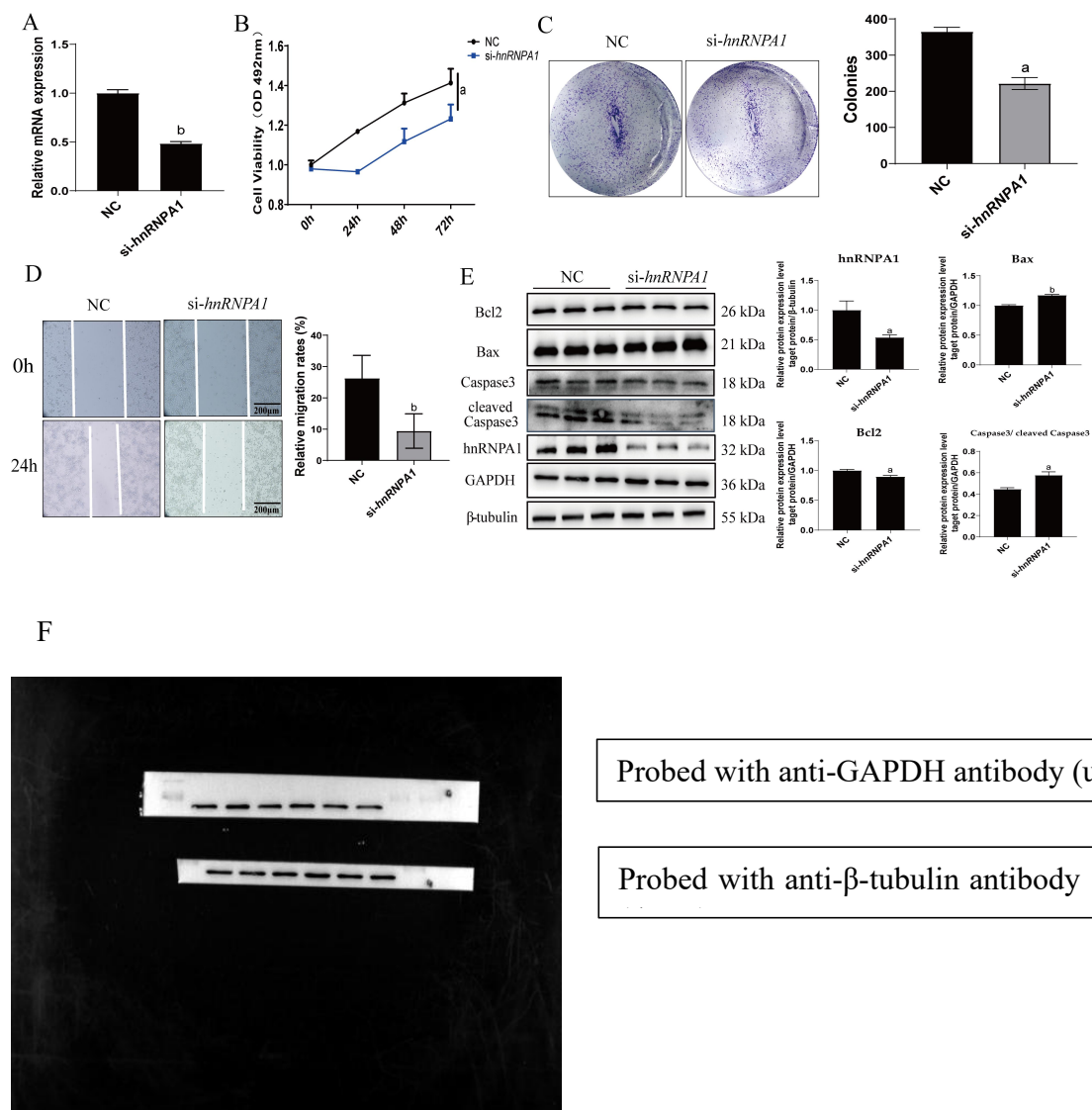
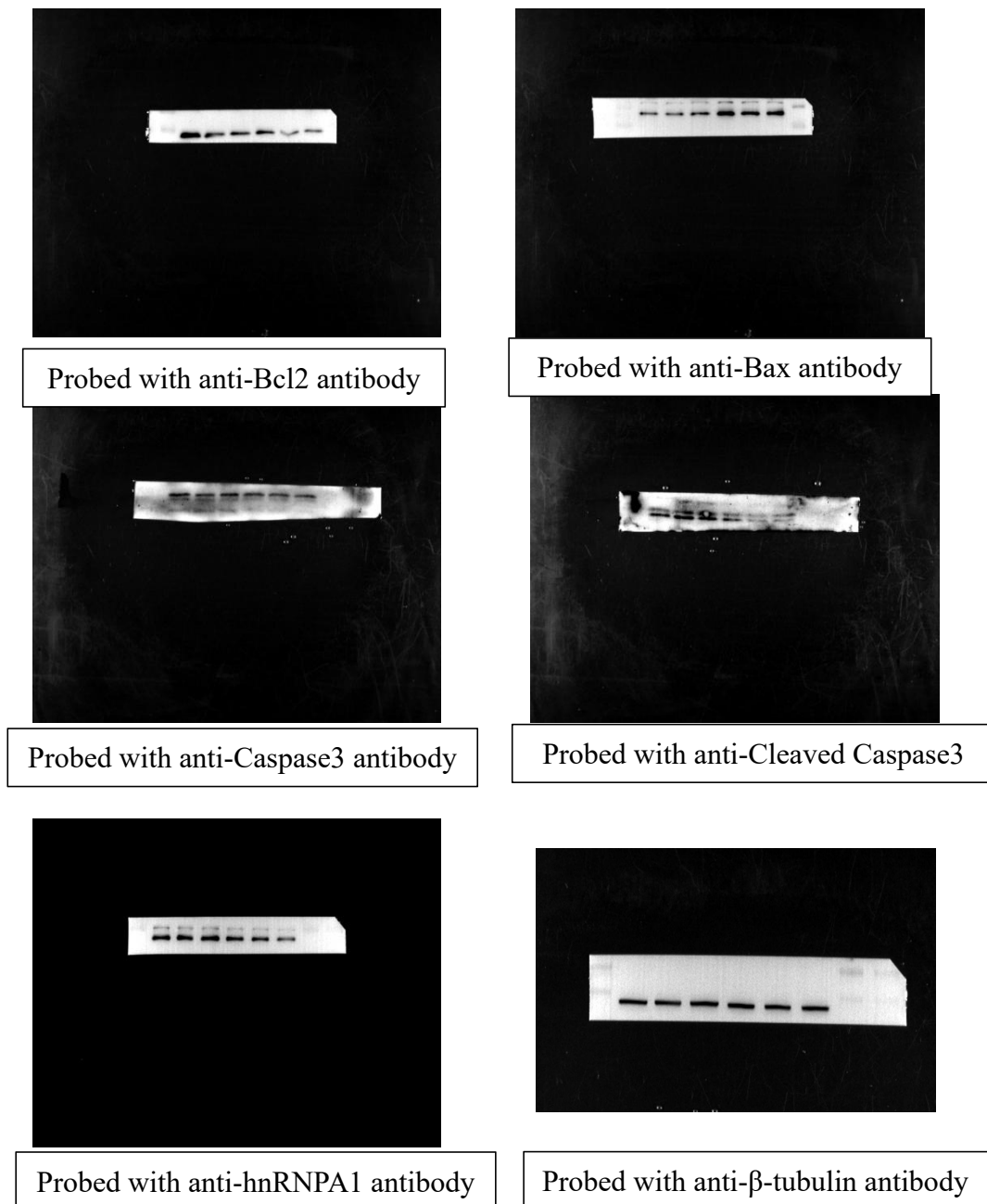


