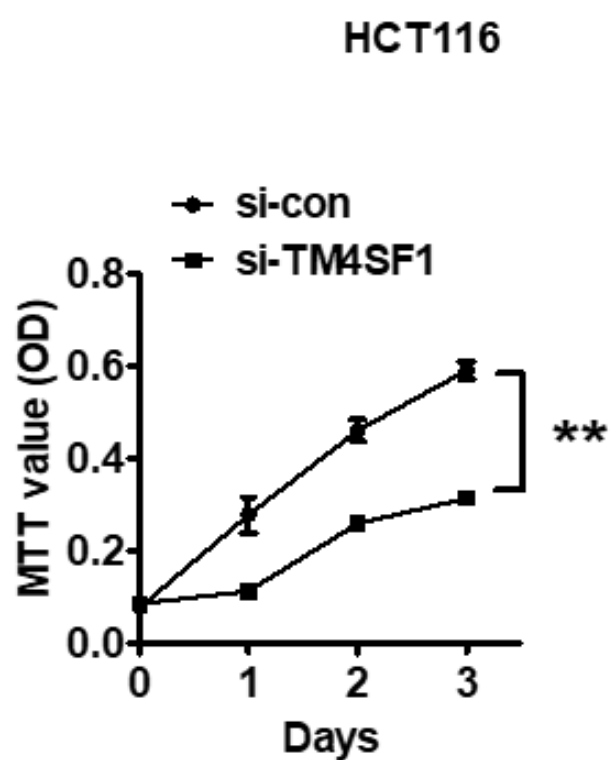
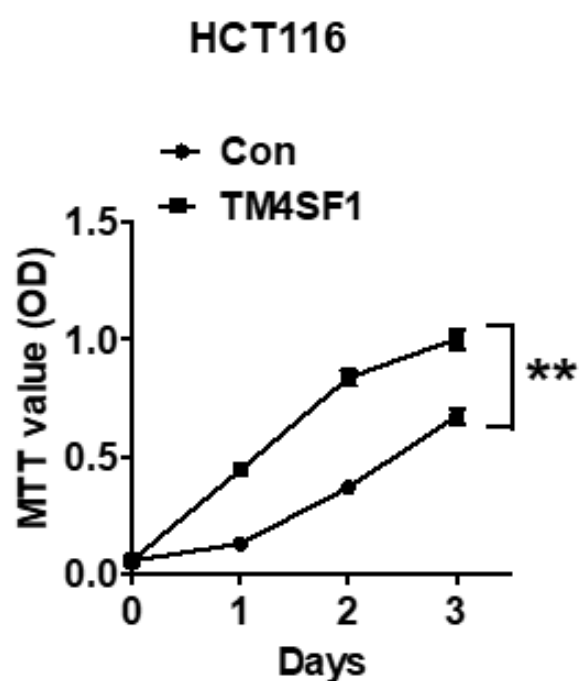
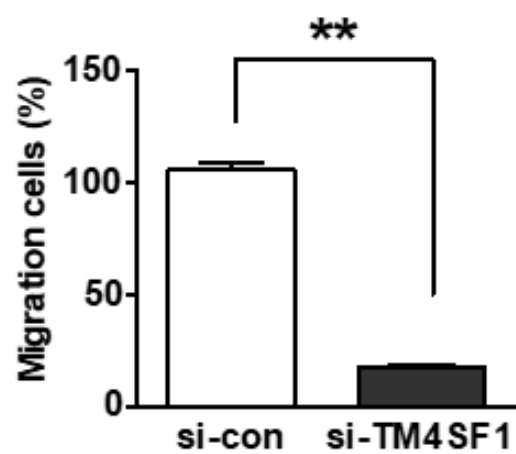
