

Fig S1. Quantification of number of GSC expressing NFIA or vector control (colonies >0.1 mm), p<0.0001 by 2-way Anova. Immunoblotting of GBM cells expressing NFIA or vector at Day 7 (Right).



Fig S2. U87 cells stably expressing NFIA or vector control (Control) were treated with etoposide (ETP 1 μ g/ml) or vehicle (DMSO) for 24 hr. Apoptosis (%) was assessed using the Apo-Direct kit; *p<0.005.



Fig S3. Immunoblotting of Caspase-8 cleavage measured in GBM1 cells expressing NFIA or vector following treatment with the increasing amount of etoposide.



Fig S4. Immunoblotting of NFIA-deleted GBM cells (transduced with shNFIA or shCont) treated with GFP-NF κ B p65 or GFP vector control; errows indicate exogenous (exo) or endogenous (endo) NF κ B p65.



Fig S5. Caspase-3 activity of NF κ B p65-depleted GBM cells (siNF κ B or siCont, or NF κ B inhibitor or DMSO) transduced with NFIA or vector control, *p<0.05.



Fig S6. Caspase-3 activity in GBM1 cells expressing NFIA or vector control, treated with the increasing amount of NF κ B inhibitor (iNF κ B), *p<0.05.



NFIA, Putative copy-number alterations from GISTIC

Fig S7. cBioPortal (cbioportal.org) was queried to explore NFIA expression profiles, copy number status in the glioblastoma dataset (TCGA, Cell 2013) which includes 580 samples in total and 291 samples with completed information. NFIA has copy number variation in 23 (out of 291, about 8%) samples, including 1) homozygous deletion in one patient, which also shows low expression level measured by RNAseq profiling; 2) heterozygous deletion in 6 patients; and 3) copy number gain in 15 patients. Compare to normal copy number status, NFIA copy loss or gain shows corresponding down and up regulation of gene expression levels. Minor dosage effect on transcription was observed in this data set and an analysis on a data set with larger cohort may strengthen the conclusion.



Fig S8. Immunoblot of immortalized human astrocytes (IHA) and GBM1 cells probed with an NFIA antibody.



Fig S9. Immunoblot of patient-derived GBM cells (GBM1-proneural; GBM2-mesenchymal; GBM3-proneural; Left) or GBM1 cells expressing vector or NFIA (Right) probed with antibodies indicated.



Fig S10. Correlation between NFIA and STAT3, NFIA and TAZ, NF κ B (ReIA) and STAT3, and NF κ B (ReIA) and TAZ mRNA expression in GBM (580 GBM samples in total; TCGA, Cell 2013; cBioPortal.org).