## **Supplementary Materials**

## Figure S1. RRM2 silencing attenuates the ability of IKBKE knockdown or overexpression to affect angiogenesis. (A–D) 786-O cells were infected with the indicated lentivirus for 72 h, and subsequently, the cell medium was collected for in vitro angiogenesis assays. Scale bar = 100 $\mu$ m. Statistical analysis was performed using one-way ANOVA for (B and D). ns, no significance; \*\*\*p < 0.001

## Figure S2. The IKBKE inhibitor CYT387 inhibits cell growth in RCC. (A) The

viability of 769-P and 786-O cells treated with CYT387 for 24 h, 48 h, and 72 h, respectively was measured through CCK-8 assays. (B) Proliferation rates of 769-P and 786-O cells in the control, DMSO, and CYT387 (5 µM) groups were compared through CCK-8 assays. (C-D) Colony numbers of 769-P and 786-O cells in the control, DMSO, and CYT387 (5  $\mu$ M) groups were compared through colony formation assays. The diameter of each well in panel C is approximately 35 mm. (E-F) Cell cycle analysis of 769-P and 786-O cells in the control, DMSO, and CYT387 (5 μM) groups was determined through flow cytometry. (G-I) 786-O cells were subcutaneously injected into nude mice for the xenograft assay (n = 6 mice/group). When the volume of xenografts reached 50 mm<sup>3</sup>, mice were randomly divided into two groups and treated with CYT387 (20 mg/kg, intraperitoneal injection with a volume of 200 µL for every 3 days) or constant volume solvents (DMSO), respectively. Xenografts in each group were photographed in panel G, the tumor mass was demonstrated in panel H, and the tumor growth curve was indicated in panel I. Statistical analysis was performed using one-way ANOVA for (D) and Student's t test for (H and I). \*\*\**p* < 0.001

Figure S1



## figure S2