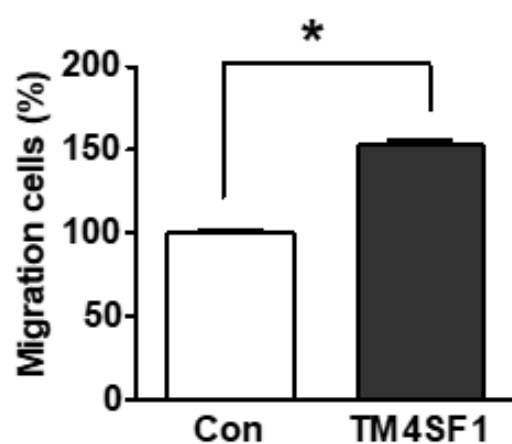
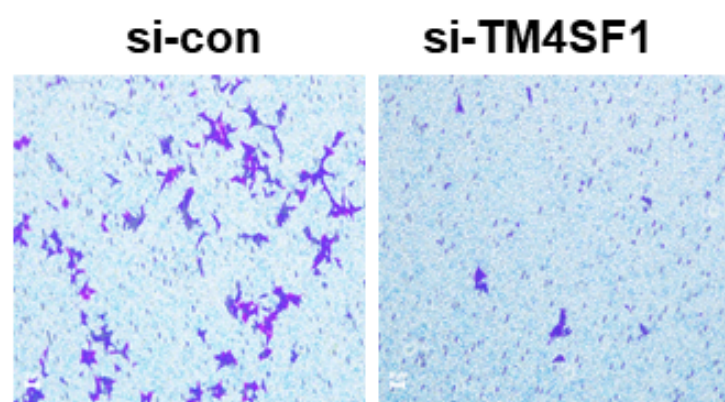
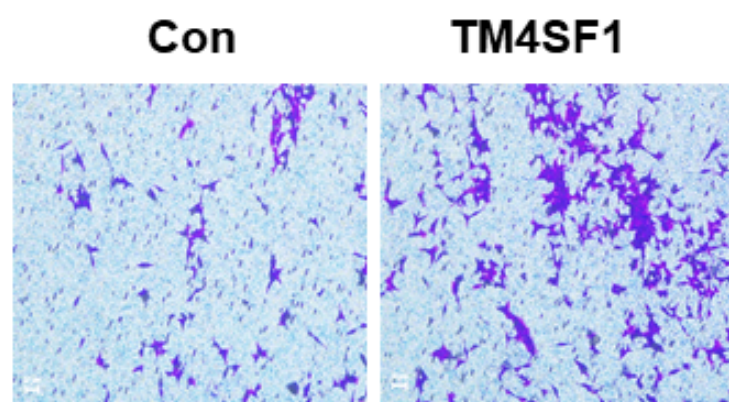


