**Supplementary Figures**

Figure S1. Doxycycline inhibits growth but not survival in dox-sensitive TSC cultures. (A) Growth curves of TSC-S2 and (B) TSC-S3 in the presence or absence of dox (50 pg/ml). (C) FACS analysis of PI stained TSC-S1 following treatment with dox (20, 50 and 100 pg/ml). Data are representative of three independent experiments, presented as mean +/- S.E.M. (*p<0.05, **p<0.01, ***p<0.005).

Figure S2. PDGFR activation alters expression of molecules that are key regulators of glycolysis. (A) Western blot analysis of Glut1 expression in lysates of untreated (-) and dox-treated (+) TSC cultures. (B) RT-PCR determination of steady state HK2 and (C) LDHA mRNA in untreated and dox-treated (20, 50 or 100 pg/ml) TSCs. Data are representative of three independent experiments in triplicate, presented as mean +/- S.E.M. (*p<0.05).

Figure S3. PDGF signaling regulates intracellular ATP levels. Intracellular ATP normalized to total cellular protein was examined in TSC-S1 treated (A) with dox (50 or 100pg/ml) for 24h or (B) with AG1295 (5 or 10 μM) for 24h. Data are representative of three independent experiments, presented as mean +/- S.E.M. (**p<0.01, ***p<0.005).

Figure S4. PDGF regulates glycolysis in cell-cycle arrested TSCs. (A) TSCs were treated with MMC (5 μg/ml) for 48h, washed, and incubated in normal growth media for 96h. Cells were subsequently fixed in ethanol and their cell cycle distribution evaluated by FACS analysis of PI staining. (B) Lactate production and (C) glucose consumption
was determined from conditioned media of MMC-treated TSCs that were additionally treated with dox (100pg/ml) for 96h. Data are representative of three independent experiments, presented as mean +/- S.E.M. (*p<0.05, ***p<0.005).

**Figure S5. Preparation of dox-resistant, TSC cultures from GFAP-tTA:TRE-hPDGFB mice.** (A, B) Schematics demonstrate methods to generate dox-resistant TSCs in the *de novo* tumor recurrence model and the xenograft tumor recurrence model, respectively. (C) Growth curves of TSCs derived from *in vivo* (left) and xenograft (right) recurrence model in the presence and absence of dox (1 μg/ml). (D) Growth Curves of dox-sensitive TSC-S3 (left panel) and dox-resistant TSC-R1 (right panel) in the presence (dotted line) and absence (solid line) of AG1295 (10 μM). Panel insets are western blots examining the activation of PDGFR following AG1295 treatment for 24h. Data in panel C and D is representative of three independent experiments, presented as mean +/- S.E.M. of triplicates.

**Figure S6. Inhibition of AKT activation decreases glycolysis in glioma-derived TSCs.** (A) Western blot analysis of HA-tag expression and p70-S6K phosphorylation in TSC-S1 clones overexpressing pCMV or pCMV-HA-AKT-DN. (B) Lactate production and (C) glucose consumption was determined from conditioned media of TSC-S1 clones overexpressing pCMV and pCMV-HA-AKT-DN at 96h after transfection. Data in panel B and C are representative of three or more independent experiments, as mean +/- S.E.M. of triplicates. (*p<0.05).