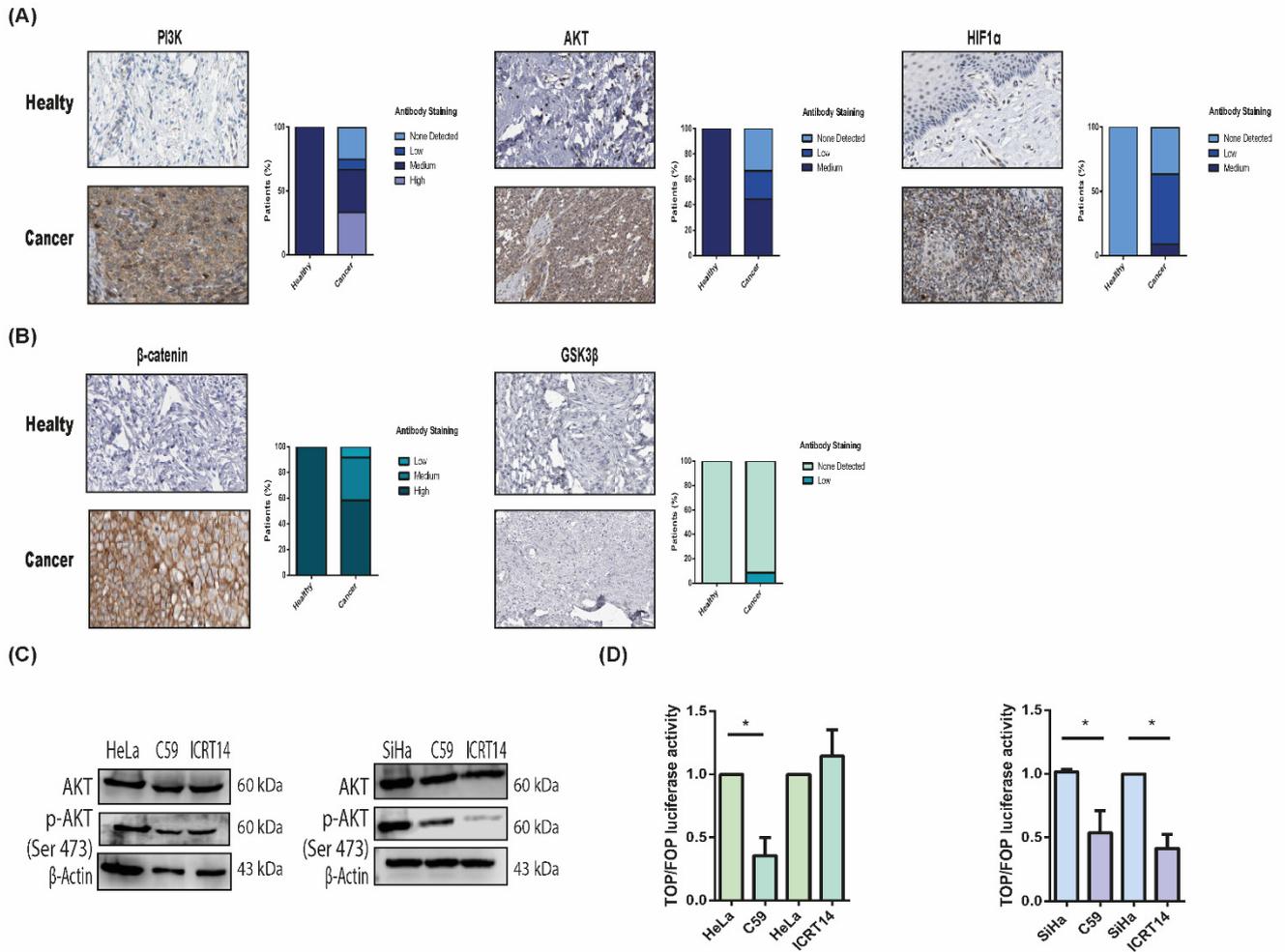