Supplementary figure 1

A

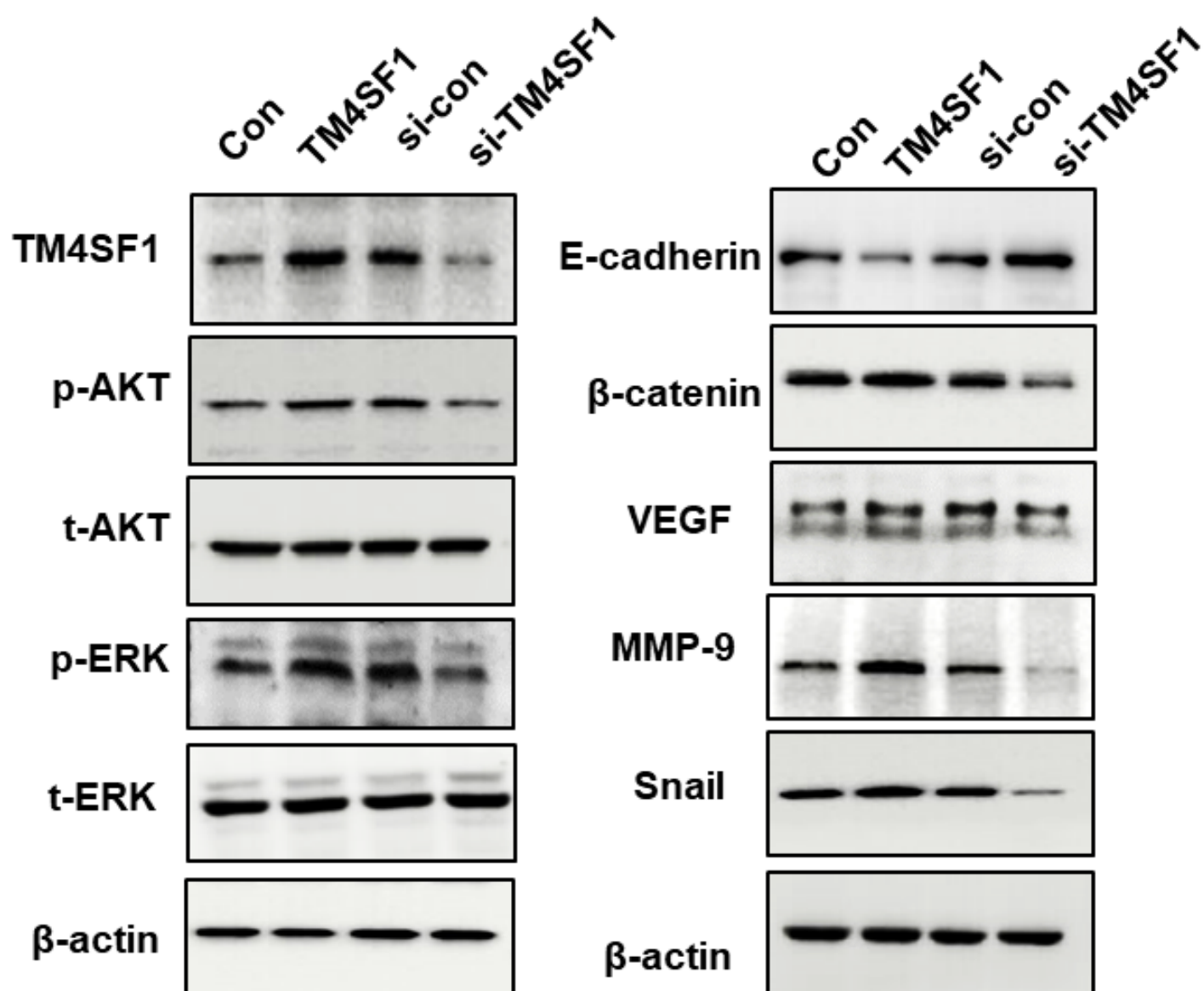


B



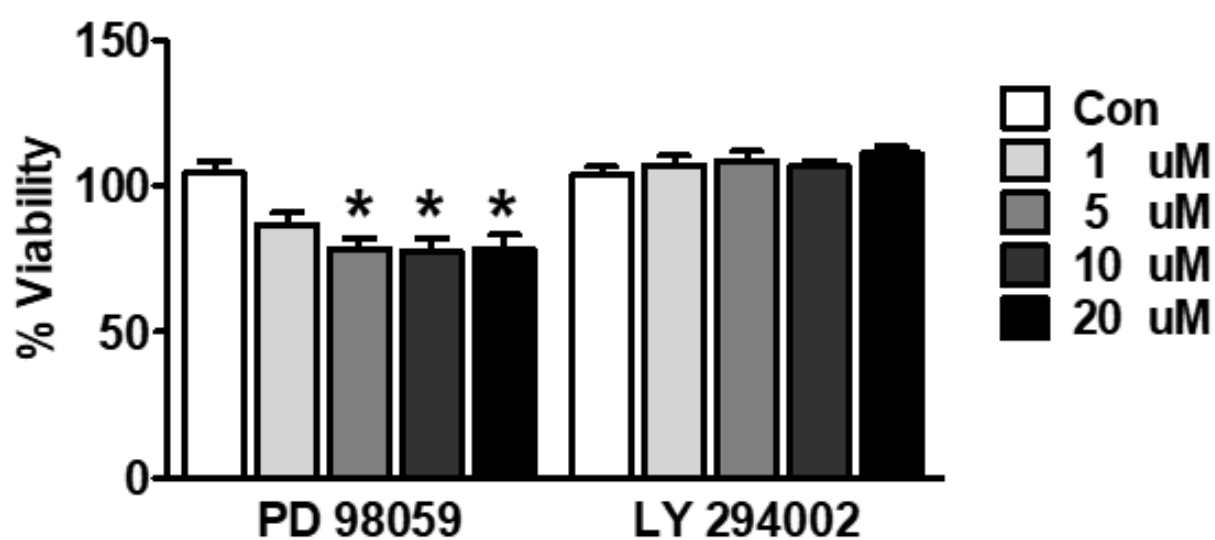
