Supplementary Figure Legends

Figure S1. Epigenetic repression of TSGs in lung cancer cells by oncogenic EGFR (Related to Figures 1 and 2)

(A) HCC827/Del and HCC827/Del-TM cells treated with decitabine (2.5 μM) and vorinostat (1 μM) for 48 h. RT-qPCR analysis was performed to analyze the expression of the indicated TSGs. The expression levels of the TSGs in treated cells are plotted relative to DMSO-treated cells. (B) HCC827/Del and HCC-827/Del-TM cells were treated with gefitinib (0.1 μM), afatinib (0.1 μM), or DMSO for 48 h. The indicated proteins were analyzed via immunoblot analyses. (C-E) HCC827/Del-TM cells were treated afatinib (0.1 μM) or DMSO for 48 h. (C) DNA methylation of the CpG islands in the promoters of the indicated genes was measured via Me-DIP. The percent of CpG island DNA methylation for the promoters of the indicated genes in drug-treated cells compared to that of DMSO-treated cells is shown. (D) RNA polymerase II enrichment on the TSG promoters was measured via ChIP. The relative enrichment observed in drug-treated cells compared to that of DMSO-treated cells is shown. (E) The amount of histone H3 lysine 9 acetylation (H3K9-Ac) marks on the indicated gene promoters was measured. The levels of H3K9-Ac marks observed in drug-treated cells are shown relative to those in DMSO-treated cells. (F) HCC827/Del cells were treated with gefitinib (0.01 μM). The indicated proteins were analyzed via immunoblot analyses. (G) An *NDRG4* promoter-luciferase reporter was stably transfected into HCC827/Del or HeLa cells, and luciferase activity was measured after 48 h of treatment with decitabine (2.5 μM), and vorinostat (1 μM), or DMSO (-). (H) HCC827/Del-TM cells were treated with afatinib (0.01 μM). The indicated proteins were analyzed via immunoblot analyses. (I). An *NDRG4* promoter-luciferase reporter was stably transfected into HCC827/Del-

TM or HeLa cells, and luciferase activity was measured with afatinib (0.01 μM) or DMSO (-) treatment for 48 h. * represents $p<0.05$.

Figure S2. MAP kinase pathway is necessary for TSG silencing in lung cancer cells and TET2, TET3, and DNMTs are not regulated by oncogenic EGFR (Related to Figure 3)

(A-C) HCC827/Del cells were treated with DMSO or U0126 (10 μM) for 24 or 48 hrs. A. Immunoblot analysis for p-ERK1/2 and total-ERK1/2 was performed using samples as indicated. (B) Relative mRNA expression of indicated genes were analyzed using RT-qPCR and plotted as indicated. (C) Relative DNA methylation for indicated gene promoters was analyzed by MeDIP in HCC827/Del cells and plotted as indicated. (D-F) HCC827/Del cells were treated with DMSO or U0126 (5 μM or 10 μM) for 48 hrs. (D) Immunoblot analysis for p-ERK1/2 and total-ERK1/2 was performed as indicated. (E) Relative mRNA expression of indicated genes were analyzed using RT-qPCR and plotted as indicated. (F) Relative DNA methylation for indicated gene promoters was analyzed by MeDIP and plotted as indicated. (G-H) HCC827/Del cells were treated with gefitinib (0.1 μM) for 48 h. (G) Expression levels of the *TET2* and *TET3* transcripts were analyzed via RT-qPCR. *TET2* and *TET3* expression levels in drug-treated cells relative to those in HCC827/Del cells treated with DMSO are shown. (H) The expression levels of the *DNMT1*, *DNMT3A*, and *DNMT3B* transcripts were analyzed via RT-qPCR. *DNMT1, DNMT3A*, and *DNMT3B* expression levels in drug-treated cells relative to those in HCC827/Del cells treated with DMSO are shown. * represents $p<0.05$.

Figure S3. Oncogenic EGFR induces epigenetic silencing of tumor suppressor genes (Related to Figure 4)

(A-C) Immortalized human airway epithelial cells (HAE/SV40+hTERT) expressing indicated EGFR mutant was treated with DMSO or Gefitinib (0.1 μM) or Afatinib (0.1 μM) for 48 h (A) mRNA expression for the indicated TSGs were analyzed using RT-qPCR. Relative expression for indicates TSGs under indicated drug treatment condition is plotted. (B-C) Immortalized human airway epithelial cells (HAE/SV40+hTERT) expressing indicated EGFR mutant was treated with DMSO or gefitinib (0.1 μM) for 48 h. (B) DNA methylation for indicated TSG promoters were analyzed by MeDIP analysis. Relative DNA methylation for the indicated TSG promoters is shown under indicated conditions. (C) H3K9Ac level was analyzed by ChIP analysis for indicated TSG promoters. Relative H3K9Ac levels are shown for indicated TSG promoters is shown under indicated conditions. (D-E) Immortalized human airway epithelial cells (HAE/SV40+hTERT) expressing indicated EGFR mutant was treated with DMSO or afatinib (0.1 μM) for 48 h. (D) DNA methylation for indicated TSG promoters were analyzed by MeDIP analysis. Relative DNA methylation for the indicated TSG promoters is shown under indicated conditions. (E) H3K9Ac level was analyzed by ChIP analysis for indicated TSG promoters. Relative H3K9Ac levels are shown for indicated TSG promoters is shown under indicated conditions. (F) Immortalized human airway epithelial cells (HAE/SV40+hTERT) expressing indicated EGFR mutant was treated with DMSO or Gefitinib (0.1 μM) or Afatinib (0.1 μM) for 48 h. The expression of *C/EBPα* mRNA was analyzed using RT-qPCR. Relative *C/EBPα* mRNA expression under indicated condition is shown. (G) Immortalized human airway epithelial cells (HAE/SV40+hTERT) expressing indicated EGFR mutant was treated with DMSO or Gefitinib (0.1 μM) or Afatinib (0.1 μM) for 48 h. The expression of *TET1* mRNA was analyzed using RT-qPCR. Relative *TET1* mRNA expression under indicated condition is shown. * represents $p<0.05$.

