Supplementary Figure Legends

Supplementary Figure 1. Chemical structures of (A) rapamycin and (B) NVP-BEZ235 (adopted from the LC Laboratories website; www.lclabs.com).

Supplementary Figure 2. Neurofibromin regulates Jagged-1 expression *in vitro* and *in vivo*. A, *Nf1*-deficient astrocytes exhibit increased Jagged-1 expression, which is inhibited by rapamycin (10nM) treatment. Equal protein loading was confirmed by total S6. B, Jagged-1 expression was reduced following rapamycin treatment of *Nf1*^{GFAP}CKO mice (2, 5 and 20mg/kg/day). α-tubulin served as an internal loading control. Asterisks (*) denote statistically significant differences ($P<0.0001$) between control and rapamycin-treated mice.

Supplementary Figure 3. Rapamycin treatment reduces proliferation in *Nf1*^{GFAP}CKO mice *in vivo*. PCNA immunolabeling demonstrates decreased cell proliferation in the dentate gyrus of rapamycin-treated mice only at 20mg/kg/day compared to vehicle-treated controls ($P=0.352$). Asterisks (*) denote a statistically significant difference.

Supplementary Figure 4. Combined rapamycin and LY294002 treatment does not induce apoptosis in *Nf1*-deficient glioma cells. Treatment with rapamycin and LY294002 either alone or together does not induce apoptosis as determined by cleaved PARP immunoblotting. Staurosporine (1μM) serves as positive control for PARP cleavage. No change in pAKT^{Thr308} phosphorylation is observed following LY294002 or rapamycin treatment. Total AKT serves as an internal protein loading control. Relative density (R.D.) values for AKT phosphorylation (pAKT/total AKT) are shown.

Supplementary Figure 5. Rapamycin inhibits Nf1-deficient glioma proliferation. 100nM and 500nM rapamycin treatment reduced K4622 glioma cell growth by ~55% ($P<0.0017$) and ~60% ($P<0.0007$), respectively, as measured by [^{3}H]-thymidine incorporation, similar to 10 nM rapamycin. Asterisks (*) denote a statistically significant difference.

Supplementary Figure 6. *Nf1-/-* astrocytes exhibit mTOR-dependent increases in cell proliferation. A, 10nM rapamycin inhibits S6 activation (P-S6). Total S6 serves as an internal protein loading control. B, *Nf1-/-* astrocytes exhibit a 2-fold increase in cell proliferation as determined by [^{3}H]-thymidine incorporation, which is reduced to wild-type levels following treatment with 100 nM, but not 10nM, rapamycin.