Supplementary figure 1

C

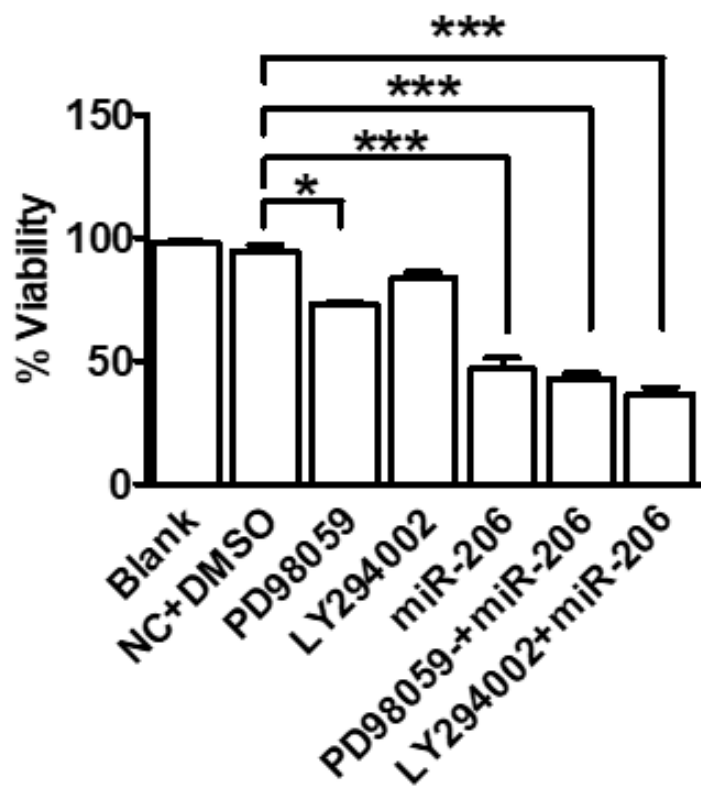


