

Molecular targeting of EHMT2 in pancreatic ductal adenocarcinoma

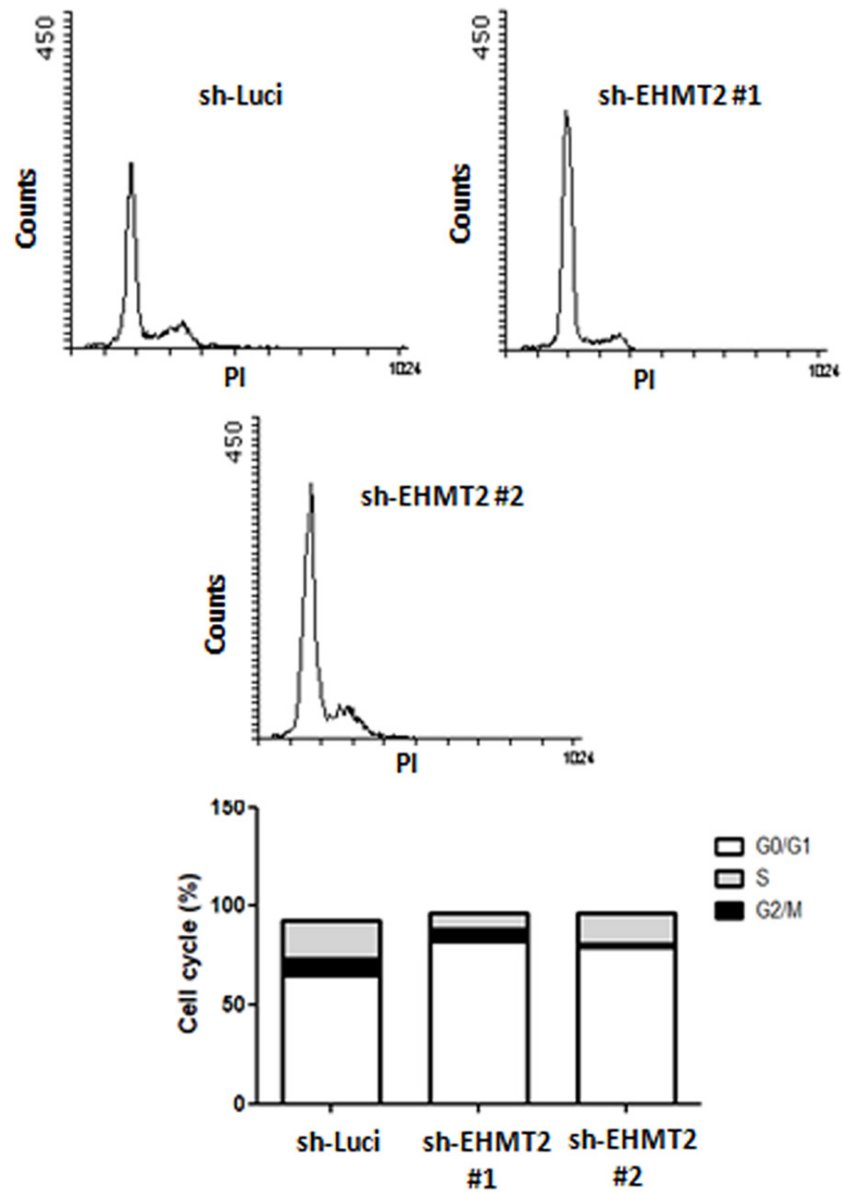
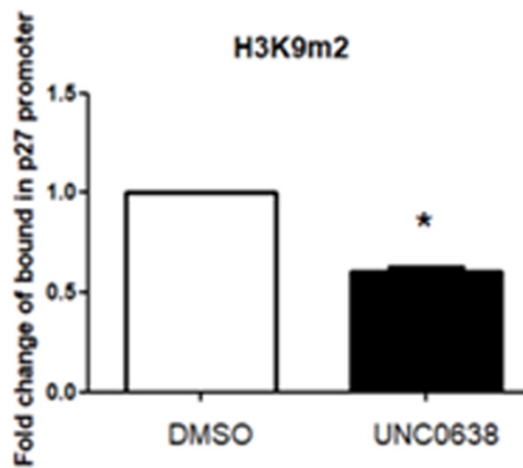


Figure S1. Expression profiles of cell cycle in Mia PaCa-2 and Mia PaCa-2 EHMT2 deficient (sh-EHMT2) cell lines were performed by flow cytometry.



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Figure S2. ChIP-qPCR analysis was performed to determine the status of H3K9m2 in the p27 gene promoter in Mia PaCa-2 cells treated with UNC0638.

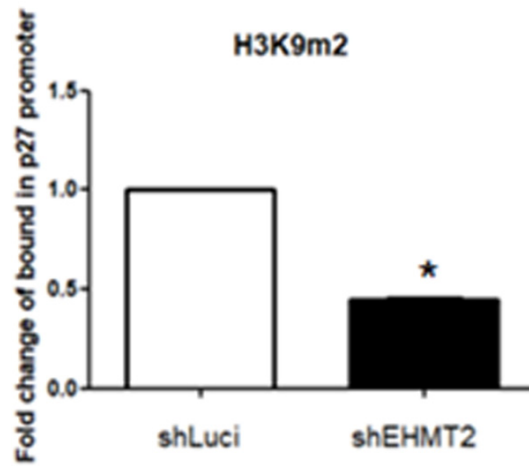


Figure S3. ChIP-qPCR analysis was used to determine the status of H3K9m2 in the p27 gene promoter in Mia PaCa-2 or EHMT2 knockdown cells.

