

Figure W1. c-Met and HGF mRNA expression are upregulated in the Mes GBM subtype. (A) Verhaak et al. [12] classified GBM tumors into four subtypes (Mes, mesenchymal; PN, proneural; Nrl, neural; CI, classical), and c-Met's expression per GBM was obtained from the TCGA database ($n = 200$; Tukey box plot; t test: $***P < .0001$, $*P < .05$). (B) HGF expression for GBM tumors in A was extracted from the TCGA database ($n = 200$; Tukey box plot; t test: $***P < .0001$, $**P < .005$, $*P < .05$). (C) Affymetrix (U133A format) gene expression data of GBMs from the National Cancer Institute (NCI) REMBRANDT database (<https://caintegrator.nci.nih.gov/rembrandt/>) was downloaded on 10 May 2011. Gene expression values were downloaded as \log_2 transformed, median centered, quantile data, which were normalized using the Robust MultiChip Average with a custom Chip Definition File, thereby removing unreliable probe set data. Tumors were assigned to GBM subtypes by z-score normalizing their highest average metagene scores according to the gene lists described by Verhaak et al. [12] and their c-Met expression reported ($n = 180$; Tukey box plot; t test: $***P < .0001$). (D) HGF expression was obtained from the REMBRANDT database for each GBM as determined in C ($n = 180$; Tukey box plot; t test: $***P < .0001$, $**P < .005$). (E) GBMs from the TCGA database were classified as either Mes, proliferative (Prolif), or PN based on each tumor's highest average z-score corrected metagene score from subtype-specific gene lists defined by Phillips et al. [3]. c-Met's expression per GBM was downloaded from the TCGA database ($n = 495$; Tukey box plot; t test: $***P < .0001$, $**P < .005$). (F) GBM tumors were classified as in E, and HGF expression was documented per tumor ($n = 495$; Tukey box plot; t test: $**P < .005$, $*P < .05$).

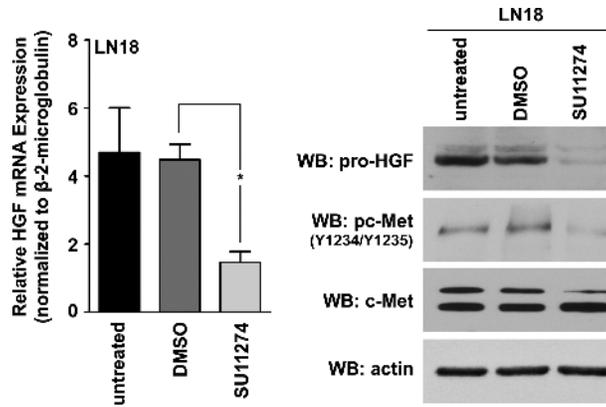


Figure W2. Inhibition of c-Met activity in LN18 GBM cells attenuates HGF expression. Left panel: Quantitative real-time PCR analysis showing inhibition of HGF mRNA expression in LN18 cells after treatment with 10 μ M SU11274 for 16 hours in Dulbecco's modified Eagle's medium containing 10% FBS ($P < .05$; t test; $n = 3$; triplicate samples per experiment). Right panel: Corresponding Western blot of HGF and pc-Met (Y1234/Y1235) levels after 16-hour treatment with 10 μ M SU11274.

Table W1. Short Tandem Repeat Fingerprinting of U87 and LN18 Cells.

Locus	U87		LN18	
	1	2	1	2
AMEL	105.92	0	110.96	105.81
D3s 1358	134.41	130.52	130.8	126.97
TH01	177.34	0	174.95	0
D13s 317	188.58	176.81	197.34	193.23
D8s 1179	217.23	213.18	230.4	222.22
D7s 820	225.18	221.12	222.03	0
TPOX	268.73	0	269.68	0
D16s 539	294.75	0	300.33	291.74
D18s 51	306.12	0	330.76	322.77
CSF1PO	341.37	337.1	346.35	0
Penta D	433.25	408.85	418.52	0
Penta E	433.77	396.6	412.9	396.98

Cells that were used in preparation of this manuscript were analyzed for their specific marker allele content using the GenomeLab Human STR Primer Set (Beckman Coulter, Indianapolis, IN). Cell line isolates may vary between different laboratories, and therefore, these data may be used by other investigators for comparative purposes.

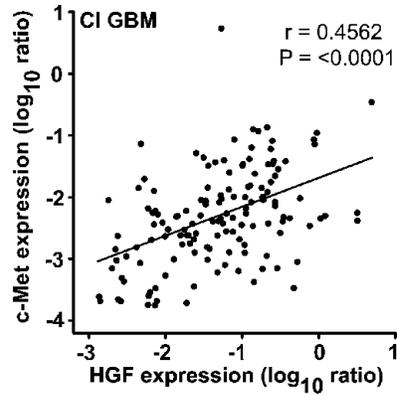


Figure W3. HGF and c-Met expression correlate in CI GBM. Four hundred ninety-five GBMs were classified into subtypes using gene expression lists defined by Verhaak et al. [12]. The highest average z-score corrected metagene score was used to establish each tumor’s subtype. Level 3 HGF and c-Met mRNA expression data were obtained from the TCGA database, and their expression levels were correlated in CI GBMs using Spearman correlation ($n = 141$; $r = 0.4562$; $P < .0001$).

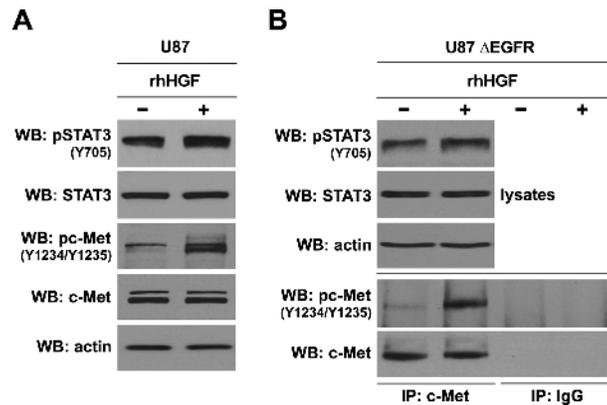


Figure W4. HGF stimulation of U87 and U87 Δ EGFR-expressing cells enhances the activity of STAT3. (A) Western blot analysis of STAT3 activation in U87 cells following 5 minutes of rhHGF stimulation (50 ng/ml) after 20-hour serum starvation. (B) The activity of STAT3 was determined in U87 Δ EGFR-expressing cells after rhHGF stimulation as described in A.