Figure S4. Regulation of *TET1* and *C/EBP*α transcription in lung cancer cells (Related to Figure 4)

(A) The indicated lung cancer cell lines were treated with afatinib (0.1 μM) for 48 h, and the expression of C/EBPβ (Left) and GATA2 mRNA (Right) were measured via qRT-PCR. (B-C) HCC827/Del cells expressing non-specific (NS) shRNAs or shRNAs targeting the indicated transcription factors were analyzed for the expression of the indicated genes via RT-qPCR (B) or immunoblot analyses (C). (D) HCC827/Del cells expressing NS shRNAs or shRNAs targeting *C/EBPβ* or *GATA2* were analyzed for the expression of TET1 via RT-qPCR. Relative *TET1* expression in cells expressing the indicated shRNAs compared to cells expressing NS shRNAs is shown. (E) The indicated lung cancer cells expressing NS or *C/EBPα* shRNA were treated with afatinib (0.1 μM) and analyzed for the expression of *TET1* and TET1 via RT-qPCR (Left) and immunoblot analyses (Right), respectively. *TET1* and TET1 expression levels in *C/EBPα* shRNA-expressing cells compared to those observed in cells expressing NS shRNAs are shown. (F) HCC827/DEL cells were treated with DMSO (-) or afatinib (0.1 μM) or U0126 (10 μM) for 48 hrs and (Left) analyzed for YY1 mRNA expression by RT-qPCR or (Right) immunoblot analysis for YY1. p-ERK1/2 and total-ERK1/2 were also measured by immunoblot analysis. (G) HCC827/DEL cells expressing NS or YY1 shRNA were analyzed for the expression of *YY1* and *C/EBPα* mRNA (Left) by RT-qPCR or protein (Right) by immunoblot analysis. Actin was used as a loading control for immunblotting. (H) YY1 enrichment on ACTIN, GAPDH or C/EBPα promoters were analyzed by ChIP assay in HCC827/Del cells treated with DMSO, afatinib or U0126. The relative enrichment of YY1 in relation to IgG on Actin, GAPDH or C/EBPα promoters is shown under indicated conditions. * and ** represents $p<0.05$ and $p<0.005$ respectively.

Figure S5. *C/EBPα* and *TET1* expression is regulated by MAP kinase pathway in GBM cells and TET1 loss confers resistance to gefitinib (Related to Figures 5 and 6)

(A-B) Indicated EGFR mutant lung cancer cell lines were treated with DMSO (-) or U0126 (10 μM) for 48 hrs (A) C/EBPα mRNA expression was analyzed using RT-qPCR. Relative mRNA expression is shown in indicated cell lines with or without U0126 (10 μM). (B) C/EBPα protein expression was analyzed using immunoblot analysis. Actin was used as a loading control. (C) HCC827/DEL cells expressing either an empty vector or KRASG12D were treated either with DMSO or afatinib and analyzed for the expression of C/EBPα mRNA by RT-qPCR analysis. Relative mRNA expression for C/EBPα under indicated conditions is shown. (D) HCC827/DEL cells expressing either an empty vector or oncogenic KRAS, KRASG12D were treated with DMSO or U0126 (10 μM) for 48 hrs and analyzed for the expression of indicated TSGs. Relative mRNA expression for indicated TSGs is shown. (E) Indicated GBM cell lines were treated with U0126 at indicated concentrations for 48 hrs. TET1 expression was analyzed by RT-qPCR analysis. Relative TET1 expression in treated cells in comparison to untreated cells is plotted. (F) HCC827/Del or H3255 cells expressing non-specific (NS) or *TET1* shRNAs were treated with gefitinib as indicated and the expression of TET1 was analyzed by immunoblot analysis. Actin was used as a loading control. (G) HCC827/Del or H3255 cells expressing indicated shRNAs were treated with gefitinib as indicated for 48 hrs and analyzed for survival. % survival at indicated concentrations is shown. * represents $p<0.05$..

Figure S6. EGFR TKI resistant oncogenic EGFR mutant tumors show higher MAP kinase target gene expression (Related to Figure 7)

(A) Indicated Erlotinib sensitive and resistant tumors were analyzed for the indicated MAP kinase gene targets by RT-qPCR. Relative mRNA expression in relation to the first sensitive tumor (ST1) is shown. (B) Indicated EGFR TKI sensitive and resistant tumors were analyzed for the indicated MAP kinase gene targets by RT-qPCR. Relative mRNA expression in relation to the first sensitive human tumor sample (SHT1) is shown. ** represents $p<0.005$.

Figure S7. Km plot dataset analysis reveals association of lower expression of indicated TSGs with poor overall survival in lung cancer patients (Related to Figure 7)

Indicated TSGs were analyzed for their association with overall survival in lung cancer dataset using Km plot database for lung cancer.

Supplementary Experimental Methodology

Cell Culture, Plasmids, and Cloning

The HCC827/Del and HCC827/Del-T790M cell lines were characterized and grown as described previously (Kobayashi et al., 2006). HeLa and PC9 cells were grown in DMEM medium supplemented with 10% fetal bovine serum (FBS) and incubated under 5% CO_2. The H3255 and H1975 cell lines were grown in RPMI medium supplemented with 10% FBS and incubated under 5% CO_2. H1650, MRC5, HBEC, H1437, A549, and H1299 cells were obtained from American Type Culture Collection (ATCC) and were grown as recommended. Immortalized human airway epithelial cells (HAE/SV40ER+hTERT) were obtained from Dr. William G. Hahn (Dana Farber Cancer Institute) and grown as described previously (Lundberg et al., 2002). U87/EGFP and U87/EGFRvIII cells were obtained from Dr. Zhimin Lu at the MD Anderson Cancer Center. The GBM6 and GBM313 cell lines were obtained from Dr. Joseph Loftus at the Mayo Clinic and maintained as described previously (Giannini et al., 2005). The pcDNA3.1 hygro-TET1 plasmid was generated by sub-cloning TET1 from the pAAV-EF1a-HA-hTet1CD-WPRE-PolyA plasmid (Addgene plasmid #39454) (Guo et al., 2011) into the *Kpn*I and *Eco*RV restriction enzyme sites of the pcDNA3.1 hygro plasmid. A CpG island was identified ~600 bp upstream of the translational start site for the *NDRG4* gene using Emboss Cpgplot island prediction software (http://www.ebi.ac.uk/Tools/seqstats/emboss_cpgplot/). The BAC clone RP11-481J2 (BACPAC Resource Center) was used to amplify 1.2 kb of the *NDRG4* promoter sequence upstream of the translational start site, which was cloned between the *Xho*I and *Bgl*II restriction enzyme sites of the pGL4.14 Luc2/Hygro vector (Promega) and into a vector

expressing a blasticidin-resistance gene that was described previously (Palakurthy et al., 2009). All cloning primers are listed in **Table S3**.

shRNAs, Transfection, Lentivirus Preparation, and Immunoblot Analysis

All shRNAs and non-specific control shRNAs were obtained from Open Biosystems (**Table S3**). Lentivirus particles were prepared by co-transfecting a shRNA plasmid with the lentiviral packaging plasmids pSPAX2 and pMD2.G into 293T cells using Effectene (Qiagen). For stable shRNA knockdowns, cells were seeded in a 12-well plate, permitted to reach 50% confluency, and subsequently transduced with 100 μl of lentiviral particles expressing shRNAs in a total volume of 1 ml of appropriate media. Media were replaced after 24 h of incubation with the viral particles, and the cells were subjected to selection using 0.25–1.0 μg/ml puromycin depending on the cell line. Immunoblot analysis was performed as described previously (Santra et al., 2009) using antibodies listed in **Table S3**.

