

Supplementary Materials:

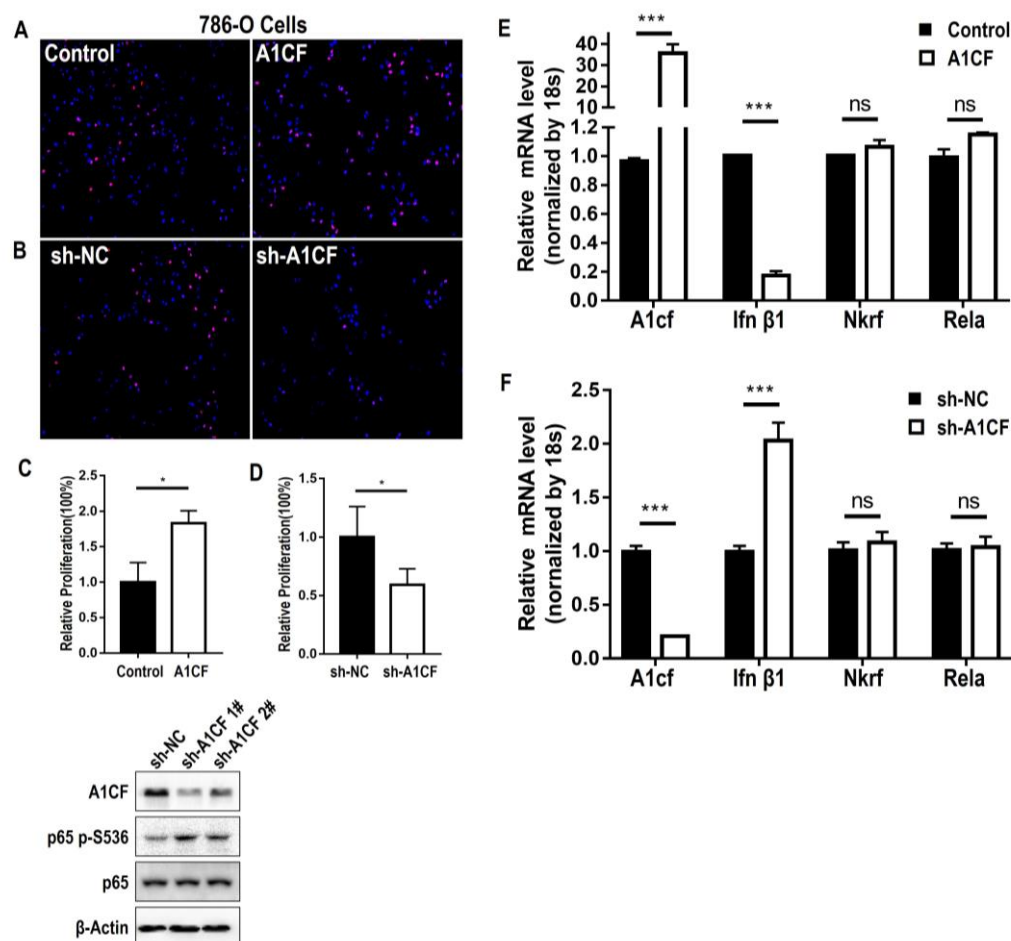


Figure S1. A1CF controlled cell growth through negative regulation of IFN- β via NF- κ B signaling activation A-D Growth of 786-O stable cells were measured by EdU assay. Results were expressed as mean \pm SEM from 3 replicate well. p-values were calculated by Student t-test, * $p < 0.05$. (A). Merged image of EdU staining (red) and Hoechst staining (blue) for 786-O A1CF overexpression stable cells. (B). Merged image of EdU staining (red) and Hoechst staining (blue) for 786-O A1CF deficiency stable cells. (C, D). Statistical analysis of 786-O cell proliferation. Values were presented as mean \pm SEM ($n = 3$). p-values were calculated by Student t-test, * $p < 0.05$. (E). 786-O cells expressing sh-NC, sh-A1CF1#, sh-A1CF2# were examined by western blotting with the indicated antibodies. (F, G). mRNA levels of indicated genes in 786-O stable cells were measured by RT-qPCR. The data are presented as means \pm SD from triplicate samples. *** $p < 0.001$.

