Supplemental Information Inventory

Supplemental Information accompanying this manuscript includes 7 supplementary figures, Supplemental Experimental Procedures and three Supplementary tables.

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

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).

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

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

Figure S5. *C/EBP*α 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).

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

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

Supplementary Experimental Procedures and related references.

Table S1. List of common genes that are upregulated in HCC827/Del and HCC827/Del-TM cell line after Decitabine and Vorinostat treatment.

Table S2: Transcription factor site prediction for TET1 promoter.

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.