RT-qPCR, ChIP Assays, and MeDIP Assay

Total RNA was extracted using TRIzol (Life Technologies), and purified using the RNeasy Mini Kit (Qiagen). The ProtoScript first-strand cDNA synthesis kit (New England BioLabs) was used to generate cDNA. Quantitative PCR (qPCR) was performed using the Power SYBR Green 2× Master Mix (Life Technologies). All RT-qPCR reactions were performed in triplicate using the primers listed in **Table S3**. The relative fold-changes in mRNA expression were calculated using the comparative C_T method (Schmittgen and Livak, 2008). ChIP experiments were performed as described previously (Raha et al., 2005) by incubating the lysates with specific antibodies as

required (**Table S3**). IgG antibody was used as a control. Fold-enrichment was calculated as a ratio of immunoprecipitated DNA to input DNA.

Me-DIP assays were performed as described (Gazin et al., 2007). Relative quantification of DNA fragments for each region was determined by plotting Ct values on the standard curve. The fold-difference of immunoprecipitated DNA compared with input DNA was calculated to determine enrichment levels of the target region.

Supplemental References

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Table S2: Transcription factor site prediction for TET1 promoter

Transcription factors
C/EBPalpha
C/EBPbeta
GATA1
GATA2
GATA3
HNF-3B
NKX2-1

Table S3. Primer sequences, clone IDs, catalog numbers, antibodies, and chemical inhibitors used in this study.

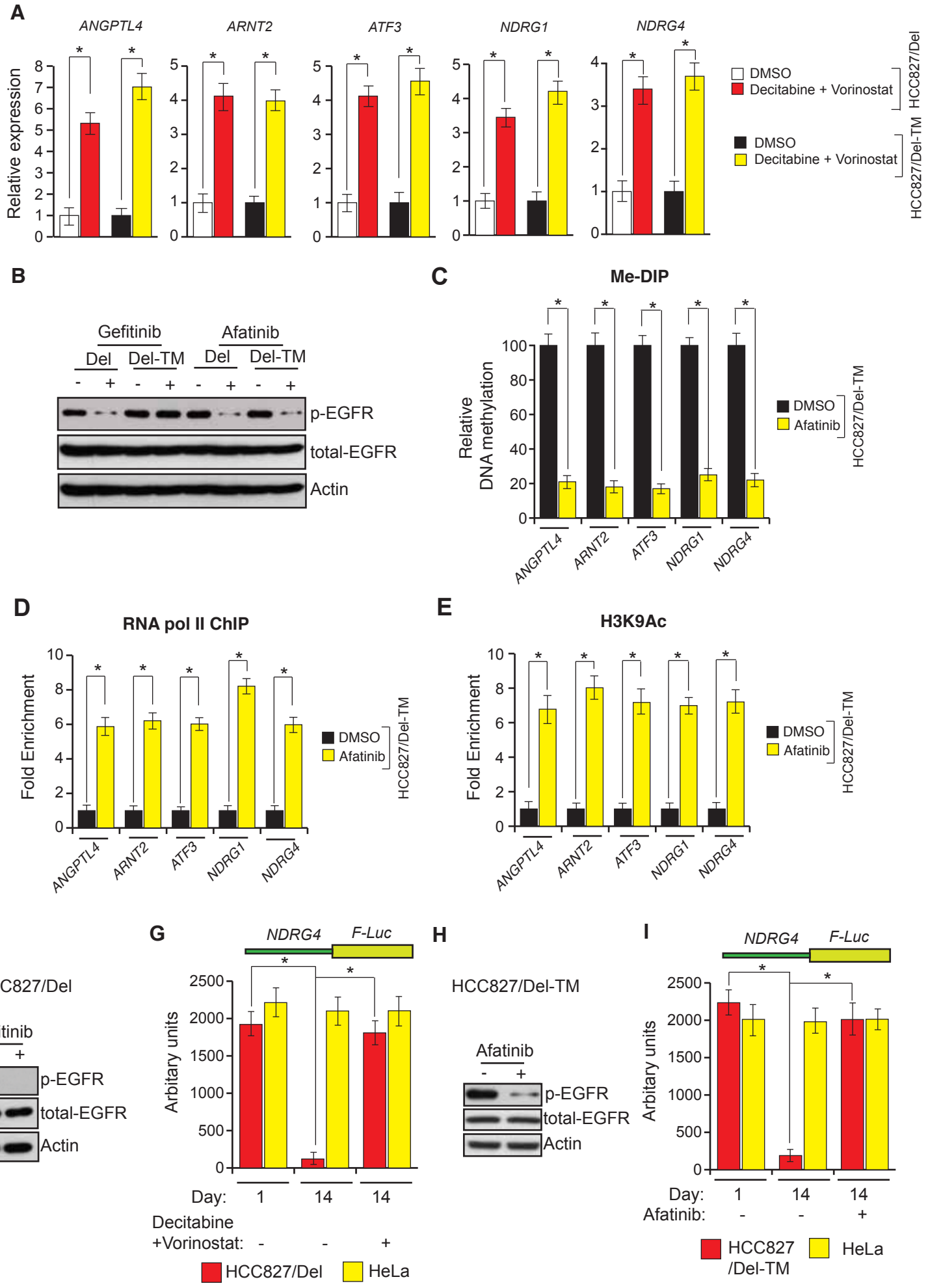
Primers were used for qRT-PCR analysis, ChIP experiments, and cloning. The shRNAs used herein were obtained from Open Biosystems; clone IDs and catalog numbers are listed. The antibodies were used for immunoblot analyses. The source and concentrations of chemical inhibitors used for drug treatment experiments are summarized.

