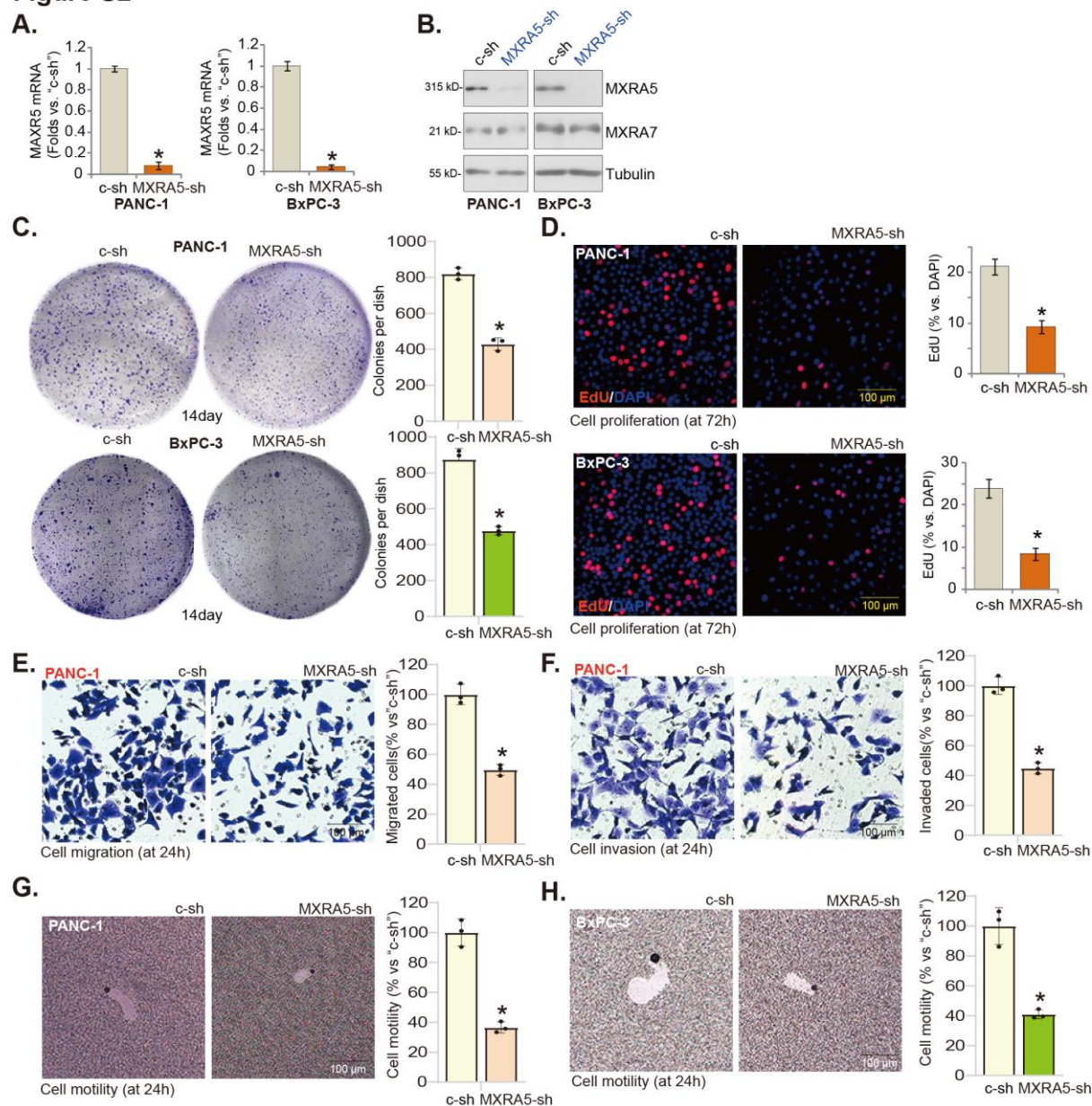


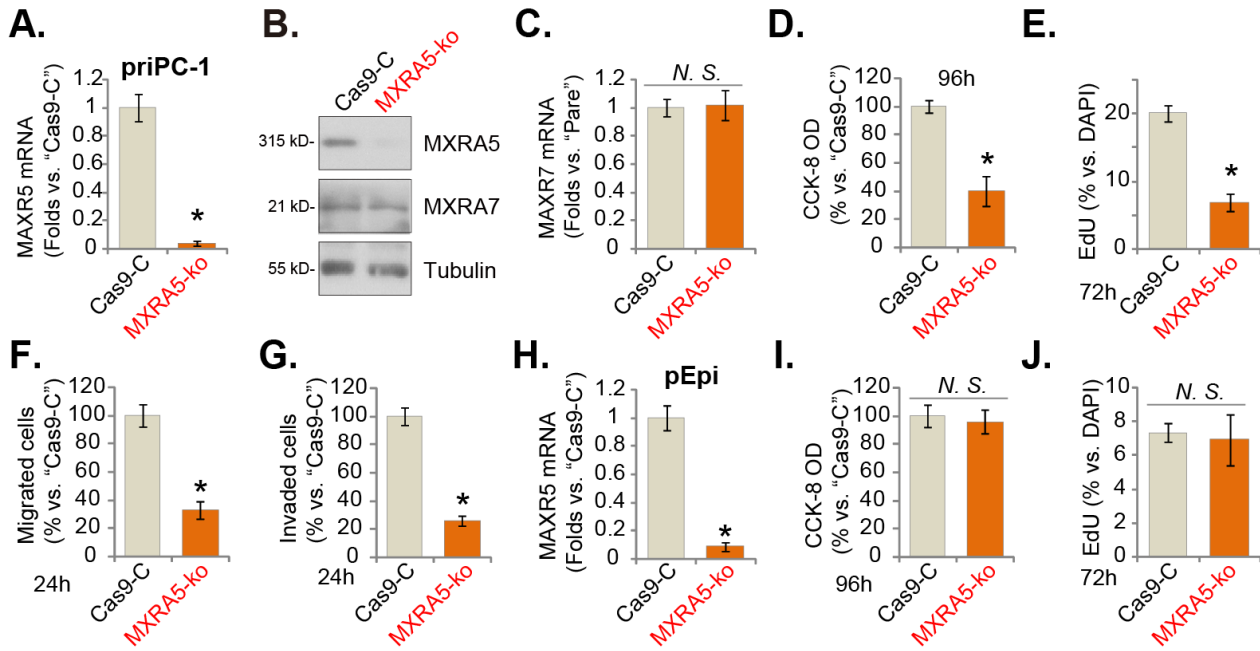


**Figure S2**



**Figure S2. MXRA5 downregulation exerts anti-tumorigenic activity in human pancreatic cancer cells.** Puromycin-selected pancreatic cancer cell lines (PANC-1 and BxPC-3), with MXRA5-sh-S1 ("MXRA5-sh") or the scramble control shRNA ("c-sh"), were cultured, and expression of listed genes and proteins was shown (A and B); Cells were further cultured for applied time periods, colony formation (C) and cell proliferation (D) as well as cell migration, invasion (E and F) and motility (G and H) were tested by the described assays, with results quantified. Data were presented as mean  $\pm$  standard deviation (SD, n= 5). \* $P < 0.05$  vs. "c-sh" cells. All experiments were repeated for five times with similar results obtained. Scale bar = 100  $\mu\text{m}$ .

