

UCA1 promotes PDAC progression by regulating KRAS

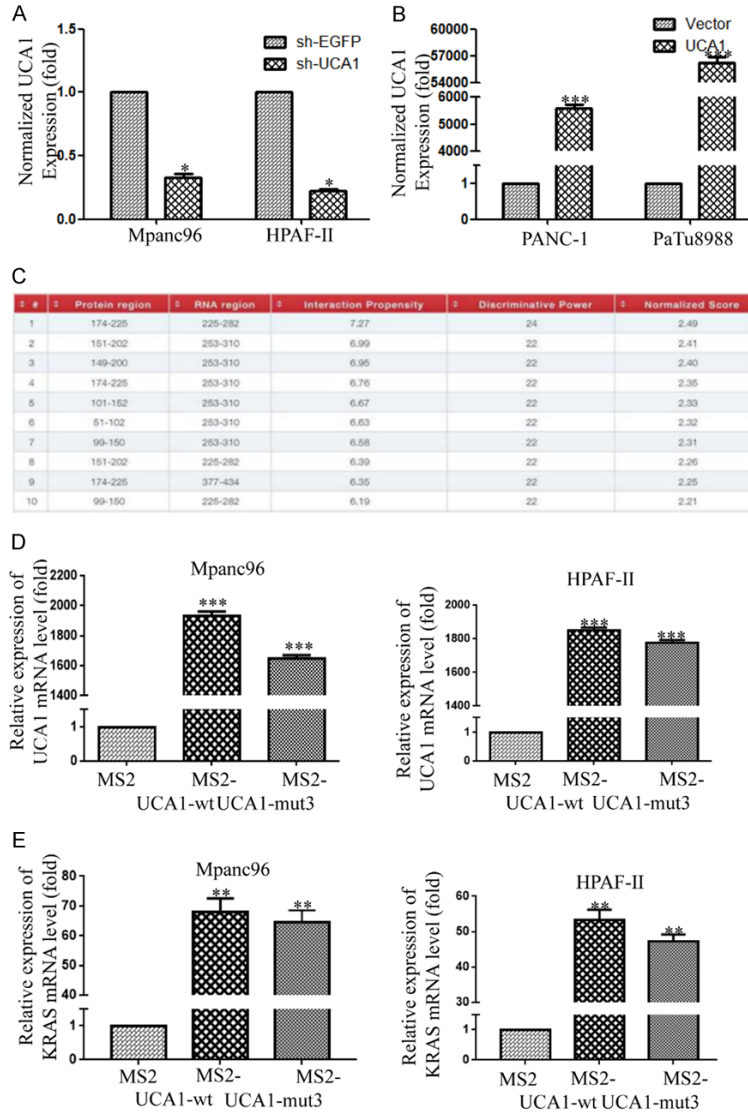


Figure S1. A, B. The UCA1 mRNA levels were reduced in Mpanc96 and HPAF-II cells transfected with sh-UCA1, whereas the UCA1 mRNA levels were increased after UCA1 overexpression in PaTu8988 and PANC-1 cells. C. Prediction of the RNA-protein interaction of UCA1 with hnRNPA2B1 using the catRAPID algorithm. D, E. The mRNA levels of UCA1 and KRAS were detected by qRT-PCR in Mpanc96 and HPAF-II cells after transfection with pSL-MS2-12X, UCA1-wt or UCA1-mut3 (Δ 271-282, mutated hnRNPA2B1 binding site in UCA1), respectively. (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$).

