Supplemental Figure Legends

Supplemental Figure 1. *Attenuation of DGKα is toxic to glioblastoma cells* in vitro. Cell viability by alamarBlue assay was significantly reduced 72 hours after DGKα silencing in both U87 and U251 GBM cell lines. (*, P<0.05 and **, P<0.01 Student t test).

Supplemental Figure 2. *DGKa knockdown suppresses several oncogenic pathways*. Immunoblot analysis of U87 and U251 cell lysates exhibit a marked reduction in c-Myc and phos-AKT_{ser473} levels with DGKα knockdown.

Supplemental Figure 3. *DGKα modulates several oncogene-related pathways*. A, In both U87 and U251 cells, mRNA levels of FDPS1, FDPS2, HMGCR, and SCD were quantified by qRT-PCR in response to DGKα knockdown via siRNA and log-scale fold expression changes in comparison to control siRNA are shown. B, c-Myc was over-expressed through plasmid transfection after DGKα knockdown via siRNA and cell proliferation was assessed in U87 cells. (*, P<0.05 and **, P<0.01 Student t test).

Supplemental Figure 4. *Lentivirus infection with DGKα shRNA is efficient* in vitro. Lentiviral infection with DGKα shRNA was significantly cytotoxic to 0308 glioblastoma stem cells (GSCs), and immunoblot confirms the shRNA silencing of target. (*, P<0.05 and **, P<0.01 Student t test).

Supplemental Table 1. Data from The Cancer Genome Atlas (15) indicating amplification and mutation rates of DGKα in GBM and several other cancers.
**Supplemental Table 2.** A statistical analysis of 576 human GBM samples (15) was conducted to correlate DGKα and mTOR mRNA expression (plot shown in Figure 4).

**Supplemental Table 3.** Values of quantitative parameters supporting predicted BBB penetration by small molecule inhibitors R59022 and R59949 (47)

**Supplemental Table 4.** A statistical analysis was performed at each time point to assess the change in tumor volume of A, U87 and B, A-375 subcutaneous tumors treated with R59022 at 10 mg/kg compared to DMSO treatment.