Supplementary Figure 1. The expression levels of *hnRNPA1* in HCC. The original blots of Figure 1D illustrate the following: the left membrane was stained with anti-β-actin antibody, the right membrane was probed with anti-hnRNPA1 antibody.

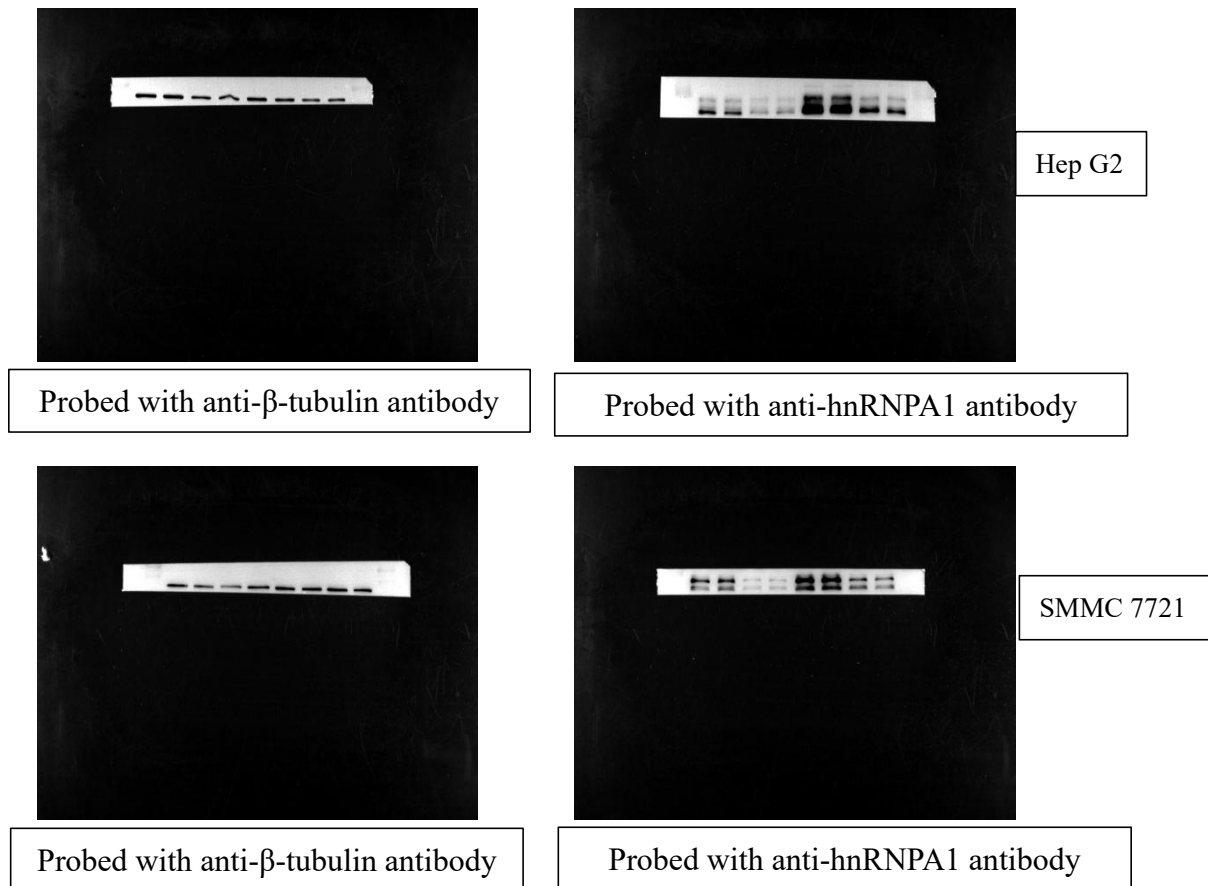




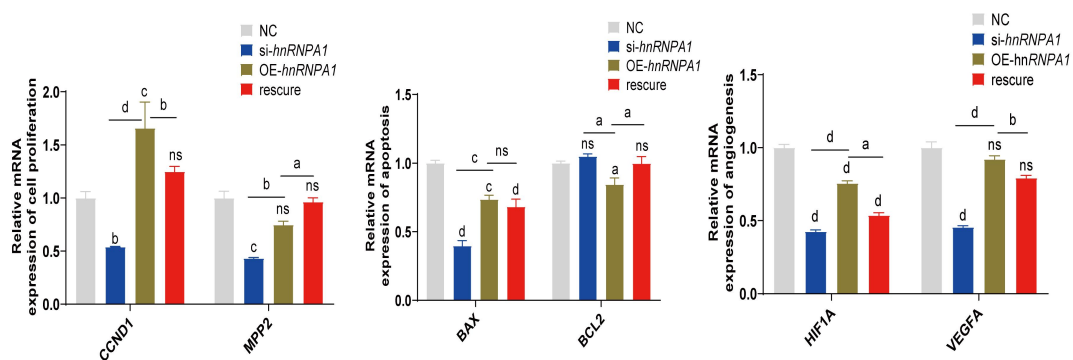
Supplementary Figure 2. Knockdown of *hnRNPA1* influences Hep G2 cell growth. (A) The expression of *hnRNPA1* in Hep G2. b: $p < 0.01$ (B) MTT assay showed that *hnRNPA1* knockdown inhibited Hep G2 proliferation. a: $p < 0.05$. (C) Cell clone formation assay showed that *hnRNPA1* knockdown inhibited Hep G2 proliferation. a: $p < 0.05$. (D) Wound healing assay showed that *hnRNPA1* knockdown inhibited Hep G2 migration. b: $p < 0.01$. Scale bar, 200 μm . (E) Western blot analysis showed that *hnRNPA1* knockdown slowed down the process of apoptosis. ns, no significance, a: $p < 0.05$, b: $p < 0.01$. (F) The original blots of Supplementary Figure S2E illustrate the following: the top membranes were stained with anti-GAPDH (top) and anti- β -tubulin (down) antibody, the second membranes were probed with anti-Bcl2 (left) and anti-Bax antibody (right), and the third membranes were probed with anti-Caspase3 (left) and anti-cleaved Caspase3 antibody (right), the low membranes were probed

with anti-hnRNPA1 (left) and anti- β -tubulin antibody (right) .