Application	Gene symbol	Forward primer	Reverse primer
qPCR	*DNMT1*	5'-CAGCAACGGGCAGATGTTTC -3'	5'-CGGAGGGGGCTTTGTAGATG -3'
	DNMT3A	5'-CTACGCACCACCTCCACCAG -3'	5'-CAATGTTCCGGCACTTCTGC -3'
	DNMT3B	5'-AAGCCCAGCTGTCCGAACTC -3'	5'-GGACTCGTCCACATGGTTGC -3'
	TET1	5'-GCGACCCTTGGTGCTAAACC -3'	5'-CAGGGCCTCACCATGAACTG -3'
	TET2	5'- ACAGAAGCAGCCACCACAGC-3'	5'-TCATAGGGCTGGTGCTTCCA -3'
	TET3	5'- CCATTCAGGACCCCGAGAAC-3'	5'-CCACTGAGGGTGGGTGTGAG -3'
	ATF3	5'- CTCGGGGTGTCCATCACAAA-3'	5'- TGCAGGCACTCCGTCTTCTC-3'
	C/EBPα	5'- AGGCCAAGAAGTCGGTGGAC-3'	5'- CAGCACCTTCTGCTGCGTCT-3'
	C/EBPβ	5'- GGACAAGCACAGCGACGAGT -3'	5'- CTTGTGCTGCGTCTCCAGGT-3'
	GATA2	5'- CTCGGAACCGGAAGATGTCC-3'	5'- ATGTGTCCGGAGTGGCTGAA-3'
	GAPDH	5'-AAGGTCGGAGTCAACGGATTT-3'	5'-ACCAGAGTTAAAAGCAGCCCTG-3'
	ANGPTL4	5'-GCAACCAAGCGGGTCTTACC-3'	5'-GGAGCACCGCTCATCCTCTT-3'
	ARNT2	5'-TCTGCCTCGGAAGTGAGTGC-3'	5'-TTCCTTGACAAAGGGCGTCA-3'
	ATF3	5'-TCTGCCTCGGAAGTGAGTGC-3'	5'-TTCCTTGACAAAGGGCGTCA-3'
	NDRG1	5'-CCTGCTCTGTTGGTGGTTGG-3'	5'-CACAGTCCGCCATCTTGAGG-3'
	NDRG4	5'- CCCGACCACTACGACCTTCC -3'	5'- ATGCTGGCTGAGGGCATGTA -3'
	GAPDH	5'-AAGGTCGGAGTCAACGGATTT-3'	5'-ACCAGAGTTAAAAGCAGCCCTG-3'
qPCR/ChIP For RNA pol II, H3K9 Ac and TET1 ChIPs and CEBP/alpha ChIP			
	ANGPTL4	5'-ATCCCCCAGCCCGCAGACT-3'	5'-CTCAGCTCGGCTTCTCCCC-3'
	ARNT2	5'-AGTCGCCGGCGCCCAACTT-3'	5'-CTCGCCGTCCCCGCCTTTCT-3'
	ATF3	5'-TCACTCAGCTTTCTCCCGTGA-3'	5'-ACTGAAGCTAACCCCACCTCC-3'
	NDRG1	5'-CTACAGGAAGTGAAGGAGTC	5'-CGTGCCCGCGTGGGTTTGG-3'
	NDRG4	5'-GCGGTCCCCGCTCGCCCT-3'	5'-CCCGGAGGAGGCGTTTGTGT-3'
	GAPDH	5'-TGCAGGACATCGTGACCTTC-3'	5'-TGGTTCCTTCCCAGCCCCCA-3'
	ACTIN	5'-ATGCGGGGCCGAACCGGG-3'	5'-CCGCTCACTCACCGGCTTC-3'
	C/EBPalpha ChIP on TET1 promoter site 1	5'-TAGGTAGAATGTTTCTAACAAG-3'	5'-TGACCTCAGGTGATCTGCCT-3'
	C/EBPalpha ChIP on TET1 promoter site 1	5'-GAATGGGGGTAGAATGGTGT-3'	5'-CAGAATTAAGACTGACTAGG-3'
	YY1 ChIP on C/EBPα promoter	5'-TTTGTTCCTAGGTAGGAAAA-3'	5'-AGCTTGACGCTTTTGGACGG-3'
MeDIP			
	ANGPTL4	5'-CATCTCGGCGCACTGCAACC-3'	5'-CCTCTAGTGTGAAATTGCAAG-3'
	ARNT2	5'-AGCACCTGCGCGAGCCCG-3'	5'-CGCCGTCCCCGCCTTTCT-3'
	ATF3	5'-CCTCCCAAAGTGCTGGGATT-3'	5'-GGTGAAACCCAGTCTCTACT-3'
	NDRG1	5'-TCAGCGAGAGCTTTGGCGA-3'	5'-GTGTCTCCGTATGGGCGG-3'
	NDRG4	5'-ACTACGGCAGCGCCGTCC-3'	5'-TTCTGCGCGGCTGGGGTG-3'

Application	Gene symbol	Clone ID	Catalog number
shRNAs	*TET1*	TRCN0000075023	AAJ55-E-8
		V3LHS_644423	172_0930-F-12
	GATA2	TRCN0000019264	AAD-33-E-3
		TRCN0000019267	AAD33-E-6
	C/EBPβ	TRCN000007443	AAC18-D-8

		V3LHS_48322	172_1327-D-5
	C/EBPα	TRCN000007304	AAC16-G-4
		TRCN000007305	AAC16-G-5
	YY1	TRCN0000019895	AAD40-E-4
		TRCN0000019898	AAD40-E-7
Application	**Protein symbol**	**Antibody source**	**Catalog number**
Immunoblot/ ChIP			
	pERK1/2	Cell Signaling Technology	#4370S
	EGFR	Cell Signaling Technology	#2232
	pEGFR	Cell Signaling Technology	#3777
	pAKT	Cell Signaling Technology	#9271
	pSTAT1	Cell Signaling Technology	#8062
	ß-actin	Sigma-Aldrich	A2066
	ANGPTL4	EMD Millipore	AB10605
	ARNT2	Abcam	ab129690
	ATF3	Santa Cruz Biotechnology	sc-188
	NDRG1	Santa Cruz Biotechnology	sc-398823
	NDRG4	Santa Cruz Biotechnology	sc-393342
	TET1	Millipore	09-872
	C/EBPα	Santa Cruz Biotechnology	sc-365318
	C/EBPβ	Santa Cruz Biotechnology	sc-398753
	GATA2	Santa Cruz Bioetchnology	sc-267
	RNA pol II	BAbCO	8WG16
	H3K9Ac	EMD Millipore	07-352
	Me-Cytosine	Active Motiff	39649
	YY1	Cell Signaling	#2185
Application	**Inhibitor**	**Concentration**	**Source**
Chemical inhibition	Erlotinib	0.5μM	LC Laboratories
	Gefitinib	0.05 - 5 μM	LC Laboratories
	Afatinib	0.05 - 5 μM	LC Laboratories
	Aphidicolin	5 μM	Sigma-Aldrich
	Decitabine	2.5 μM	
	Vorinostat	1 μM	LC Laboratories
	U0126	10 μM	Cell Signaling Technology
	Wortmannin	10 μM	Cell Signaling Technology
	Ruxolitinib	1 μM	SelleckChem

