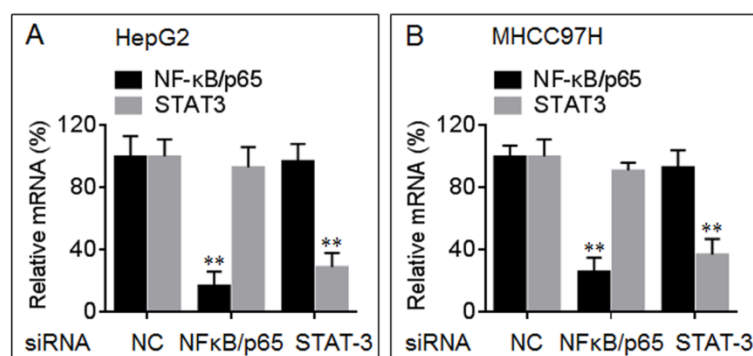


SUPPLEMENTARY DATA

Fig. S1. The efficiency of knock down of NF- κ B or STAT-3



(A) HepG2 and (B) MHCC97H cells were transfected by si-NC, si-NF- κ B/p65, or si-STAT-3 for 12 h. qRT-PCR analyses in triplicate of *NF- κ B/p65* and *STAT-3* mRNAs. **p < 0.01 compared with cells transfected by si-NC.

Fig. S2. The miR-124 binding sites in *NF- κ B* and *STAT-3* mRNAs

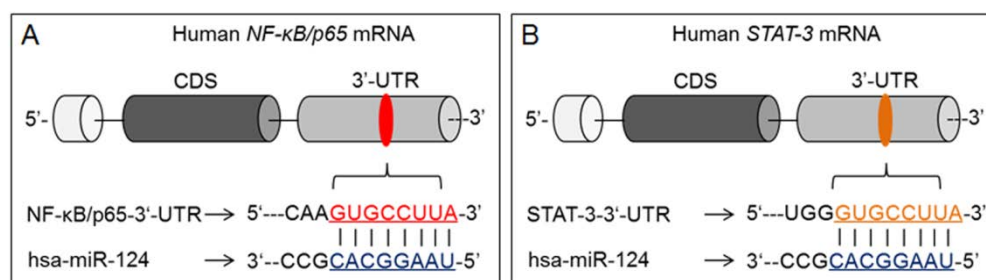
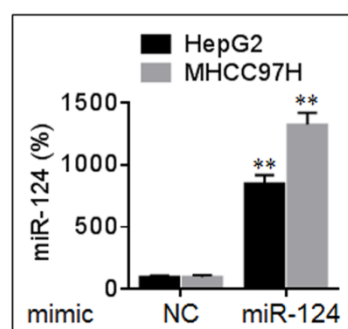
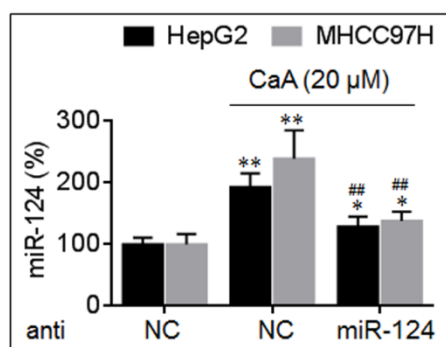


Fig. S3. The efficiency of overexpression of miR-124



HepG2 and MHCC97H cells were transfected by 20 nM mimic-NC or mimic-miR-124 for 12 h, qRT-PCR analyses in triplicate of the miR-124. **p < 0.01 compared with cells transfected by mimic-NC.

Fig. S4. The efficiency of knockdown of miR-124 in CaA-treated cells



After HepG2 or MHCC97H cells were pre-transfected by anti-NC or anti-miR-124 for 12 h, they were exposed to 20 μM CaA for 24 h. qRT-PCR analyses in triplicate of the miR-124 expression. * $p < 0.05$ and ** $p < 0.01$ compared with anti-NC-transfected cells treated with no CaA, ## $p < 0.01$ compared with anti-NC-transfected cells treated with 20 μM CaA.

Fig. S5. A pathway showing the demethylation effects induced by CaA metabolism

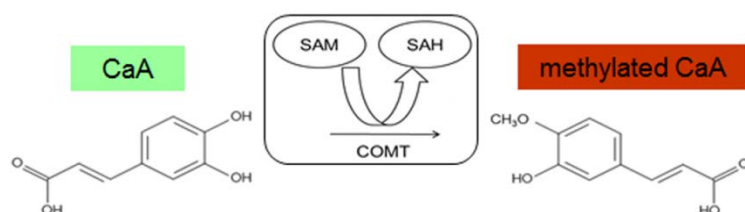
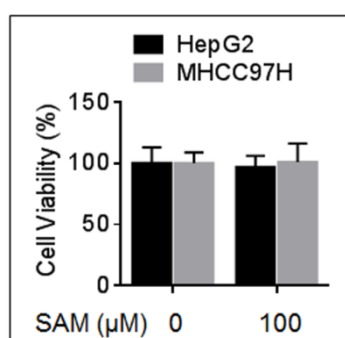


Fig. S6. Effects of SAM on the viability in HCC cells



HepG2 and MHCC97H cells were treated by 0 or 100 μM SAM for 24 h. The cell viabilities were evaluated in triplicate by WST-8 hydrolysis using a Cell Counting Kit-8 assay.