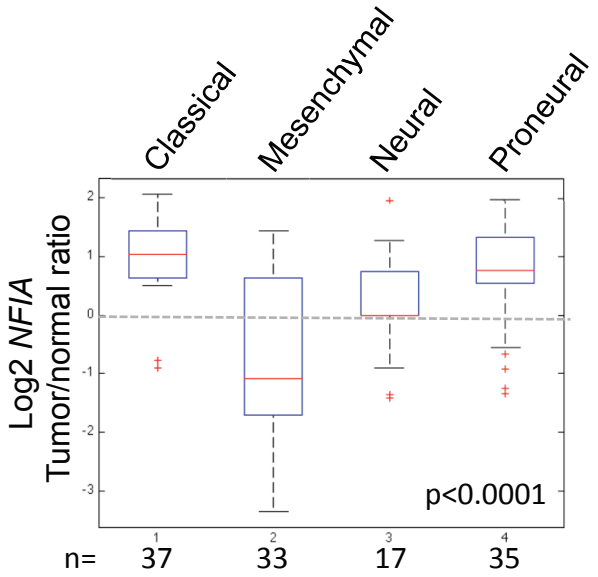

Fig S1. NFIA mRNA is higher than non-tumor brains in proneural, neural, and classic GBM and is lower in mesenchymal GBM. Data for 202 GBM patients were extracted from a published study. Gene expression values were extracted from The Cancer Genome Atlas (TCGA) data portal (Verhaak *et al*, Cancer Cell, 2010) for 54 classical, 58 mesenchymal, 33 neural and 57 proneural GBMs. Log2 transformed tumor/normal ratio on AgilentG4502A_07 platform was used to measure the expression level of NFIA in the patient samples. Low gene expression ratio change between -0.5 and 0.5 was filtered out. This resulted in 37 out of 54 classical, 33 out of 58 mesenchymal, 17 out of 33 neural, and 35 out of 57 proneural samples with expression change above the threshold. NFIA is upregulated in 95% of the classical, 82% of the proneural, and 65% of the neural samples, and downregulated in 67% of mesenchymal samples; p-value <0.0001 by one-way ANOVA.

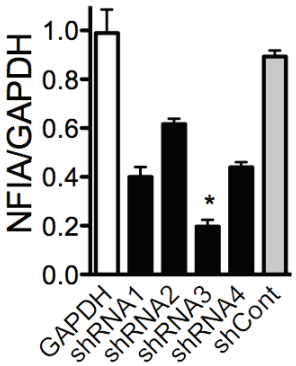

Fig S2. Knockdown of endogenous NFIA by shRNA in U87 glioma cells. qPCR analysis of NFIA relative to GAPDH in glioma cells expressing shRNA targeting human NFIA. shRNA3 showed greater than 80% knockdown and was used in all subsequent experiments (shNFIA); an shRNA containing six nucleotide mismatch had minimal effect on NFIA expression and served as control (shCont).