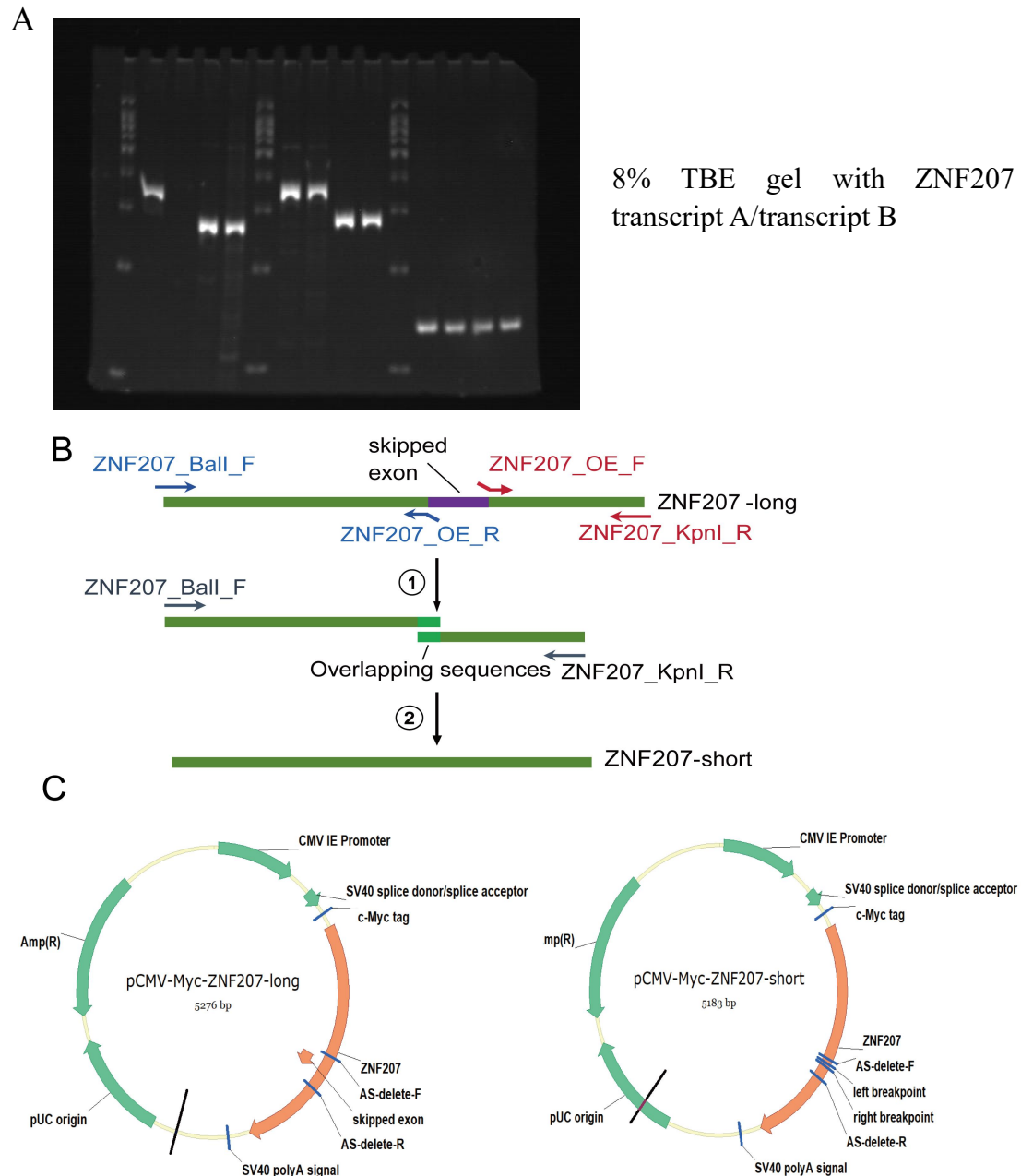
A



B

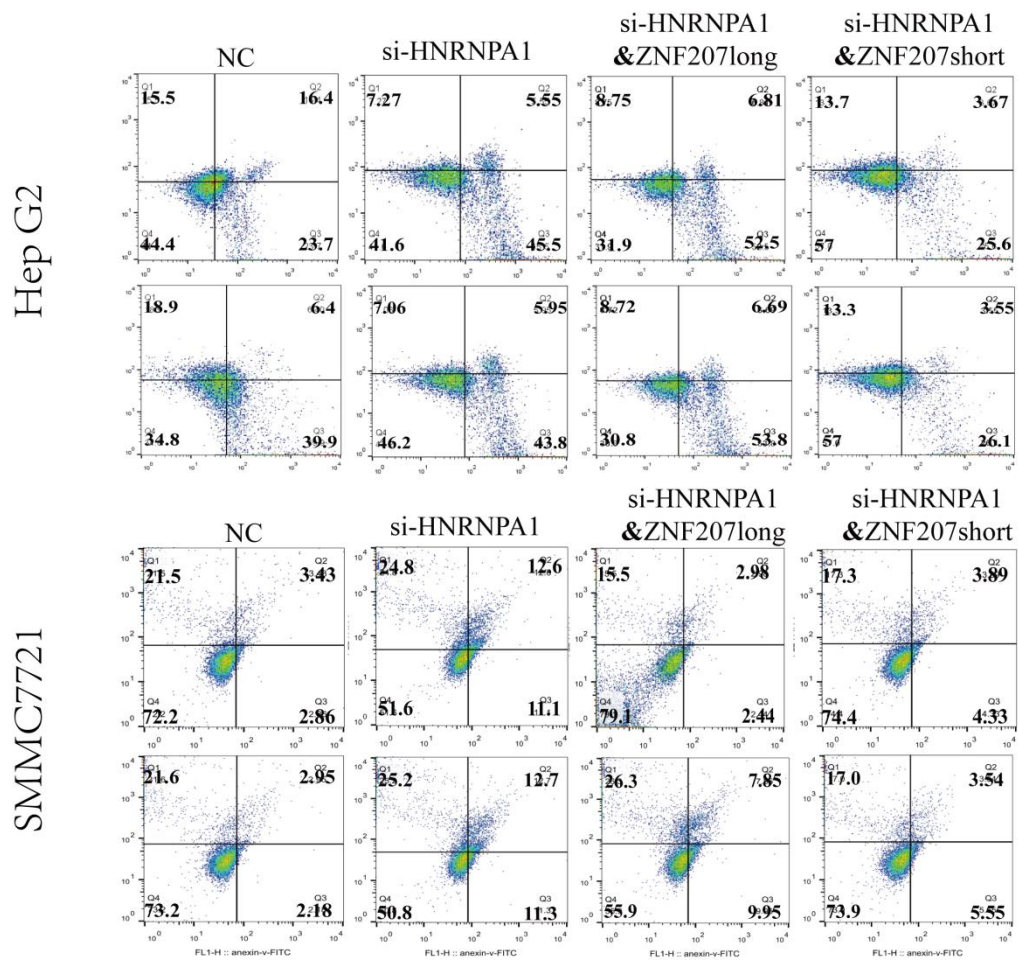


Supplementary Figure 3. Knockdown of *hnRNPA1* inhibits HCC cells proliferation and migration. (A) The original blots of Figure 3A illustrate the following: the top membrane was stained with anti- β -tubulin antibody (left) and anti-hnRNPA1 antibody (right) in Hep G2, the down membrane represents he top membrane was stained with anti- β -tubulin antibody (left) and anti-hnRNPA1 antibody (right) in SMMC7721. (B) Relative mRNA expression of genes involved in cell proliferation, cell apoptosis and vascular endothelial growth. *ns*, no significance, a: $p < 0.05$, b: $p < 0.01$, c: $p < 0.005$, d: $p < 0.001$.

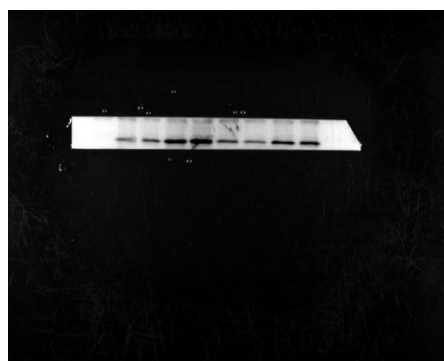


Supplementary Figure 4. ZNF207-short but not ZNF207-long promoted cell proliferation and migration. (A) The full gel electrophoresis shows ZNF207 transcription level in knockdown of *hnRNP1* in Hep G2 cell lines. A representative assay illustrated the change of the alternative splicing, as determined by the TBE analysis. The top gel was the β -actin PCR products (~139 bp) of control and knockdown *hnRNP1* in Hep G2. The down gel was the ZNF207 transcripts PCR products (~236bp) were digested into one fragment (~144bp), indicative of skipped exons. (B) Overlapping PCR flowchart. (C) Mutant plasmids for ZNF207-short and ZNF207-long.