**Figure S3**

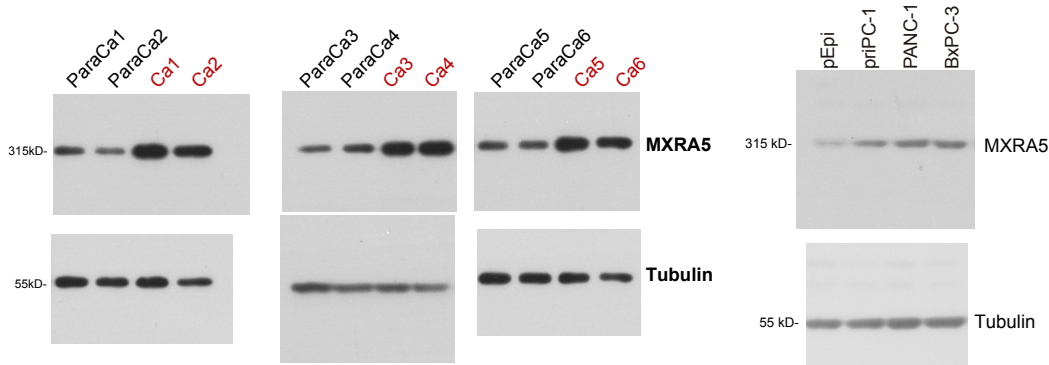


**Figure S3. MXRA5 knockout potently inhibits pancreatic cancer cell progression *in vitro*.**

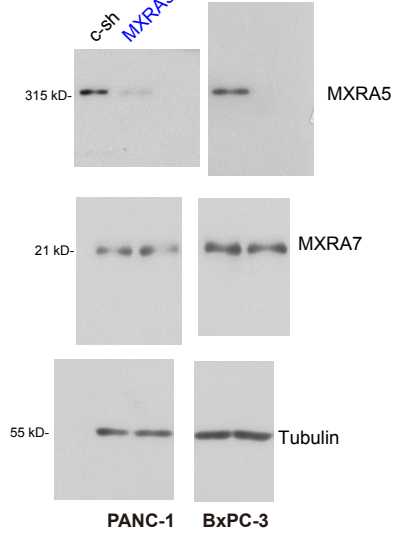
Puromycin-selected priPC-1 primary human pancreatic cancer cells (A-G), with sgRNA-CRISPR/dCas-9 MXRA5-KO lentiviral construct ("MXRA5-ko") or Cas9 control construct ("Cas9-C"), were established, and expression of listed genes and proteins was shown (A-C); Cells were further cultivated for indicated time periods, and cell viability (D), and EdU incorporation (E) as well as *in vitro* cell migration (F) and invasion (G) were tested using the described methods, and results were quantified. Puromycin-selected pEpi epithelial cells, expressing the sgRNA-CRISPR/dCas-9 MXRA5-KO lentiviral construct ("MXRA5-ko") or Cas9-C construct, were established. *MXRA5* mRNA expression was shown (H). Cells were further cultivated for indicated time periods, cell viability (I) and proliferation (J) were examined, and results were quantified. Error bars stand for mean  $\pm$  standard deviation (SD, n=5). \*  $P < 0.05$  versus "Cas9-C" cells. "N.S." stands for  $P > 0.05$ . Experiments in this figure were repeated five times.