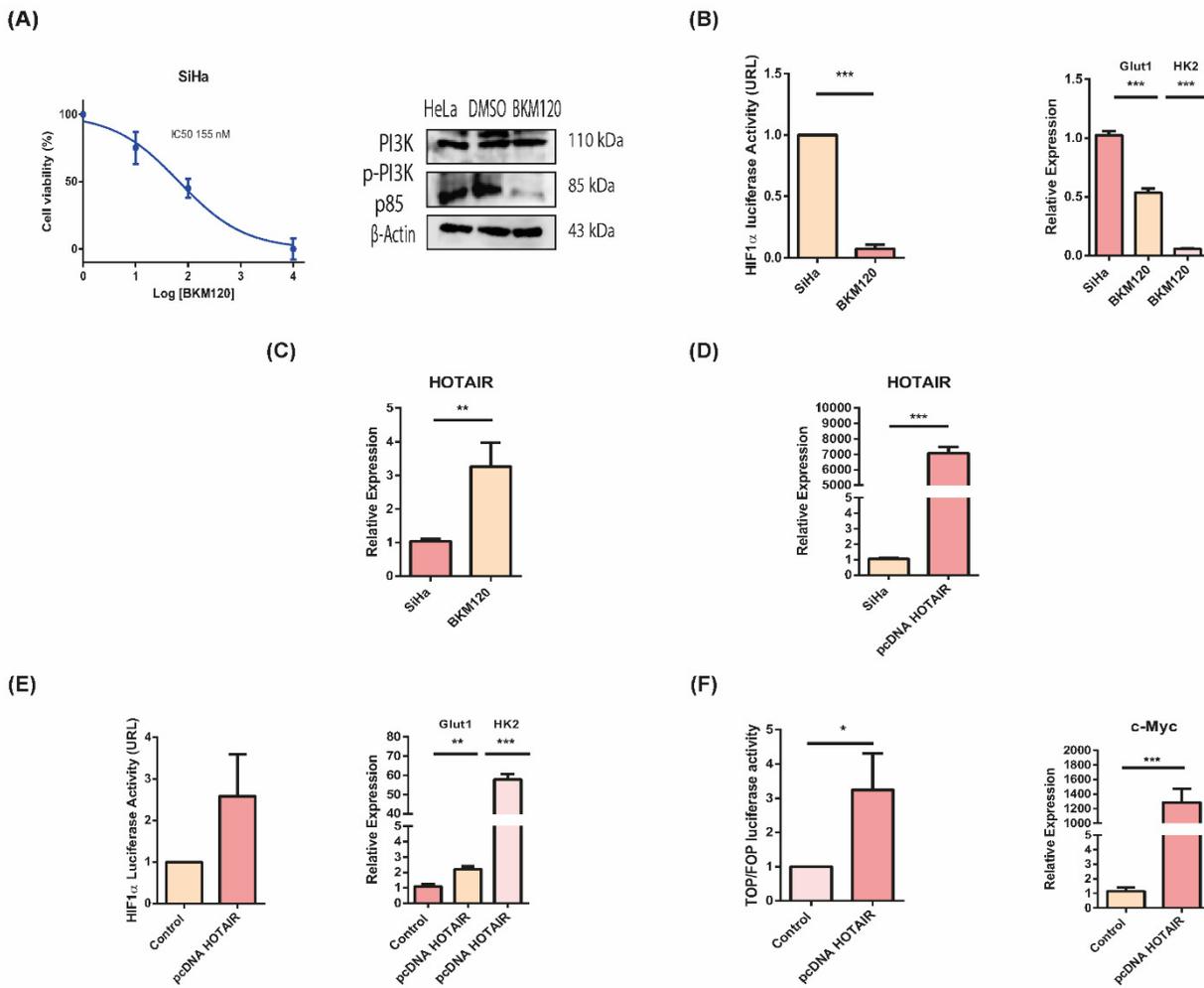