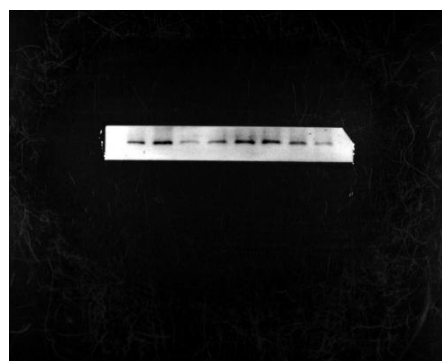
A



B

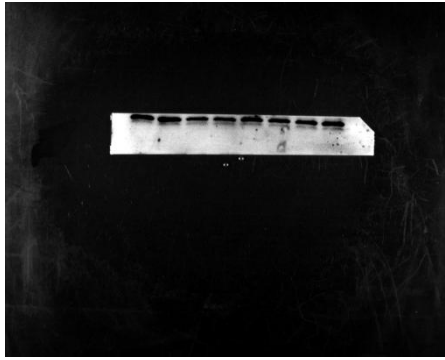


Probed with anti-Bax antibody

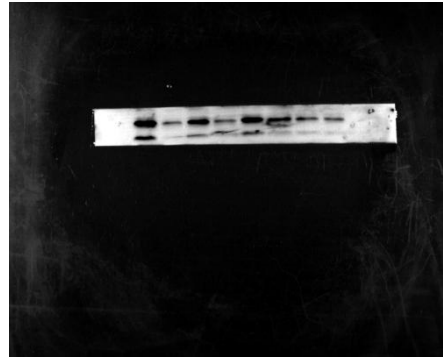


Probed with anti-Bcl2 antibody

Hep G2

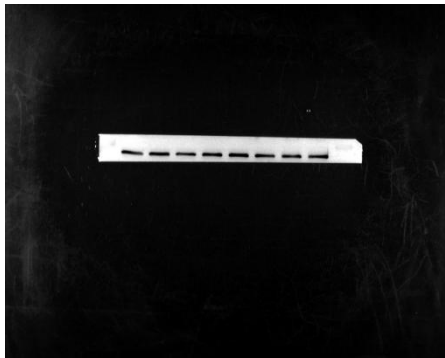


Probed with anti-Caspase3 antibody



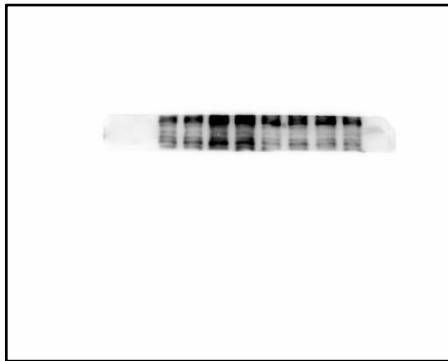
Probed with anti-Cleaved Caspase3 antibody