Figure S1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B
Gefitinib
Afatinib
Del
Del-TM
Del
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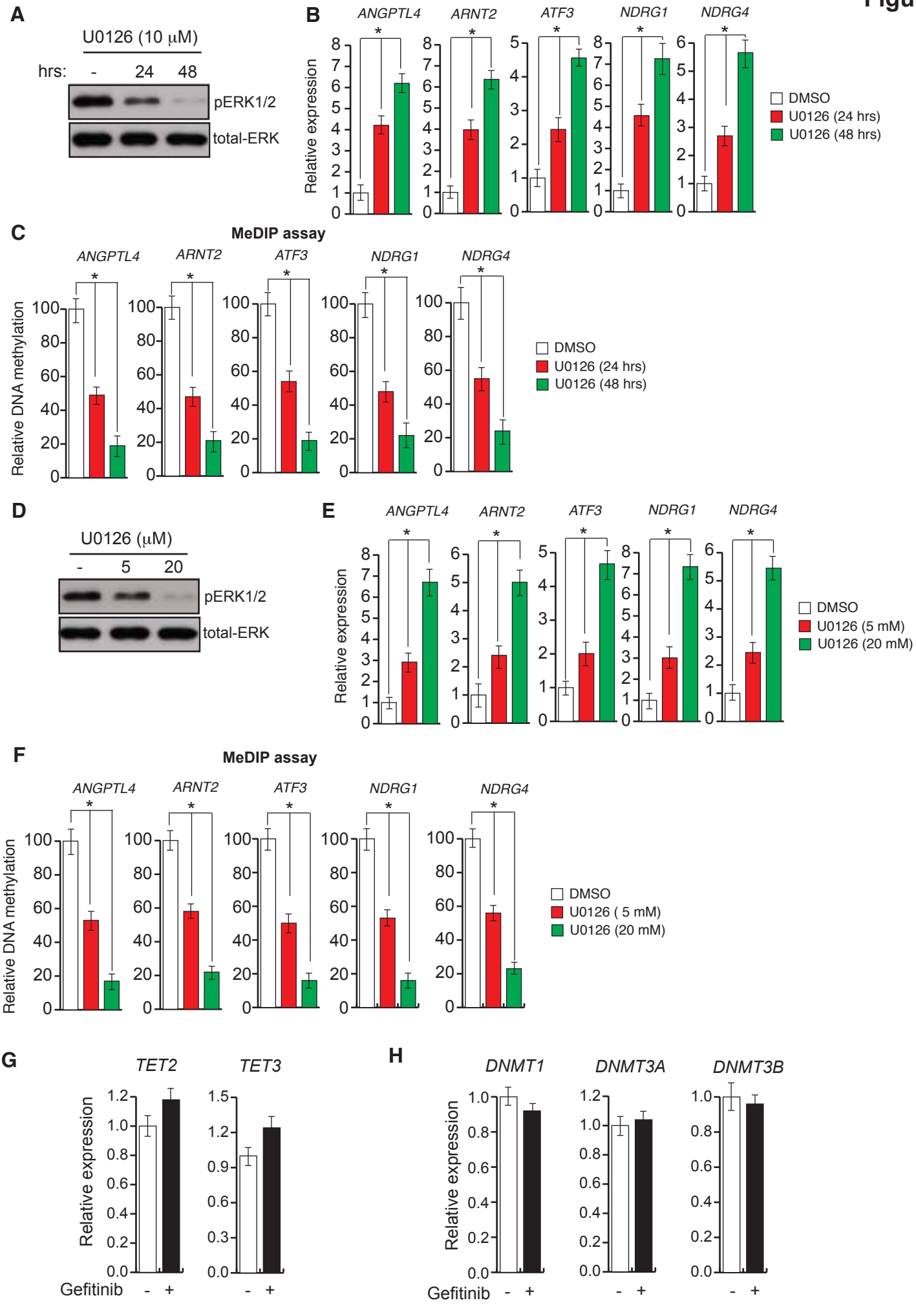
Figure S2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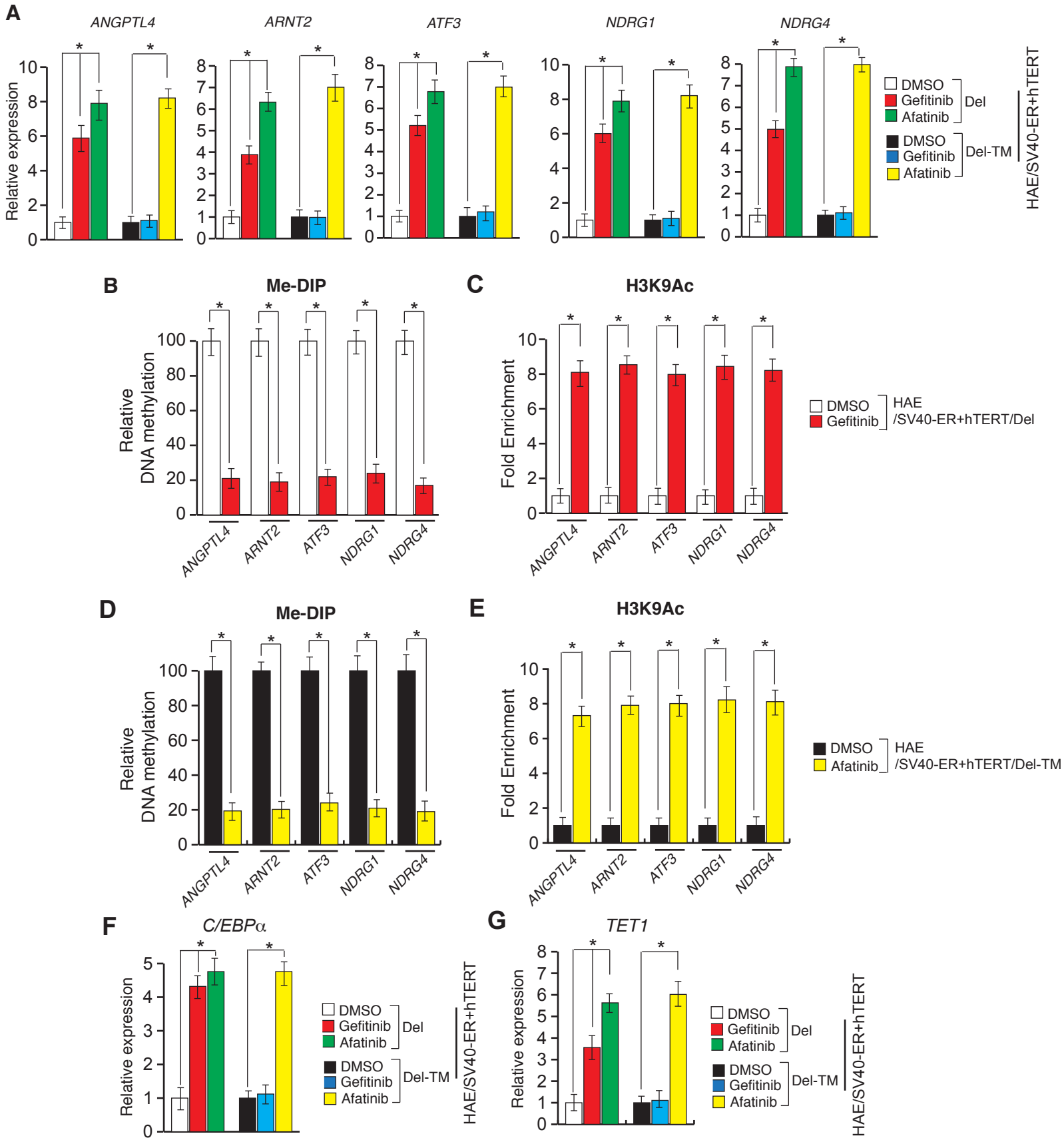
Relative expression
Gefitinib - + - +

