

Supplementary Material: Therapeutic Efficacy of Gc118, a Novel Anti-Egfr Antibody, against Glioblastoma with High Egfr Amplification in Patient-Derived Xenografts

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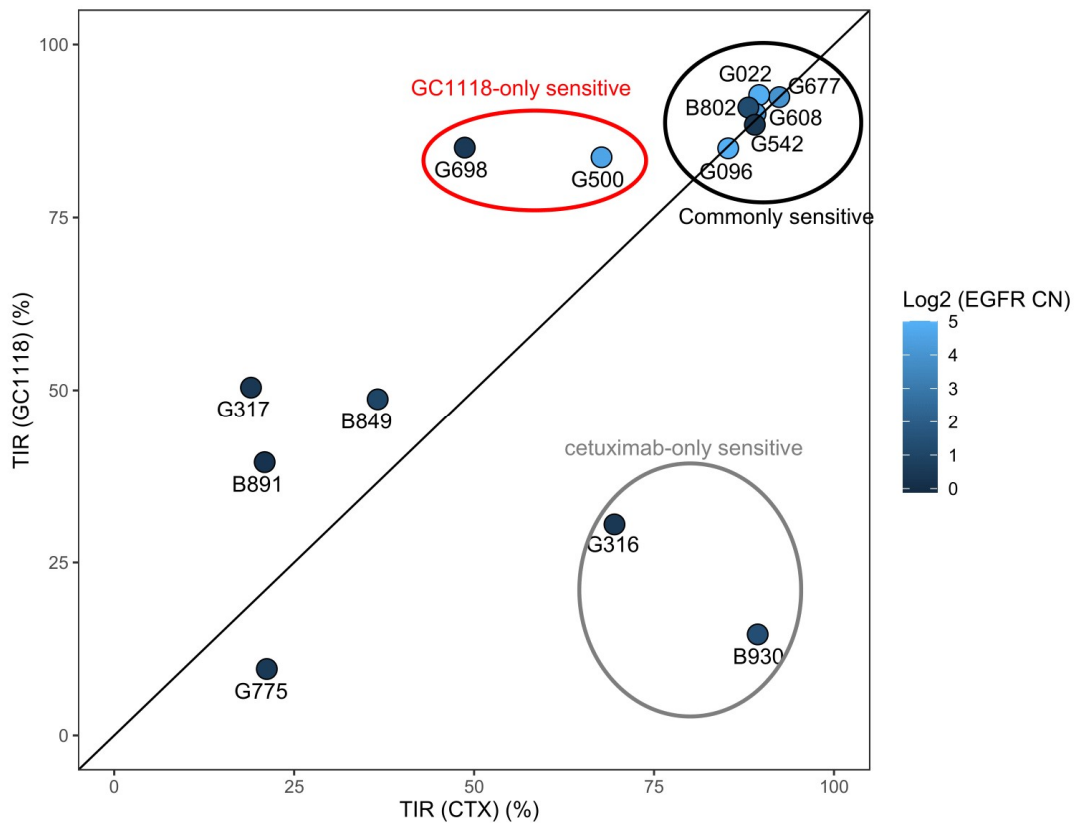


Figure S1. Comparison of anti-tumor effect of GC118 and cetuximab. GBM PDXs exhibited different therapeutic vulnerability against GC118 and cetuximab, despite their common mechanism of action ($\rho = 0.57$, p -value = 0.03, Pearson correlation test). G698 and G500 were more sensitive to GC118, while G316 and G930 exhibited specific sensitivity to cetuximab.

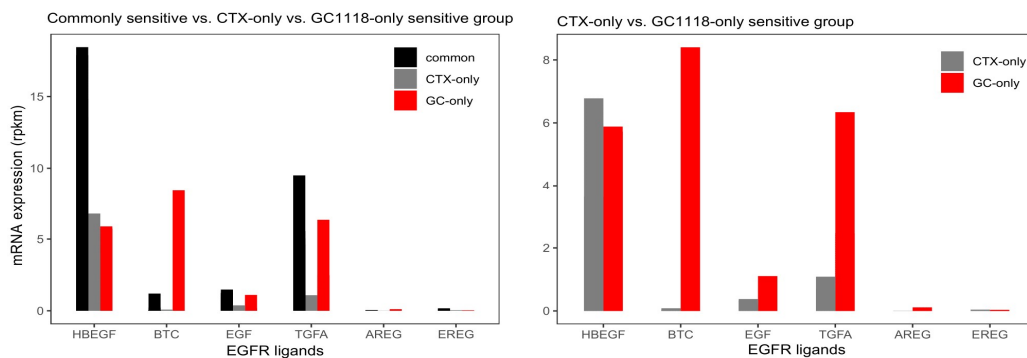


Figure S2. mRNA expression of EGFR ligands in GBM tumors. Using patient-derived transcriptomic data ($n = 8$), we compared the mRNA expression of EGFR ligands. EGFR ligands are classified into

two subsets, high-affinity (heparin-binding EGF-like growth factor (HB-EGF), β -cellulin (BTC), EGF, and TGF α) vs. low-affinity ligands (amphiregulin (AREG) and epiregulin (EREG)). GBM tumors exhibited predominant expression of high-affinity ligands. The expression of BTC and TGF α was upregulated in the GC1118-only sensitive group, although not statistically significant.

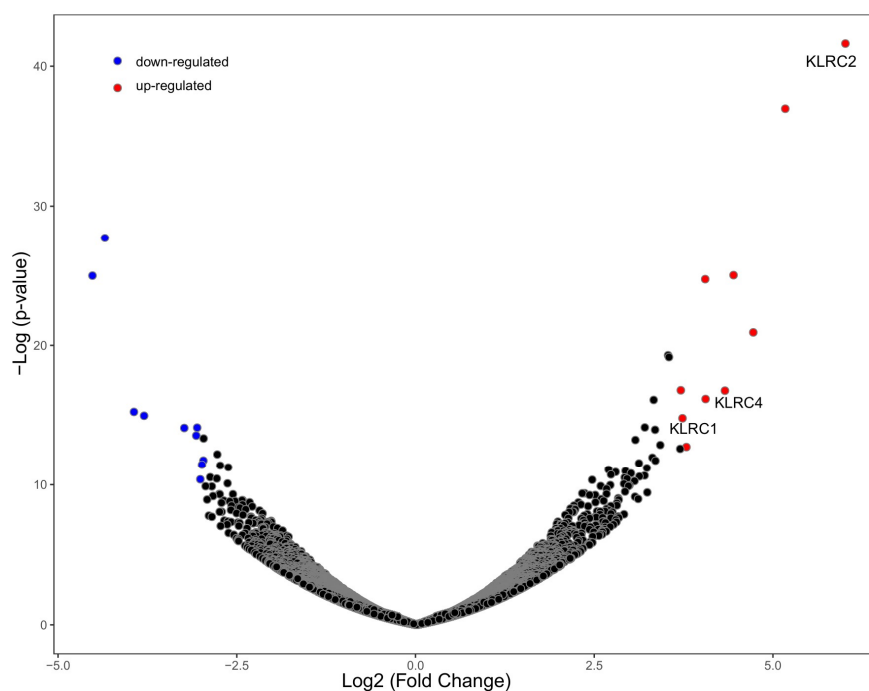


Figure S3. A volcano plot depicting differential gene expression analysis between the GC1118-only ($n = 2$) and cetuximab-only sensitive group ($n = 2$).



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