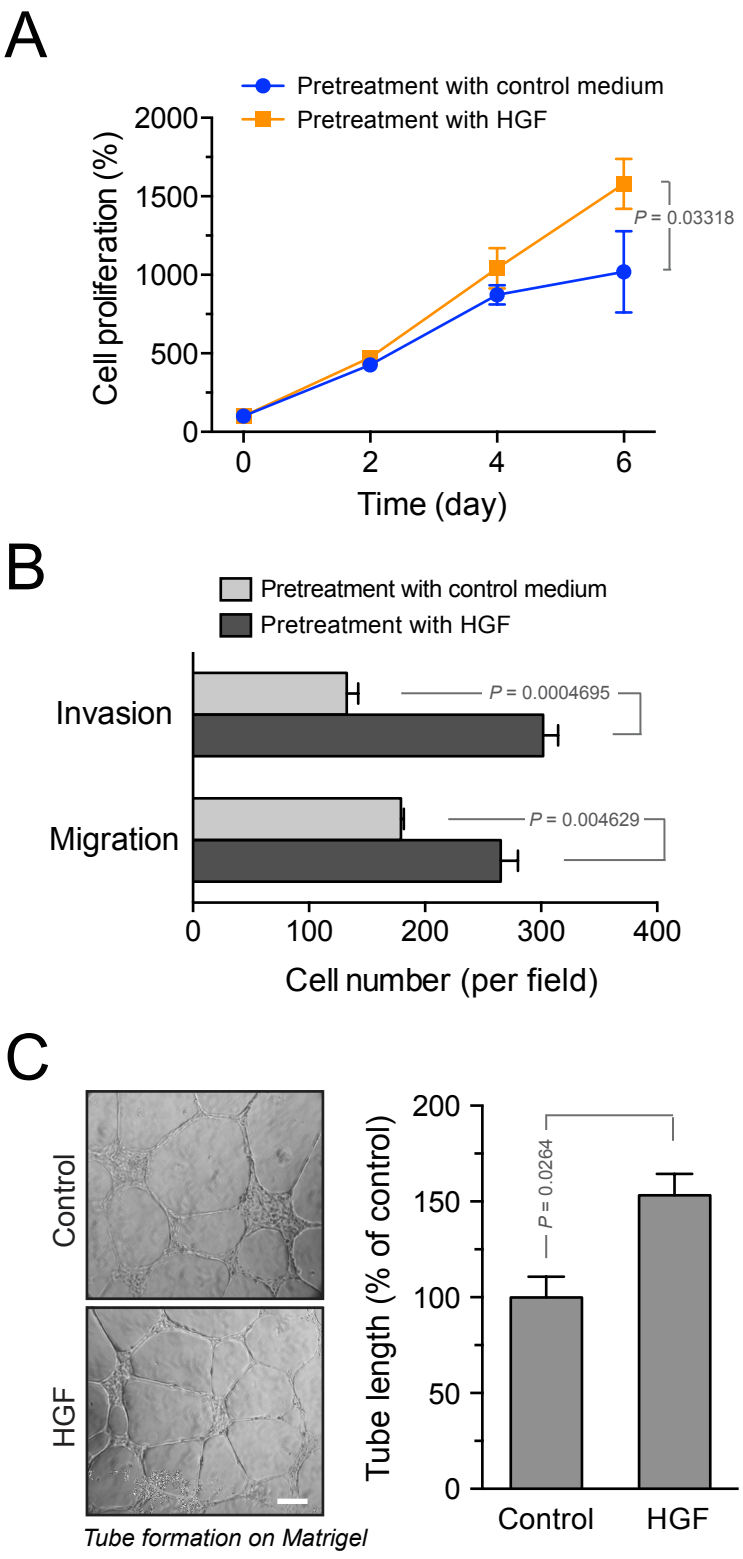
Supplemental Figure 6. Co-culture with glioma cells induces Endo-MT in ECs.
Human brain microvascular ECs were co-cultured in a transwell system with human U251 or U87 glioma cells or primary tumor cells isolated from patient #5377 for 3 days. (**A**) Schematic approach. (**B**) ECs were lyzed and subjected to immunoblot analysis. Representative data are shown from 2 independent experiments.