Hep G2

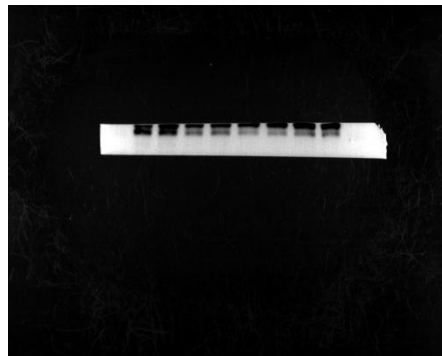


Hep G2

Probed with anti-GAPDH antibody

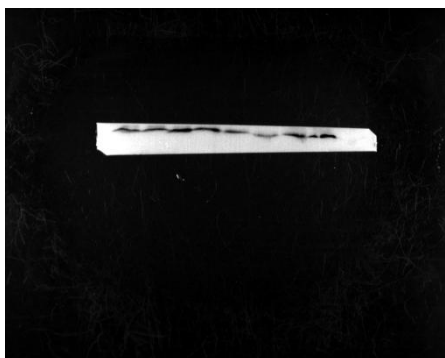


Probed with anti-Bax antibody

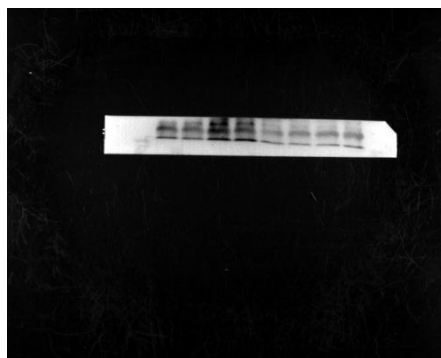


SMMC 7721

Probed with anti-Bcl2 antibody

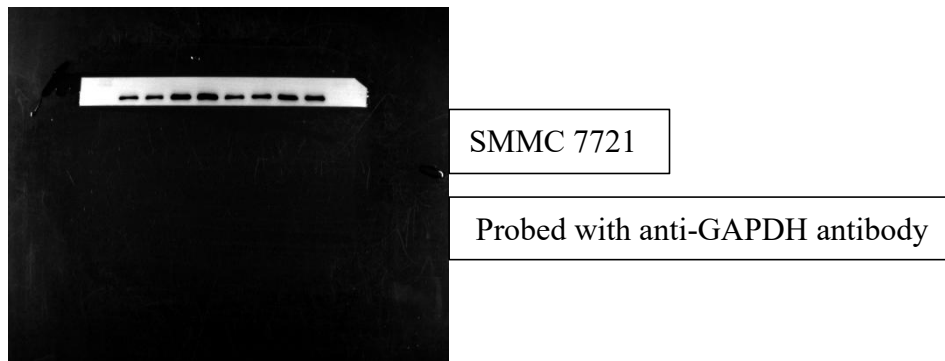


Probed with anti-Caspase3 antibody

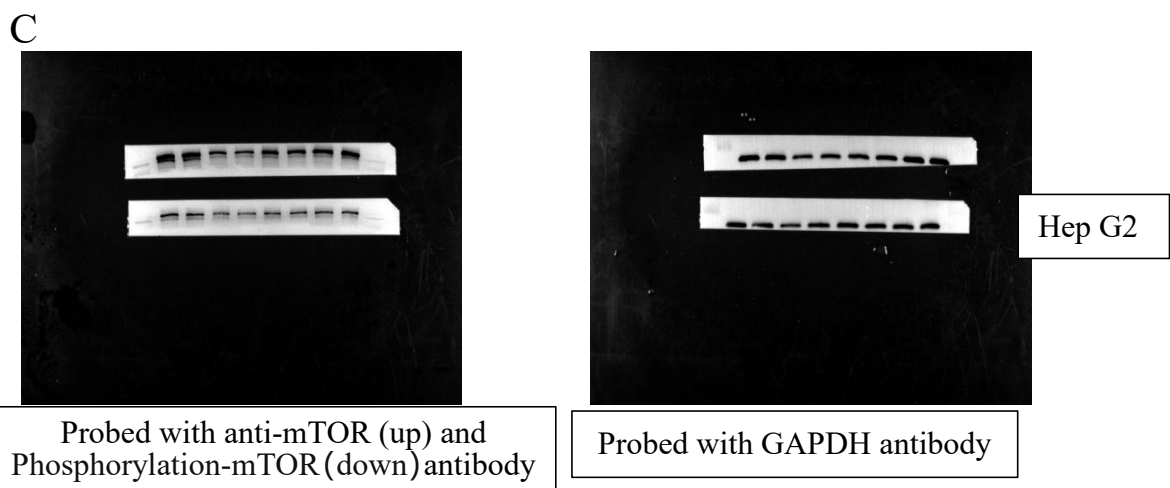
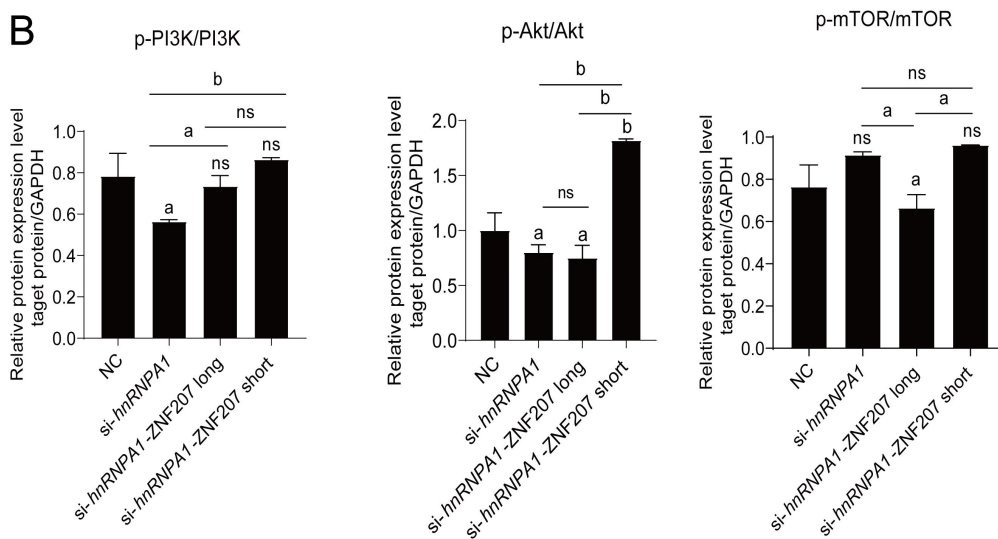
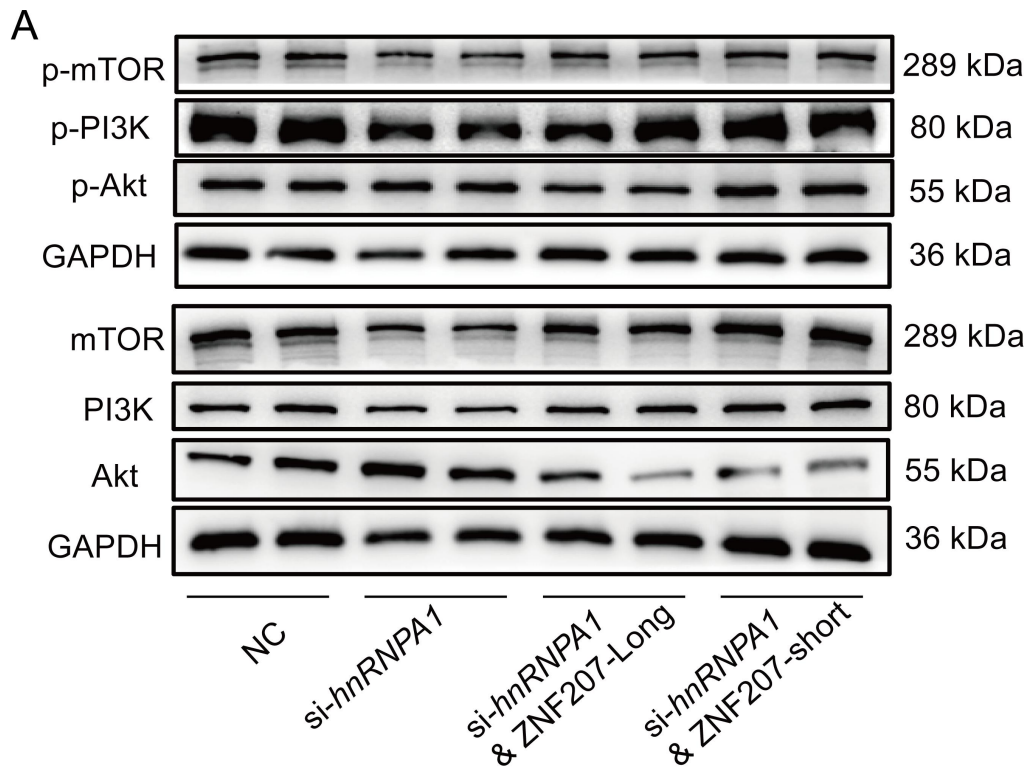