**Figure S4: The uncropped blotting images of the study**

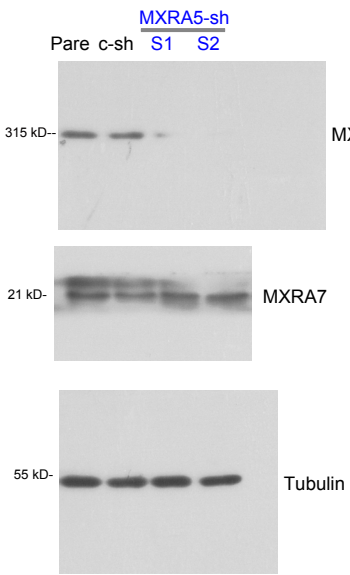
**Figure 1.**



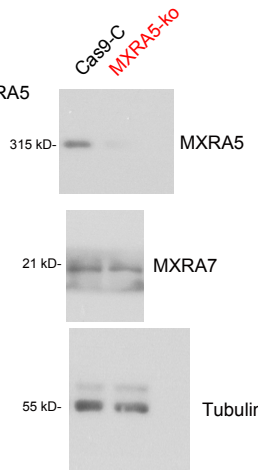
**Figure S2**



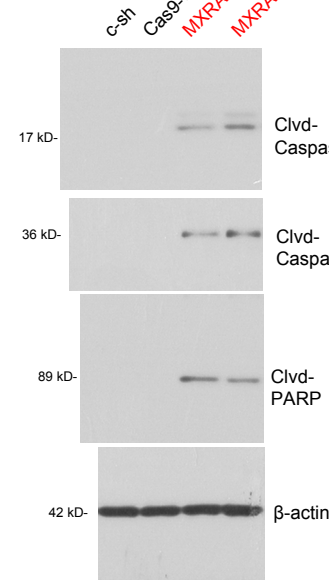
**Figure 2**



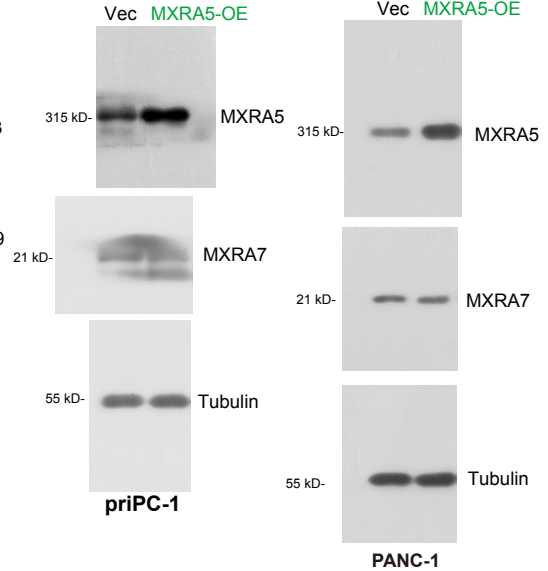
**Figure S3**



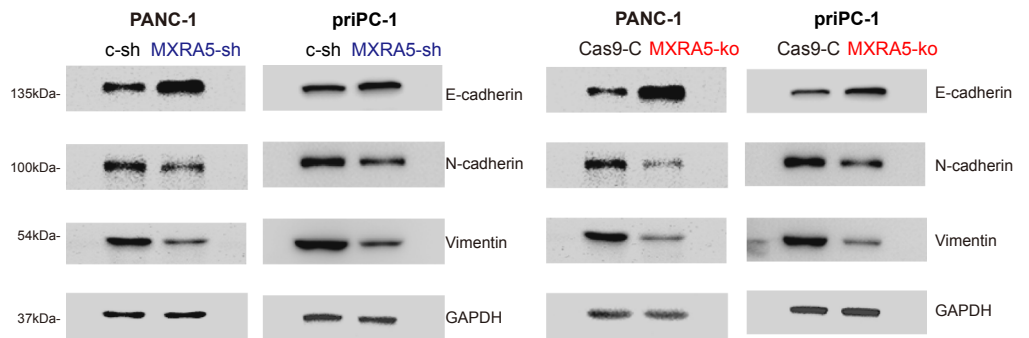
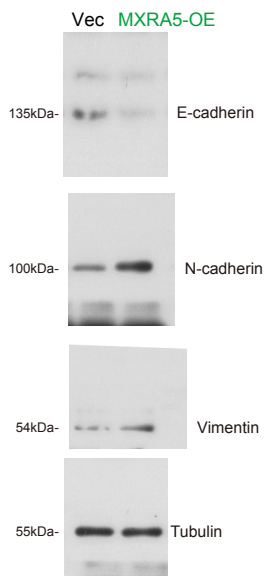
**Figure 3**