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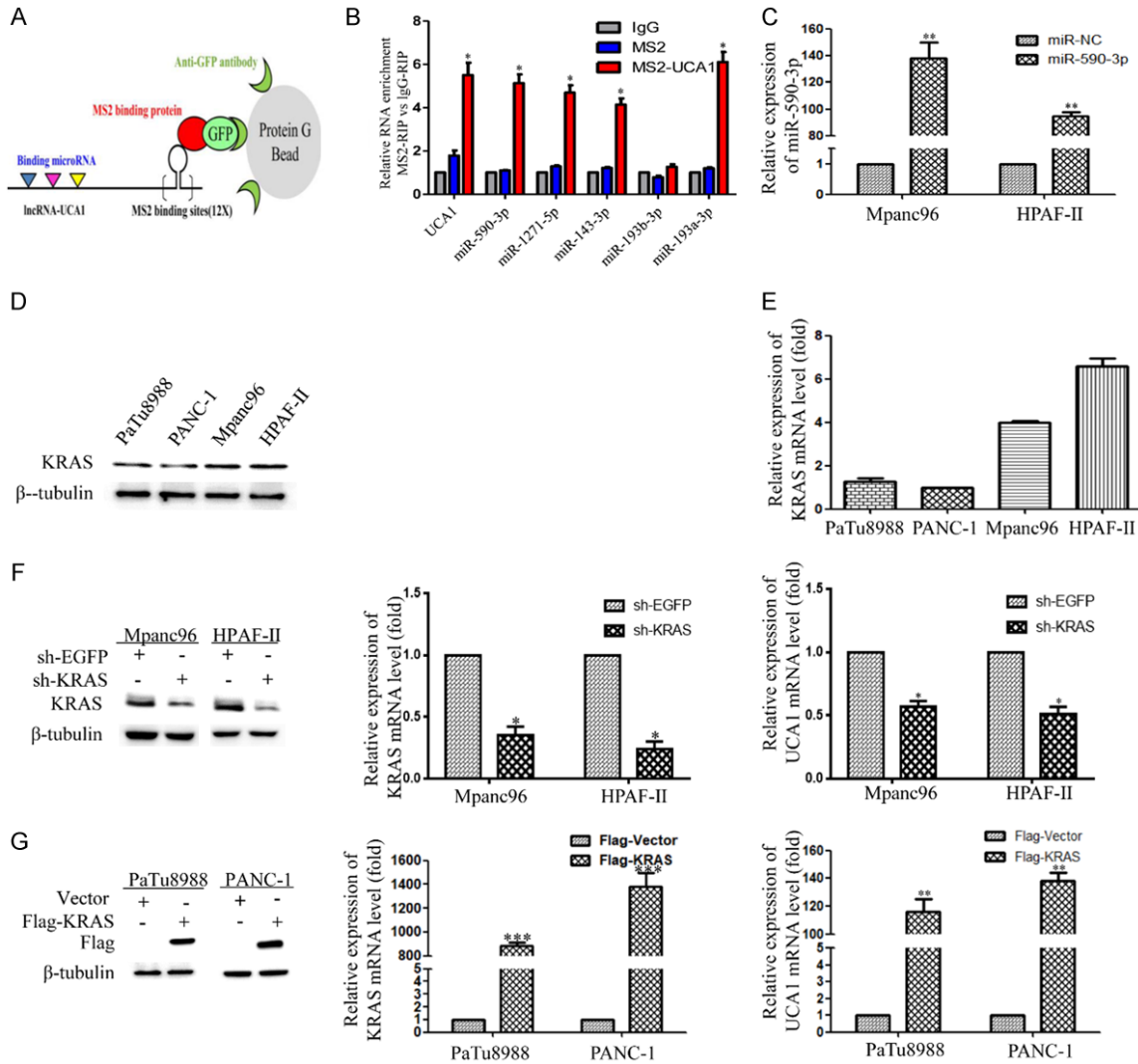


Figure S2. A. A schematic diagram of RNA immunoprecipitation (RIP) using MS2. B. Candidate miRNAs interacting with UCA1 were identified by the RIP assay. C. The expression of miR-590-3p in miR-590-3p-overexpressing cells was assessed by qRT-PCR. D, E. KRAS protein and mRNA expression levels were assessed in 4 PDAC cell lines by western blotting and qRT-PCR, respectively. F, G. The effect of KRAS on UCA1 expression was explored by qRT-PCR in Mpanc96/HPAF-II cells with KRAS knockdown and PaTu8988/Panc-1 cells overexpressing KRAS.

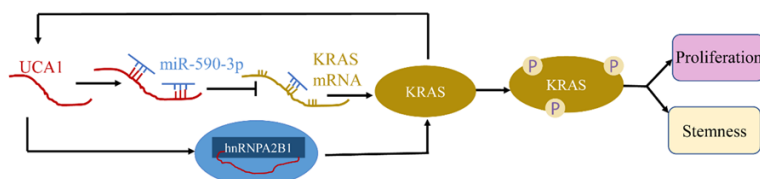


Figure S3. A schematic showing the results of this study. UCA1 is upregulated in PDAC. Moreover, UCA1 promotes the activity and expression of oncogenic KRAS, which, through interacting with hnRNP A2B1 and sponging miR-590-3p, is involved in the growth and progression of PDAC.