Supplementary figure 2

A

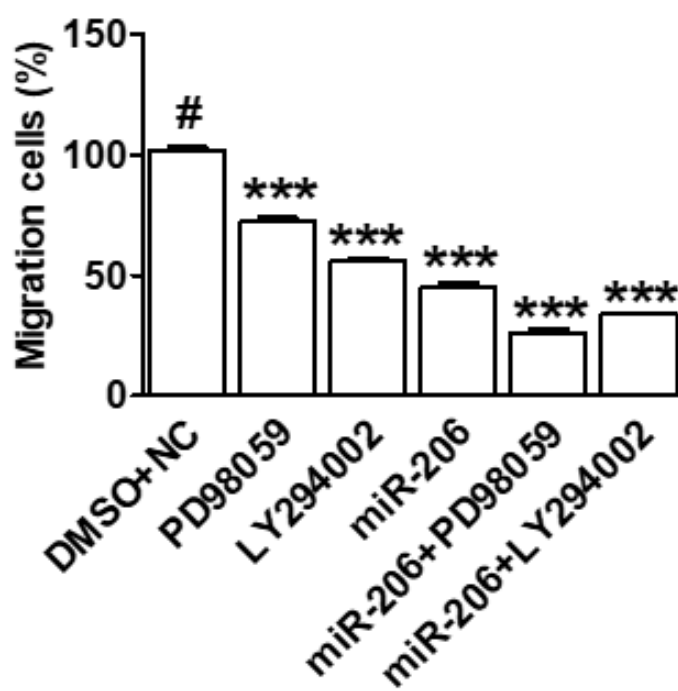
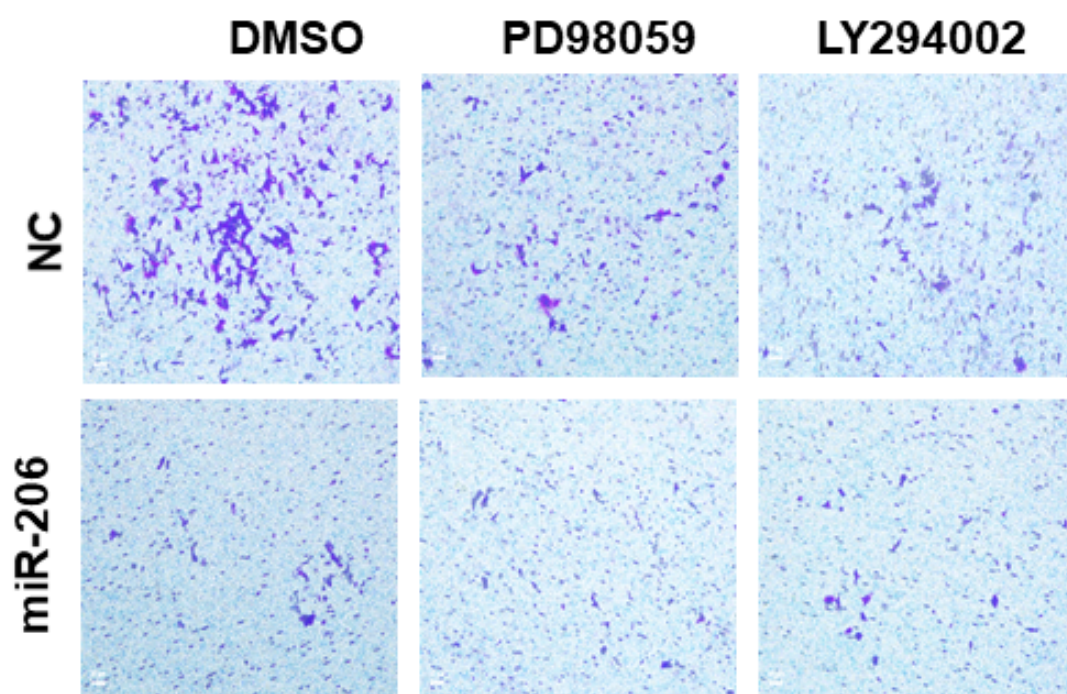