Figure S3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B
Me-DIP
Relative DNA methylation
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ARNT2
ATF3
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NDRG4
C
H3K9Ac
Fold Enrichment
DMSO
Gefitinib
HAE /SV40-ER+hTERT/Del
D
Me-DIP
Relative DNA methylation
E
H3K9Ac
Fold Enrichment
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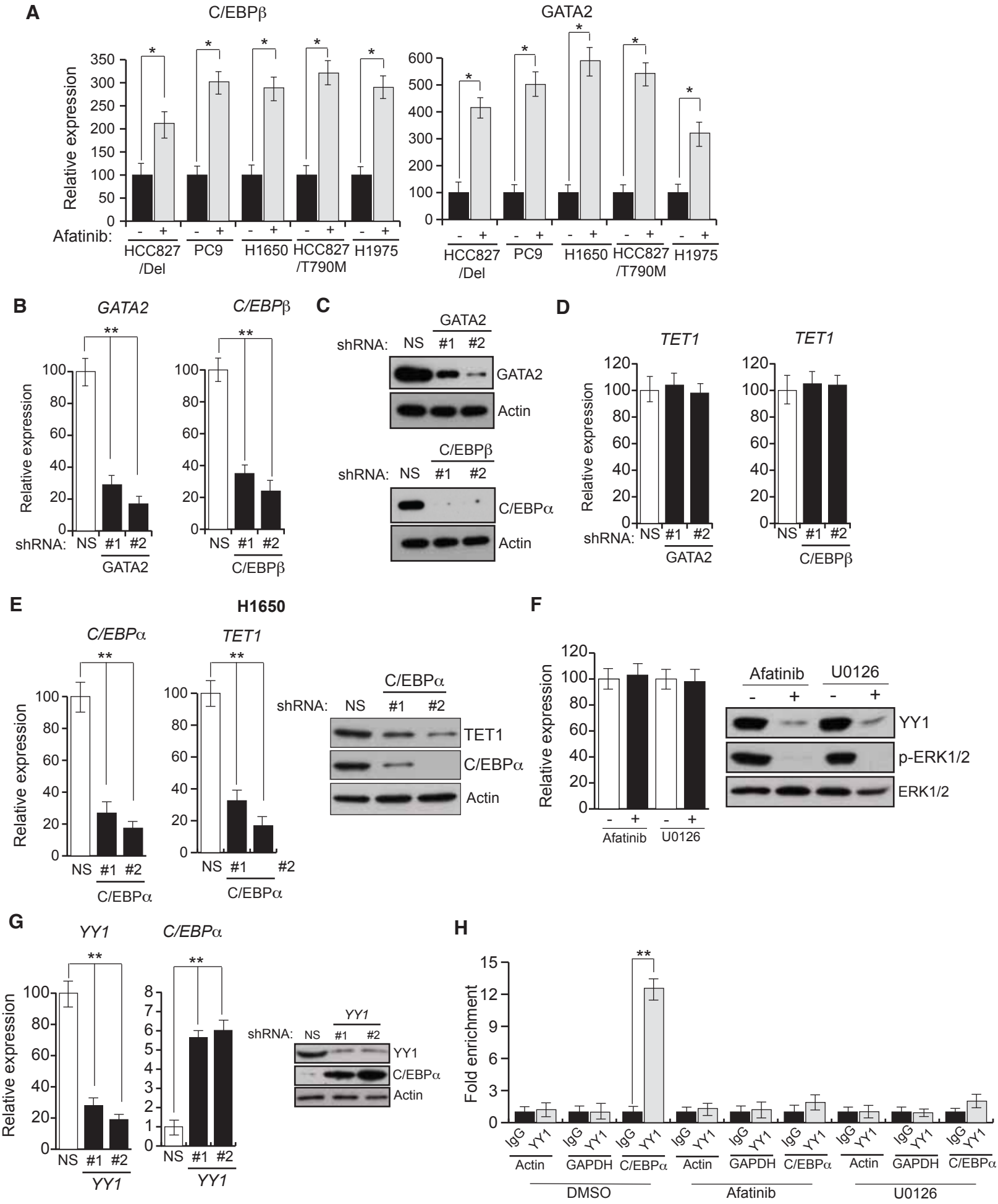
C/EBPα
Relative expression
G
TET1
Relative expression

Figure S4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C/EBPα
Actin
D
TET1
TET1
E
H1650
C/EBPα
TET1
F
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U0126
YY1
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ERK1/2
G
YY1
C/EBPα
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IgG
YY1
Actin
GAPDH
C/EBPα
DMSO
Afatinib
U0126