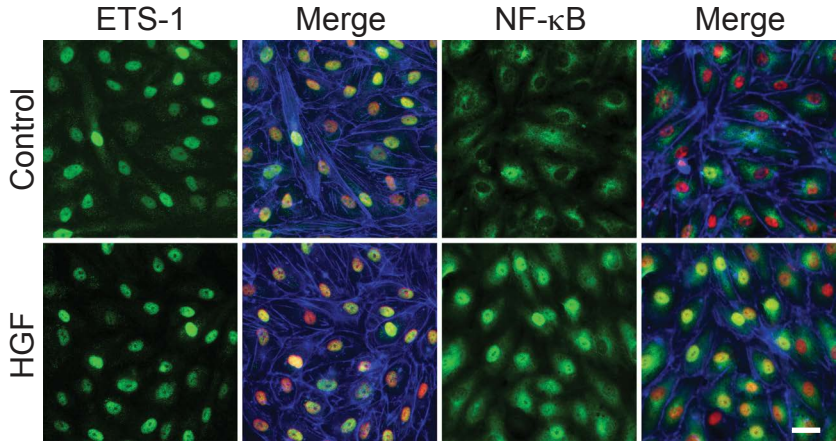
Supplemental Figure 7. Glioma-CM induces time-dependent expression of transcriptional factors.
Human brain microvascular ECs were treated with glioma-CM for different time. mRNA was isolated and subjected to real-time RT-PCR analysis (mean ± SD, n = 3).



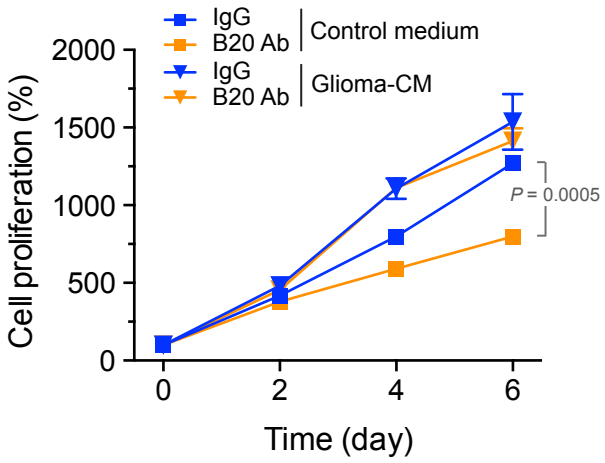
Supplemental Figure 8. Glioma-CM downregulates expression of junction proteins in ECs.
Human brain microvascular ECs were treated with difference glioma-CM or control medium for 24 hrs. Cells were lyzed and subjected to immunoblot analysis. Representative data are shown from 2 independent experiments.



Supplemental Figure 9. HGF induces vascular abnormalities *in vitro*.
Human brain microvascular ECs were treated with 50 ng/ml HGF for 3 dyas, and cultured in normal culture medium. (**A**) Cell proliferation was determined by MTT assay (mean ± SEM, n = 3, paired *t* test). (**B**) Cells were seeded on transwell membranes that were pre-coated with and without Matrigel, for invasion and migration assays, respectively. Cell invasion and migration were induced on induced by addition of 10% FBS in the bottom chamber, followed by incubation for 24 hrs (invasion assay) and 4 hrs (migration assay). Cells on the upper membranes were removed, and invaded and migrated cells were stained and counted (mean ± SEM, n = 3, paired *t* test). (**C**) Tube formation was induced on matrigel. Left, representative images. Right, quantified total tube length (mean ± SEM, n = 3, paired *t* test). Scale bar: 200 μm.



Supplemental Figure 10. HGF induces ETS-1 and NF-κB translocalization to nuclei.
Human brain microvascular ECs were treated with 100 ng/ml HGF. Cells were fixed and stained with anti-ETS-1 and anti-NF-κB antibodies, followed by immunofluorescence analysis (bar: 20 μm). Representative data are shown from 2 independent experiments.



Supplemental Figure 11. Glioma-CM induces treatment resistance to anti-VEGF antibody in ECs.
Human brain microvascular ECs were pre-treated with U251 glioma-CM or control normal medium for 1 day, and cultured in normal culture medium. Anti-VEGF B20 antibody or control IgG (20 μg/ml) were added in the culture medium. Cell proliferation was determined (mean ± SEM, n = 3, paired *t* test).