B



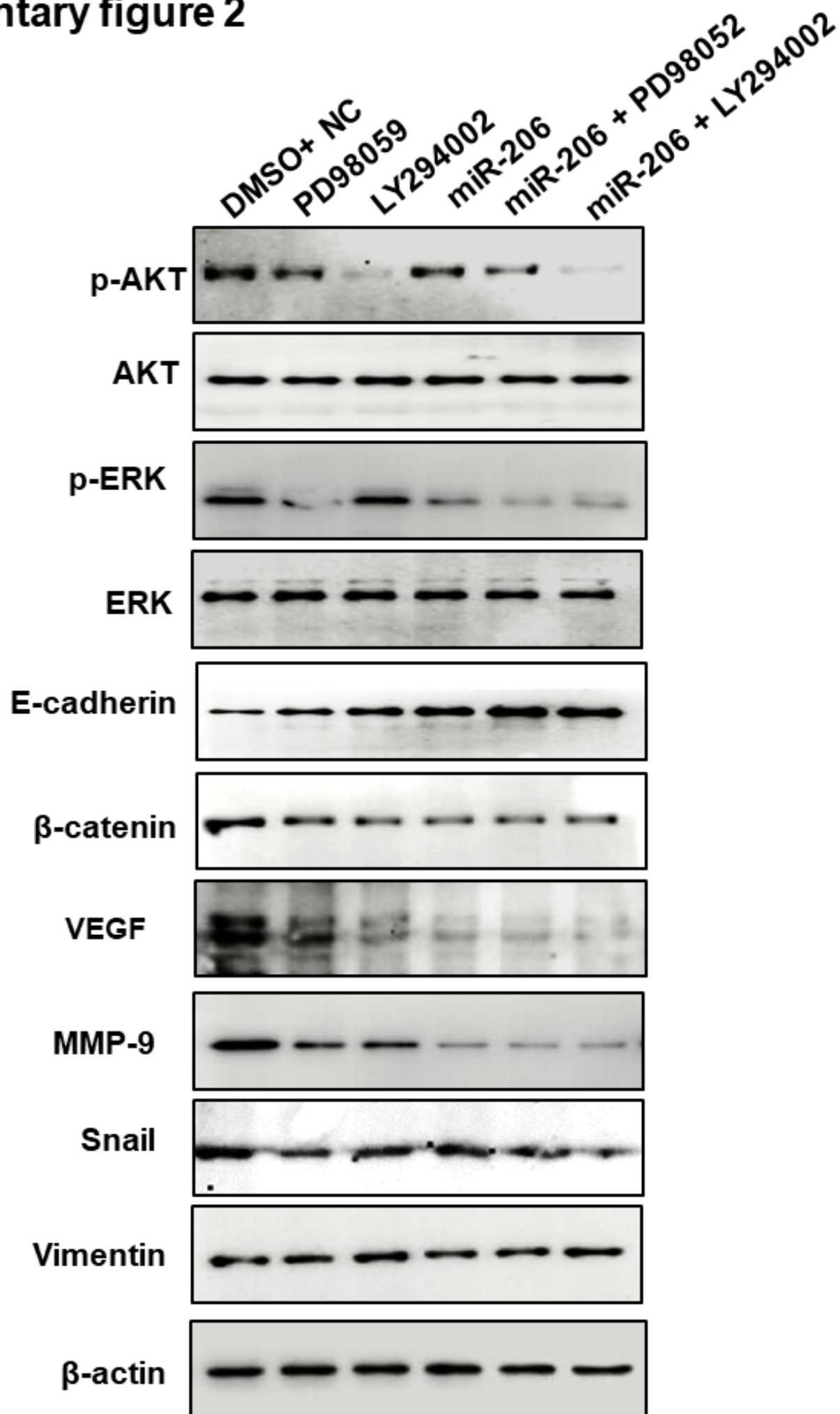
Supplementary figure 2

C



Supplementary figure 2

D



Supplementary figure 1.

TM4SF1 promotes proliferation and migration of colon cancer cells.

(A) HCT116 cells were transfected with TM4SF1 plasmid or si-TM4SF1 for 0, 1, 2, 3 days. The cell proliferation was determined using MTT assay. (B) The transwell migration assay was performed on HCT116 cells transfected with TM4SF1 plasmid or si-TM4SF1. (C) Western blot analysis was used to measure the protein levels of EMT markers or p-AKT and p-ERK signaling pathway in HCT116 cells. β -actin was detected as a loading control. * $p < 0.05$, ** $p < 0.01$.

Supplementary figure 2.

miR-206 decreases PGE2-induced cell proliferation, migration, and EMT process through p-AKT and p-ERK pathway on colon cancer cells.

(A) HCT116 cells were treated with PD98059 (MEK inhibitor) or LY294002 (PI3K inhibitor) under various concentration (0, 1, 5, 10, 20 μ M) for 24 h. The cell viability was determined by MTT assay. * $p < 0.05$ compared with con (0 μ M). HCT116 cells were transfected with miR-206 for 48 h and treated with PD98059 (5 μ M) or LY294002 (10 μ M) for 24 h, and then PGE2 was treated for 12 h, and then determined using MTT assay (B) and transwell migration assay (C). (D) The protein levels of p-AKT, p-ERK, E-cadherin, β -catenin, VEGF, MMP-9, Snail and Vimentin in HCT116 cells were detected by performing immunoblotting. The data are presented as mean \pm S. D. of three independent experiments.