Figure S5

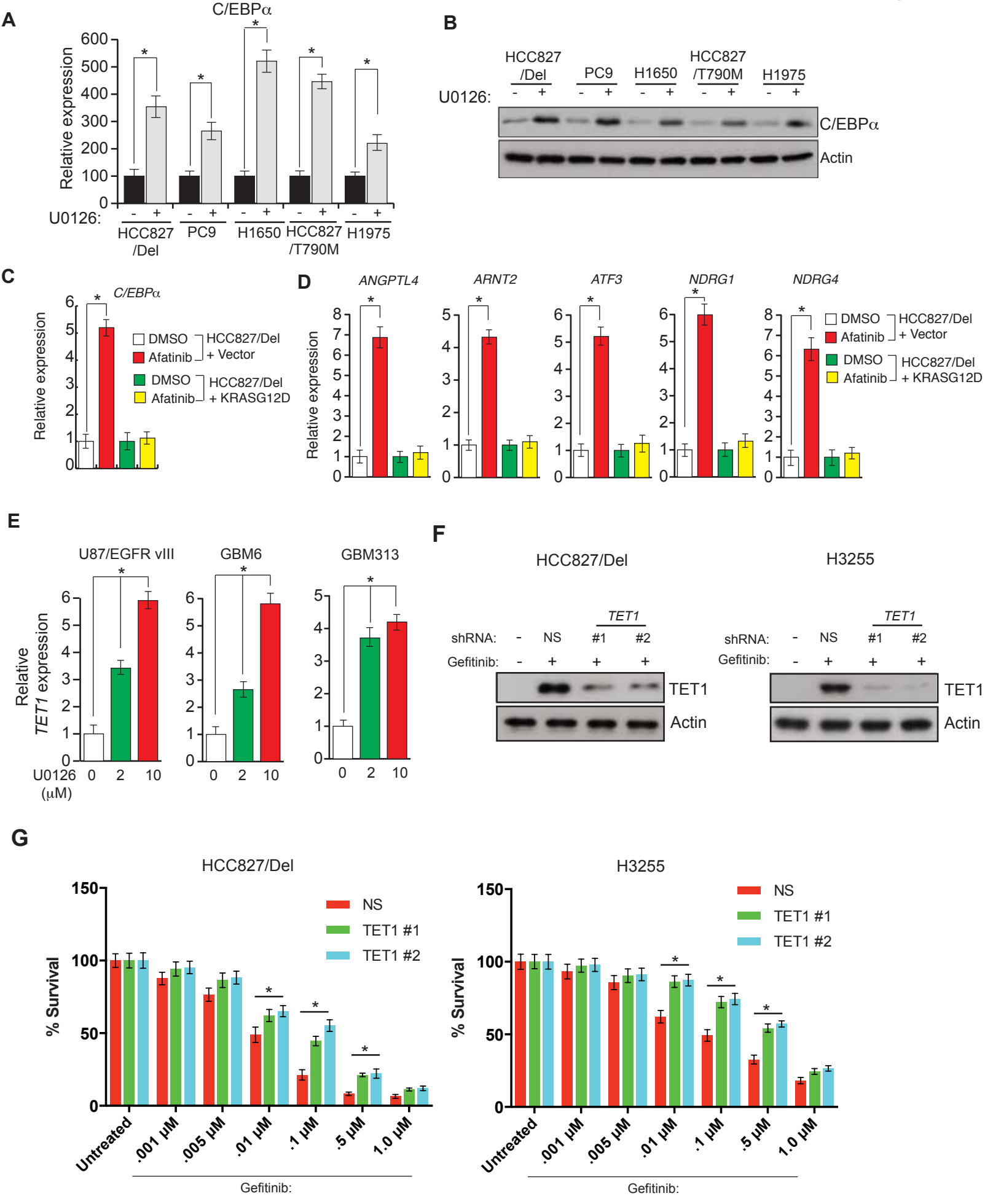

A
C/EBPα
Relative expression
U0126:
HCC827 /Del
PC9
H1650
HCC827 /T790M
H1975
B
HCC827 /Del
PC9
H1650
HCC827 /T790M
H1975
U0126:
C/EBPα
Actin
C
C/EBPα
Relative expression
DMSO
Afatinib
HCC827/Del + Vector
DMSO
Afatinib
HCC827/Del + KRASG12D
D
ANGPTL4
ARNT2
ATF3
NDRG1
NDRG4
Relative expression
DMSO
Afatinib
HCC827/Del + Vector
DMSO
Afatinib
HCC827/Del + KRASG12D
E
U87/EGFR vIII
GBM6
GBM313
Relative TET1 expression
U0126 (μM)
F
HCC827/Del
H3255
TET1
shRNA:
NS
#1
#2
Gefitinib:
TET1
Actin
G
HCC827/Del
H3255
% Survival
NS
TET1 #1
TET1 #2
Untreated
.001 μM
.005 μM
.01 μM
.1 μM
.5 μM
1.0 μM
Gefitinib:

Figure S6

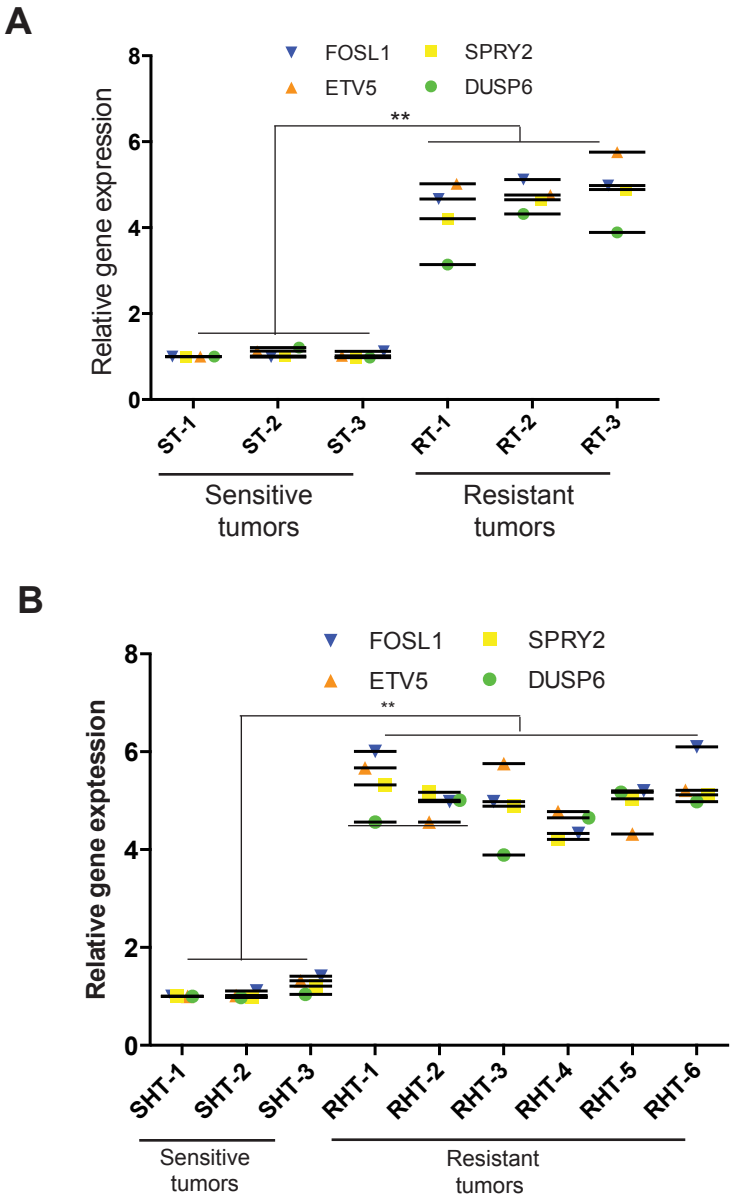
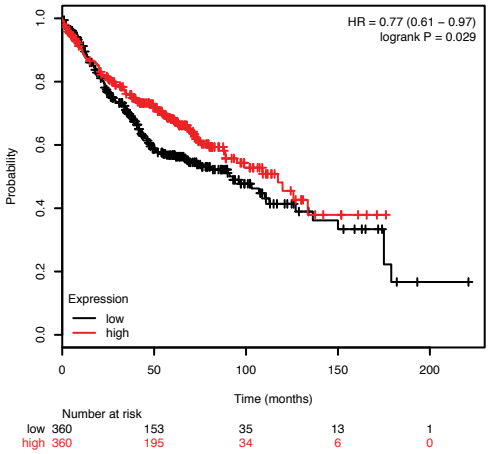
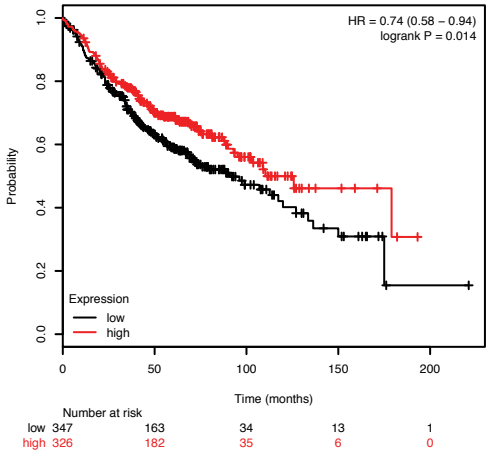

