Supplement Figure legends:

S1: Primary transcript qPCR indicates HDAC1 and 2 are regulated transcriptionally, HDAC3 is not.

S2: Analysis of golden gate methylation array identifies 42/807 genes hypermethylated after long term carcinogen exposure.

S3: Cycloheximide 2ug/ml final was added to cultures of 3kt or T31 cells for the indicated times (hours). Cycloheximide blocks the VPA induced degradation of DNMT1.

S4: HEK293 cells transiently transfected with pDest51-V5tagged HDAC1, 2 demonstrates protective role of HDACs on DNMT1. % indicates percent fold change of DNMT1 expression over parental cell.

S5: 3kt cells stably transfected with HDAC3 demonstrates protective role on DNMT1. % indicates percent fold change of DNMT1 expression over parental cell.

S6: Overexpression of HDAC1 or HDAC3 do not increase cellular proliferation compared to empty vector control.

S7: 6 tumor samples and matched resected histologically normal lungs were stained for PCNA. PCNA staining intensity is quantified by weighted index.

S8: AKT phosphorylation (S473) was assessed with and without VPA exposure in several different passages of carcinogen exposure. VPA had no effect on AKT phosphorylation.

S9: T31 wells were grown in triplicates in logarithmic phase and counted manually. VPA's effects on proliferation were assessed at 0.1 mM and 0.5 mM. No discernable effect of particularly the low dose of VPA on growth was observed.

S10: carcinogen-transformed T31 cells were passaged for 28 days in the presence of VPA (0.5mM). Bisulfite sequencing of the RASSF1 locus was performed on cells derived from day 0 and day 28. Long term VPA treatment of carcinogen transformed cells decreases methylation of RASSF1 promoter CpG island ($p < 0.05$).