**Figure 4**



**Figure 5.**



**Figure 6.**

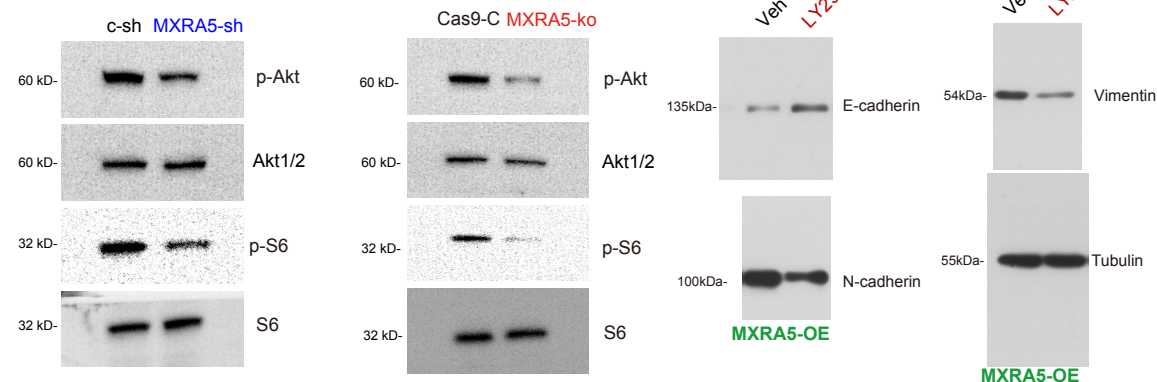


Figure S5.

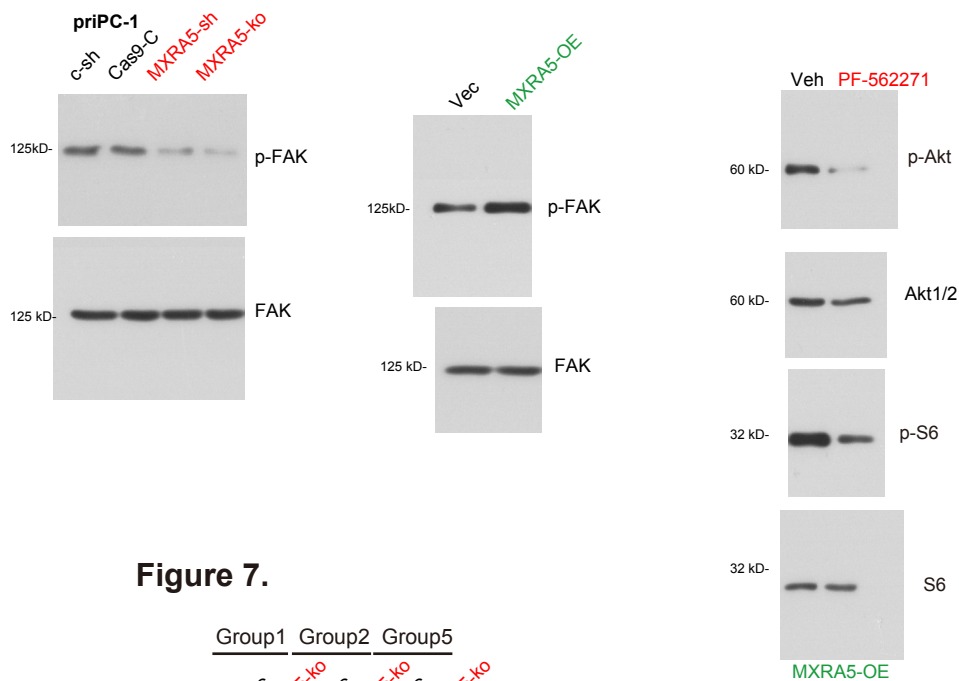


Figure 7.