A
FOSL1
SPRY2
ETV5
DUSP6
**
Relative gene expression
0
2
4
6
8
ST-1
ST-2
ST-3
RT-1
RT-2
RT-3
Sensitive tumors
Resistant tumors
B
FOSL1
SPRY2
ETV5
DUSP6
**
Relative gene exptession
0
2
4
6
8
SHT-1
SHT-2
SHT-3
RHT-1
RHT-2
RHT-3
RHT-4
RHT-5
RHT-6
Sensitive tumors
Resistant tumors

Figure S7

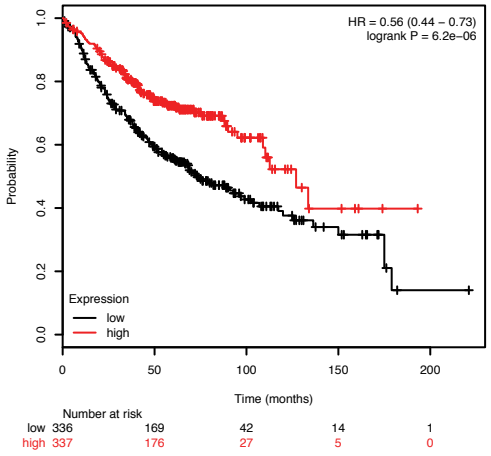
ATF3



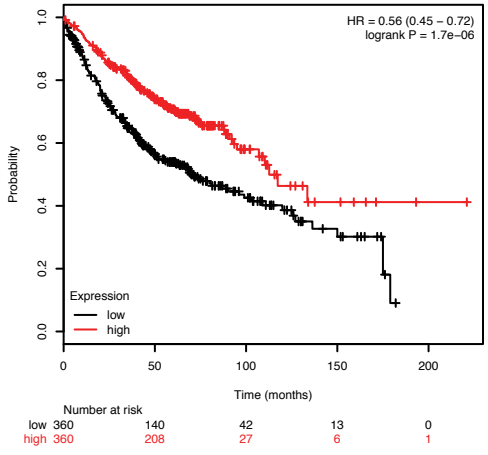
CCDC148



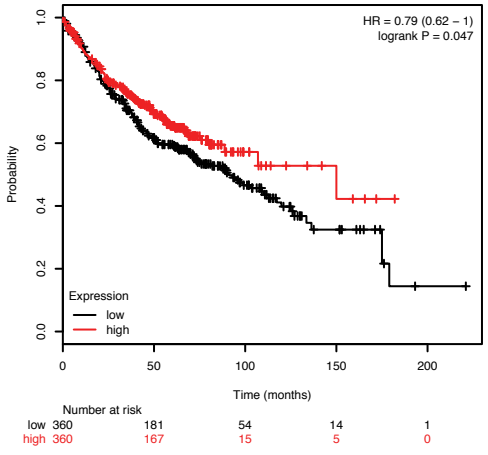
CREB5



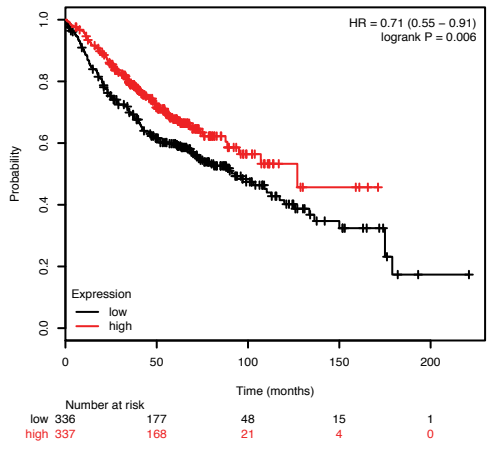
EMP1



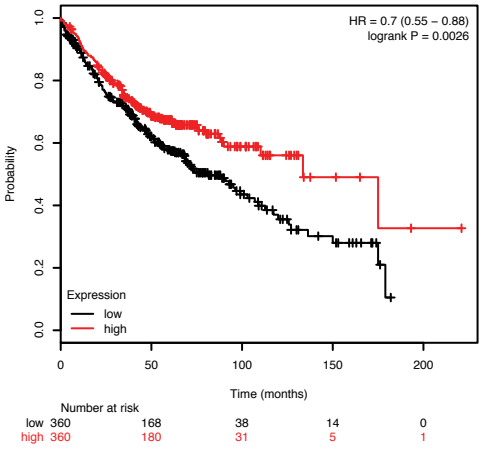
LPXN



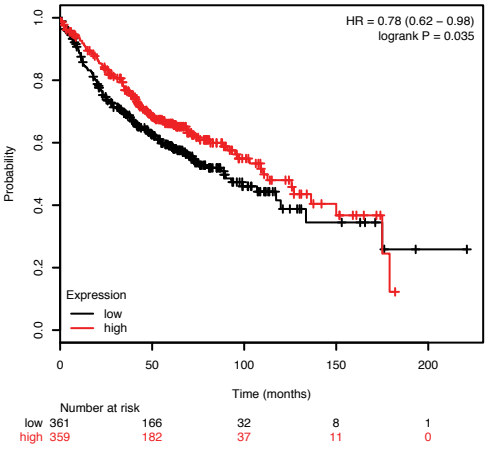
LRRC6



SH3KBP1



SPINK2



TRIM55