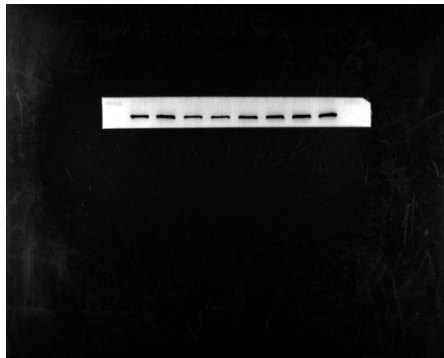
SMMC 7721

Probed with anti-Cleaved Caspase3 antibody

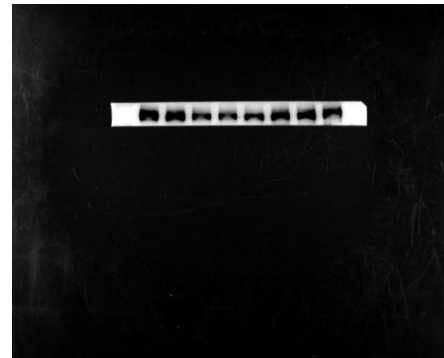


Supplementary Figure 5. ZNF207-short but not ZNF207-long promotes proliferation and migration. (A) Flow cytometry experiments showed that ZNF207-short inhibited apoptosis in HCC cells. (B) The original blots of Figure 5E illustrate the following: the top membrane was stained with anti-Bax antibody (left) and anti-Bcl2 antibody (right) in Hep G2, the middle membrane was probed with anti-Caspase3 antibody (left) and anti-Cleaved Caspase3 antibody (right) in Hep G2. The lower membrane was probed with anti-GAPDH antibody in Hep G2. The rest membranes in SMMC 7721 were probed with antibody in equal order.



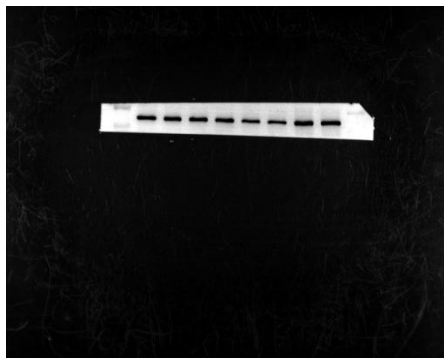


Probed with anti-PI3K antibody

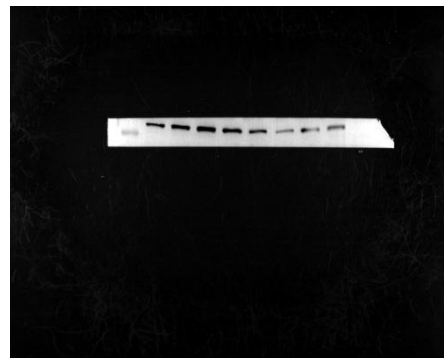


Probed with anti-Phosphorylation-PI3K antibody

Hep G2



Probed with anti-Akt antibody



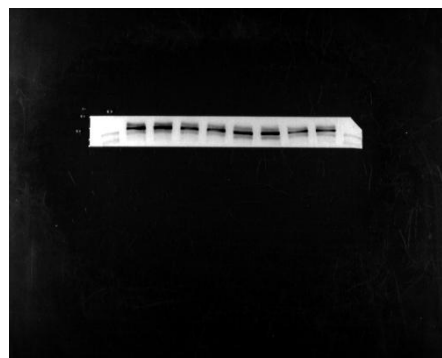
Probed with anti-Phosphorylation-Akt antibody