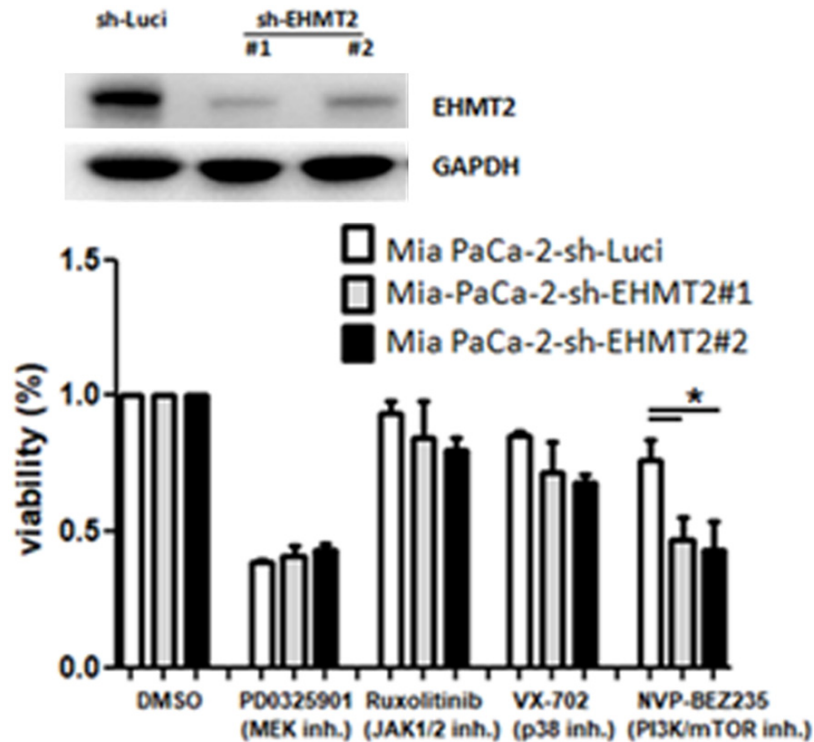


Figure S4. Parental Mia PaCa-2 cells (sh-Luci) and EHMT2-depleted cells (sh-EHMT2 #1 and sh-EHMT2 #2) were treated with different compounds for 72 h. Cell viability was determined by MTT assay (* $P < 0.05$). Protein levels of EHMT2 were detected by western blot analysis.

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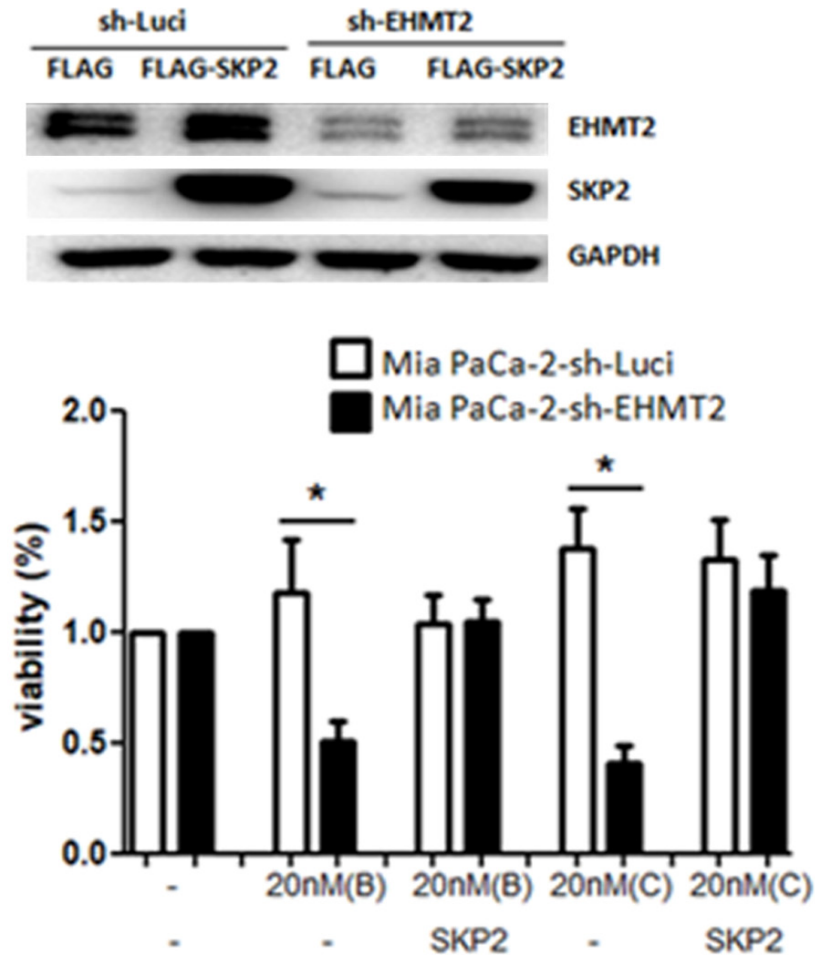


Figure S5. Parental Mia PaCa-2 (sh-Luci) and EHMT2-deficient (sh-EHMT2) cells were transfected with FLAG or FLAG-SKP2 plasmids and treated with dual PI3K/mTOR inhibitor (NVP-BEZ235, B and Cay10626, C) for 72 h. Cell viability was determined by MTT assay. The protein level of EHMT2 was detected by western blot analysis. Statistical comparisons were evaluated by a one-way ANOVA (* $P < 0.05$).