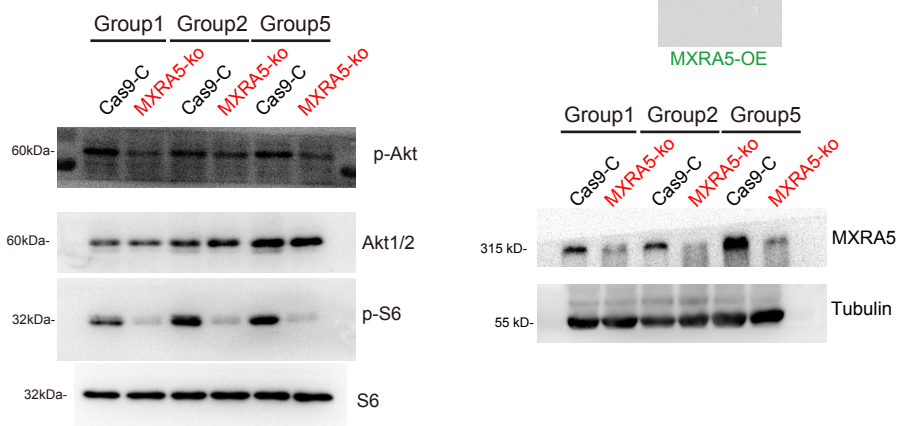
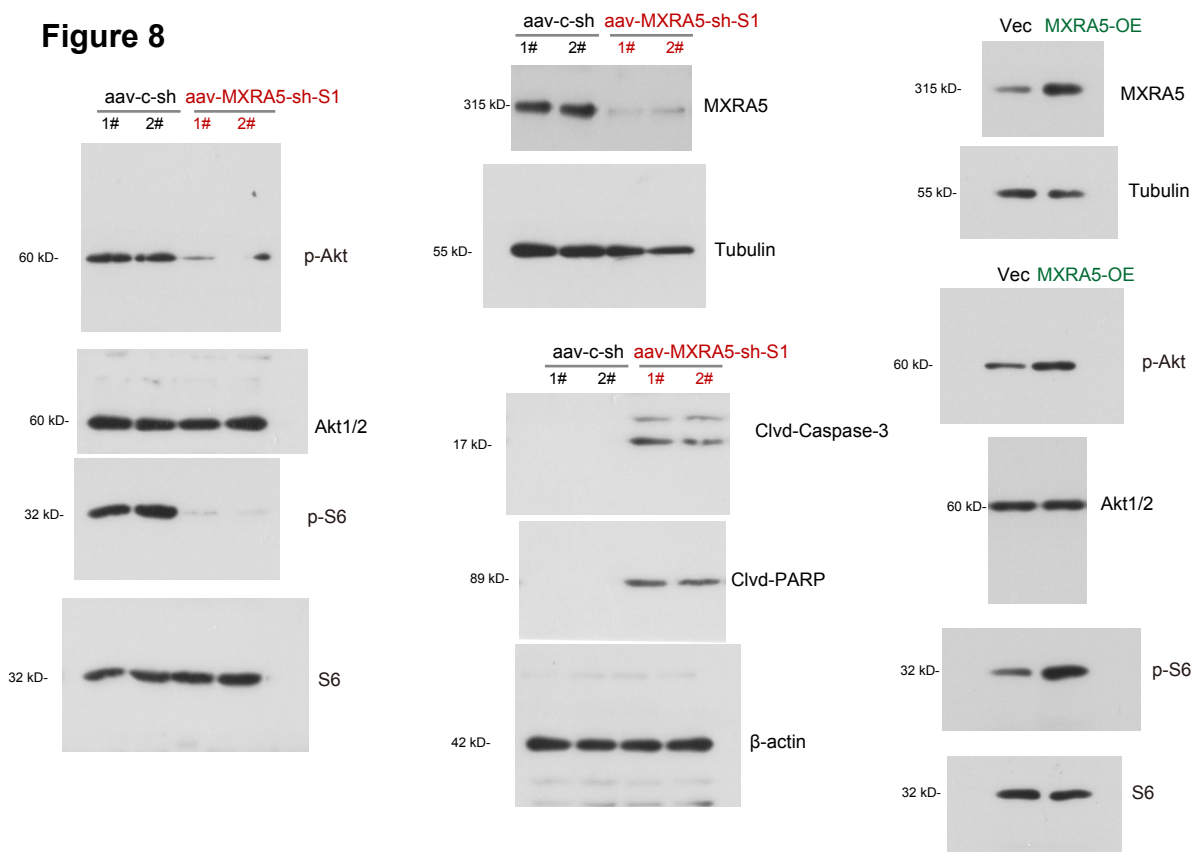
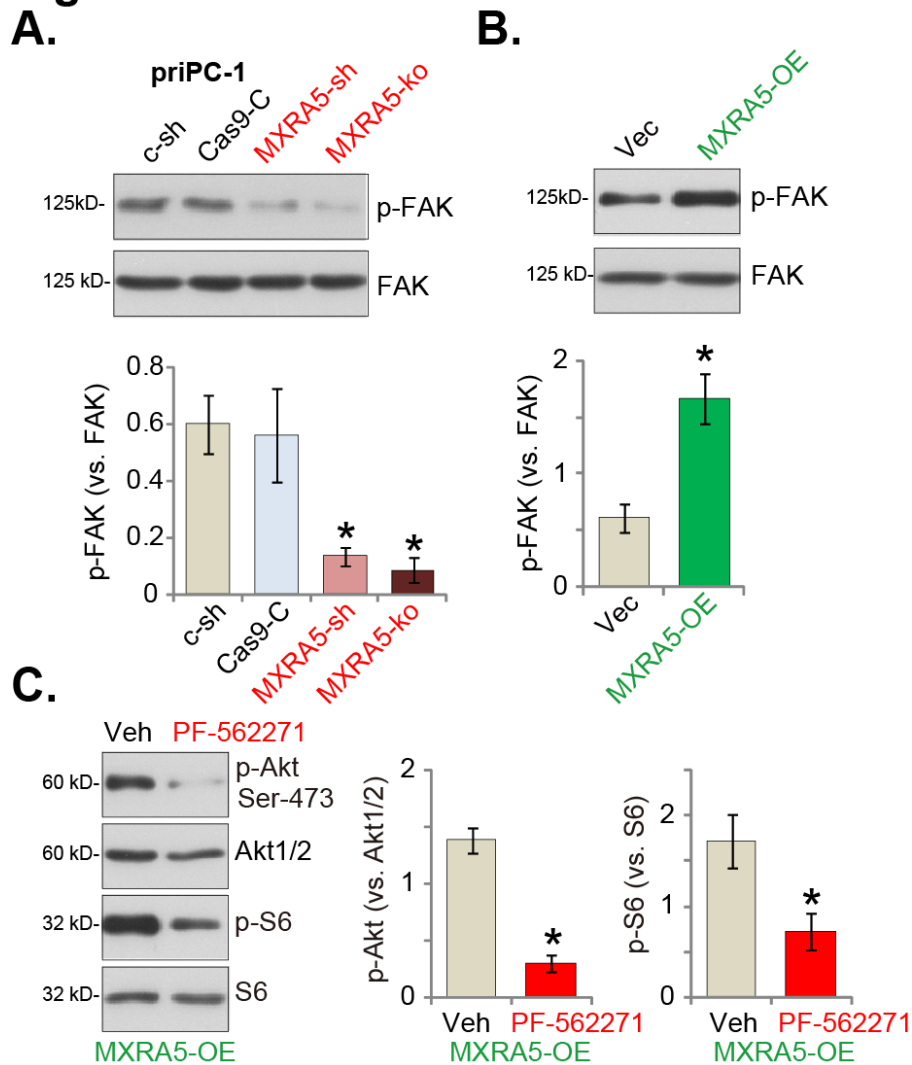


Figure 8



## Figure S5.



**Figure S5. FAK is important for MXRA5-promoted Akt-mTOR activation in pancreatic cancer cells.** Puromycin-selected priPC-1 cells with MXRA5-sh-S1 (“MXRA5-sh”), the scramble control shRNA (“c-sh”), the sgRNA-CRISPR/dCas-9 MXRA5-KO lentiviral construct (“MXRA5-ko”), Cas9 control construct (“Cas9-C”), the lentiviral MXRA5 overexpression construct (“MXRA5-OE”) or the corresponding vector (“Vec”) were cultured, and listed proteins tested (A and B). The OE-MXRA5 priPC-1 cells were treated with the FAK inhibitor PF-562271 (250 nM) or vehicle control (0.1% DMSO, “Veh”) for 6h, expression of listed proteins was shown (C). Error bars stand for mean  $\pm$  standard deviation (SD, n=5). \*  $P < 0.05$  versus “Vec”/“c-sh” cells or “Veh” treatment. Experiments in this figure were repeated five times.