# *Additional file*

**Additional file 1: Table S1. Clinicopathological characteristics of the prostate tissue samples.**

**Additional file 2: Table S2. List of antibodies used in this study.**

**Additional file 3: Table S3. Sequences of primers and siRNA (human).**

**Additional file 4: Fig. S1** BS analysis of RWPE-1, LNCaP, DU145 and PC-3 cells after using BS primer 1. One point represents one CG locus, in which black point represents methylated CG site and white point represents unmethylated CG site. One horizontal row represents one clone and one vertical row represents one CG site. Finally, ten clones were selected randomly.

**Additional file 5: Fig. S2** DNA methylation level of CAMK2N1 in TCGA dataset and PCa tissues. **A** DNA methylation level of CAMK2N1 in PCa patients with different T or N stages, Gleason scores and PSA values from TCGA database. **B** The quantification of pyrosequencing results at site 6 in BPH and PCa tissues. PCa patients were divided into TNM stage 2 and stage 3-4 groups, Gleason score 6, 7 and 8-10 groups, PSA value < 10 ng/ml, 10-20 ng/ml and > 20 ng/ml groups (n=16-52). **C** Pyrosequencing results at site 7. **D** Pyrosequencing results at site 8. **E** DNA methylation level of CAMK2N1 at cg22942704 locus in PCa patients with different T or N stages, Gleason scores and PSA values from TCGA database. Data are presented as mean ± SD, t test, one-way ANOVA test and log rank test were used, *P < 0.05.

**Additional file 6: Fig. S3** The immunofluorescence staining of **A** CAMK2N1 (Cy3, red) and **B** DNMT1 (FITC, green) in DMSO-treated and 20 μM 5-Aza-CdR-treated DU145 cells. The nuclei were stained with DAPI. Scale bars represent 50 μm.

**Additional file 7: Fig. S4.** CAMK2N1 inhibits the expression of DNMT1 via AKT or ERK signaling pathway without the changes of genome-wide DNA methylation level in PCa cells. **A** LNCaP cells were transfected with CAMK2N1 cDNA clones. After 2 days, the mRNA expression of DNMT1 and CAMK2N1 was analyzed by qRT-PCR (GAPDH was used as a control; n=3). **B** The expression of CAMK2N1, DNMT1, p-AKT, AKT, p-MEK1, MEK1, p-ERK1/2, ERK1/2 was determined by western blot with CAMK2N1 overexpression in LNCaP cells (GAPDH was used as a control; n=3). **C, D** CAMK2N1 knockdown (si-CA) LNCaP cells were treated with 10 μM AKT signaling pathway inhibitor AKTi for 1 day. The expression of DNMT1, p-AKT and t-AKT was analyzed by qRT-PCR and western blot (GAPDH was used as a loading control, n=3). **E, F** CAMK2N1 knockdown (si-CA) LNCaP cells were treated with 10 μM ERK signaling pathway inhibitor U0126 for 1 day. The expression of DNMT1, p-AKT, t-AKT, p-ERK1/2 and t-ERK1/2 was analyzed by qRT-PCR and western blot (GAPDH was used as a loading control, n=3). **G** DU145 cells were transfected with CAMK2N1 siRNA and cDNA clones. After 2 days, DNA was extracted and the genome-wide DNA methylation level was assessed by a Methylated DNA Quantification Kit (n=6-7). Data are presented as mean ± SD, one-way ANOVA test were used, *P < 0.05, **P < 0.01 and ***P < 0.001.