Supplementary Figures

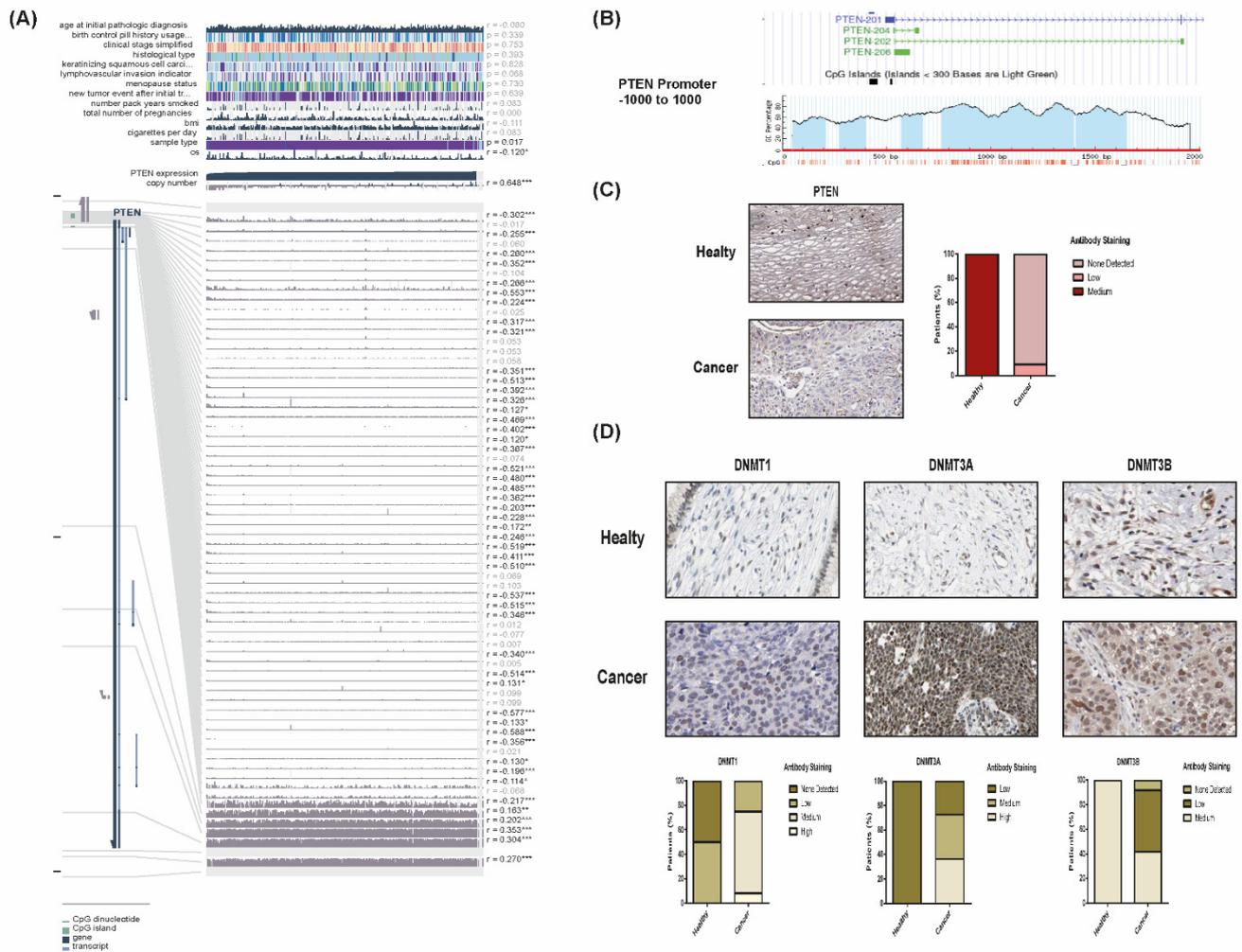


Supplementary Figure S1. Expression of key components of PI3K/AKT and Wnt/β-catenin in Cervical Cancer and normal tissues. (A-B) Protein atlas *In silico* meta-analysis to PI3K/AKT and Wnt/β-catenin signaling pathways effectors in CC (13) in comparison to healthy samples (3). **(C)** Western blots to PI3K/AKT pathway treated with C59 and ICRT14 Wnt/β-catenin inhibitors in CC cell lines. **(D)** TOP Flash assay for C59 and ICRT14 inhibitors in CC cell lines. $p < 0.05$ (*).



Supplementary Figure S2. PI3K/AKT regulates HOTAIR expression and Wnt/ β -catenin activation in SiHa cell line.

(A) IC₅₀ and Western Blot of PI3K/AKT pathway with BKM120 inhibitor. (B) BKM120 inhibits transcriptional activity of HIF1 α by measuring luciferase reporter and Glut1 and HK2 expression in SiHa cells. (C) HOTAIR expression with BKM120 inhibitor in SiHa cell line. (D) HOTAIR overexpression in SiHa cell line. (E) HOTAIR overexpression increases HIF1 α transcriptional activity by luciferase activity and HIF1 α targets expression. (F) HOTAIR overexpression increases Wnt/ β -catenin transcriptional activity evaluated by TOP Flash activity and c-Myc expression. $p < 0.05$ (*). $p < 0.01$ (**). $p < 0.001$ (***)



Supplementary Figure S3. PTEN promoter is methylated in Cervical Cancer. (A-B) In silico analysis to PTEN methylation pattern on 317 CC samples and sequence analysis through MEXPRESS data base and Genome browser-MethPrimer respectively. **(C-D)** Protein atlas In silico meta-analysis to PTEN and DNMT1, 3A and 3B in CC (13) in comparison to healthy samples (3).