Hep G2

D

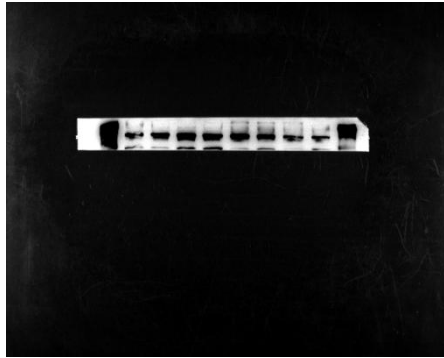


Probed with anti-mTOR antibody

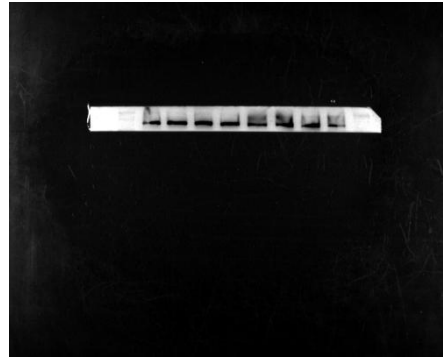


Probed with Phosphorylation-mTOR antibody

Hep

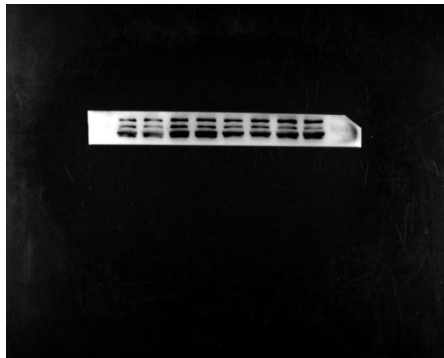


Probed with anti-PI3K antibody

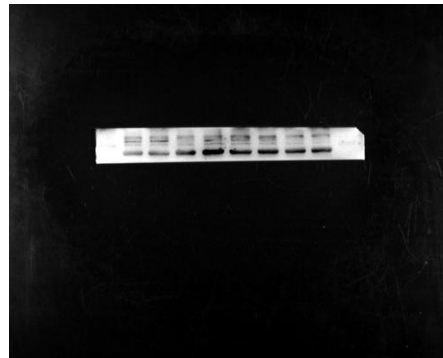


Hep G2

Probed with Phosphorylation-PI3K antibody

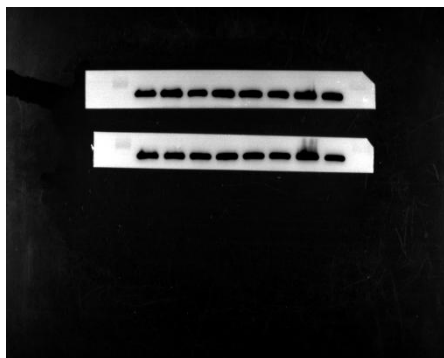


Probed with anti-Akt antibody



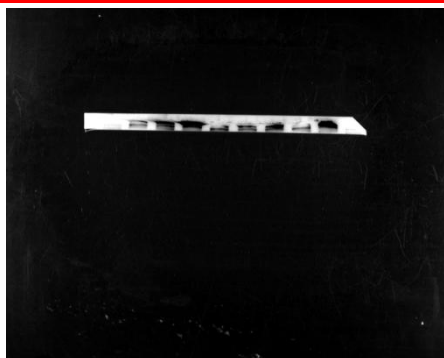
Hep G2

Probed with Phosphorylation-Akt antibody

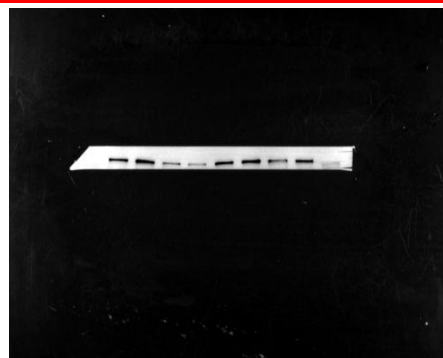


Hep G2

Probed with GAPDH antibody

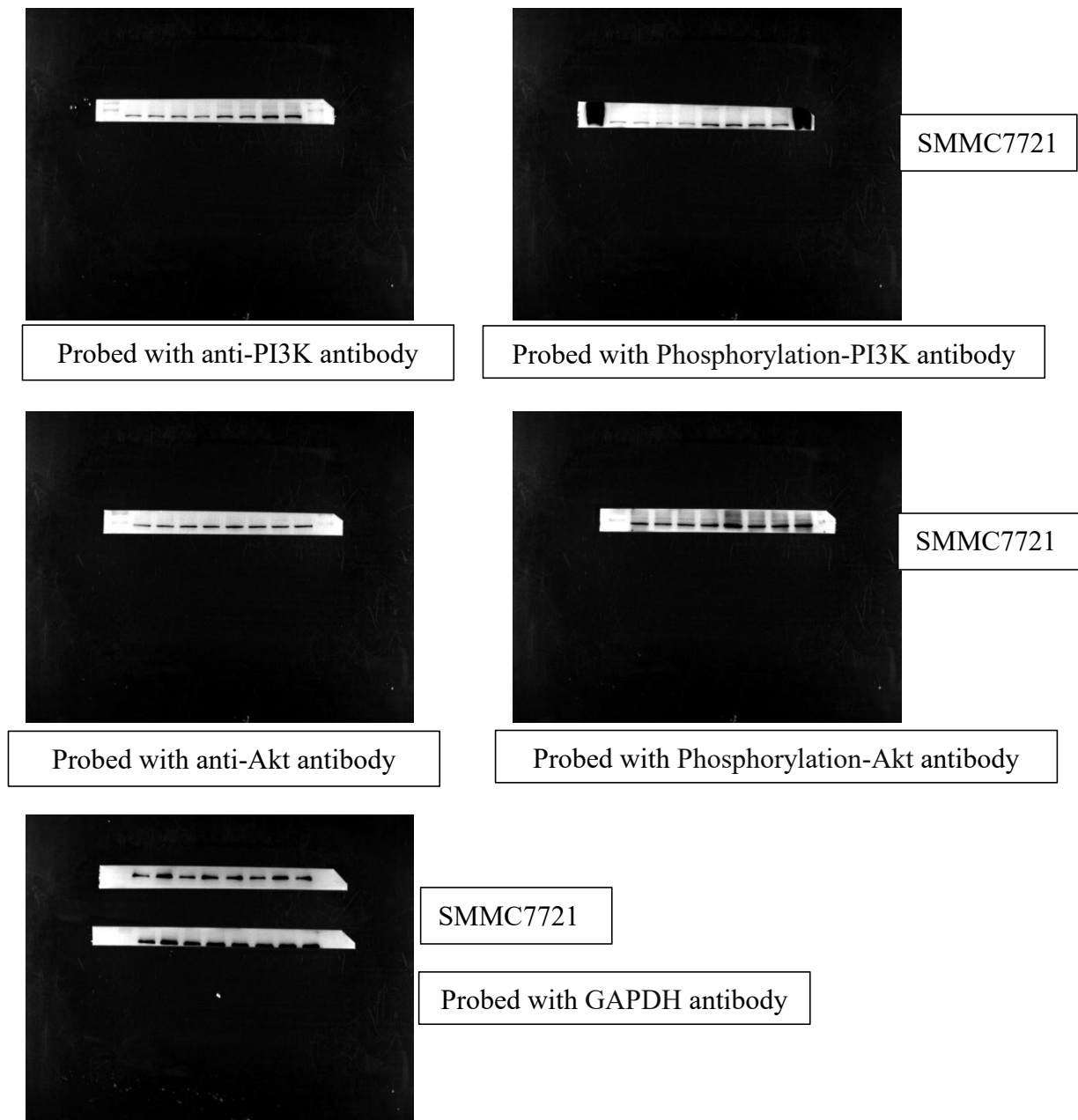


Probed with anti-mTOR antibody



SMMC7721

Probed with Phosphorylation-mTOR antibody



Supplementary Figure 6. ZNF207 affects the PI3K/Akt signaling pathway in Hep G2. (A) Western blot analysis of PI3K, Akt and mTOR in Hep G2. (B) The densitometric analysis for PI3K, Akt and mTOR in Hep G2. *ns*, no significance, a: $p < 0.05$, b: $p < 0.01$. (C) The original blots of Supplementary Figure S6A illustrate the following: the top membrane was stained with anti-mTOR & p-mTOR antibody (left) and anti-GAPDH antibody (right) in Hep G2, the middle membrane was probed with anti-PI3K antibody (left) and p-PI3K antibody (right) in Hep G2. The lower membrane was probed with anti-Akt antibody (left) and p-Akt antibody (right) in Hep G2. (D) The original blots of Figure 6A illustrate the following: the top membrane was stained with anti-mTOR & p-mTOR antibody (left) and anti-GAPDH antibody (right) in Hep G2, the middle membrane was probed with anti-PI3K antibody (left) and p-PI3K antibody (right) in Hep G2. The lower membrane was probed with anti-Akt antibody (left) and p-Akt antibody (right) in Hep G2. The rest membranes in

SMMC 7721 were probed with antibody in equal order.