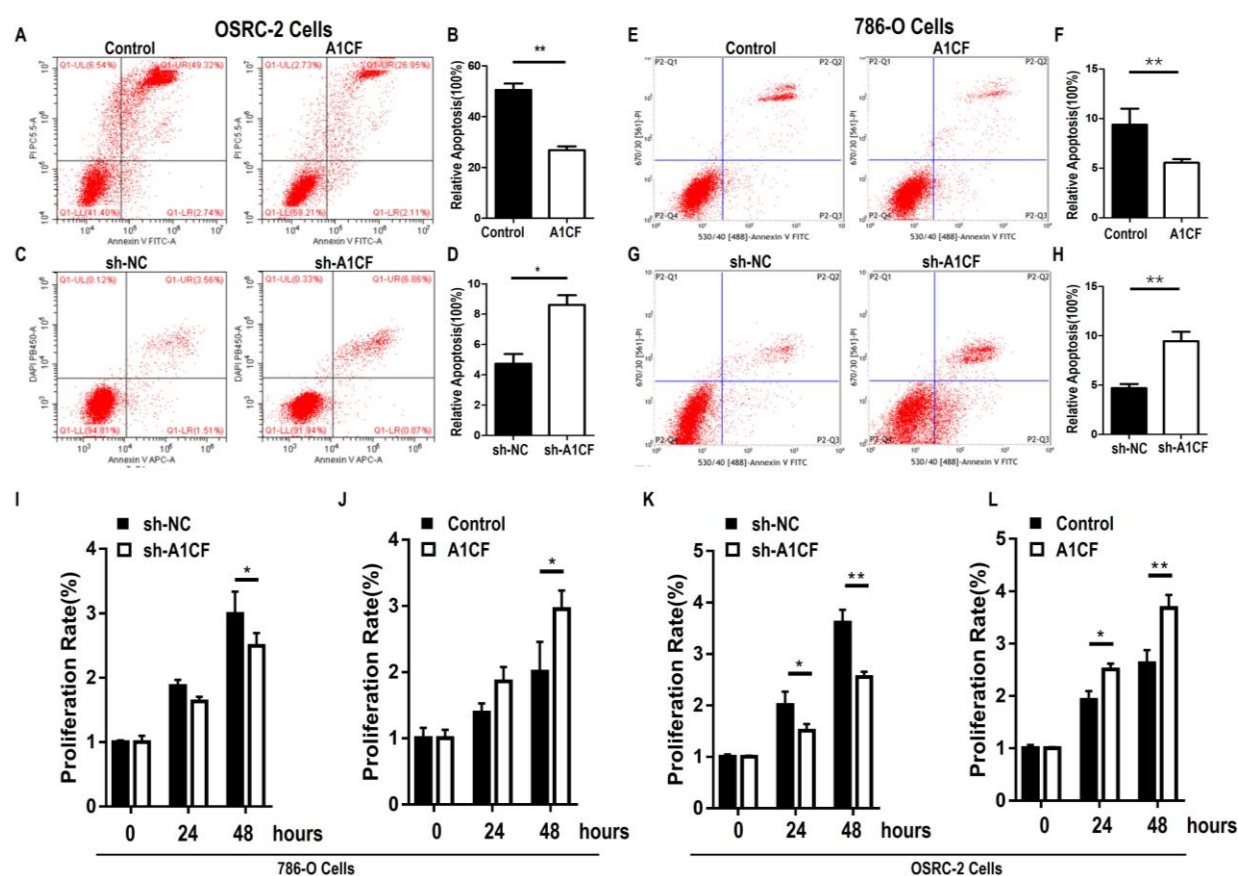


Figure S2. A1CF promoted proliferation and inhibited apoptosis in stable renal cells. **(A–H)** Apoptosis of stable renal cancer cells were measured by flow cytometry with Annexin V-PI staining. Results were expressed as mean \pm SEM from 3 replicates. p-values were calculated by Student t-test, * $p < 0.05$, ** $p < 0.01$. **(A)**. Flow cytometry analysis was performed to evaluate the percentage of apoptotic cells in OSRC-2 A1CF overexpression stable cells. **(C)**. Flow cytometry analysis was performed to evaluate the percentage of apoptotic cells in OSRC-2 A1CF deficiency stable cells. **(B, D)**. Statistical analysis of cell apoptosis in OSRC-2 stable cell lines and histogram was drawn in GraphPad Prism 5. Values were presented as mean \pm SEM ($n = 3$). p-values were calculated by Student t-test, * $p < 0.05$. **(E)**. Flow cytometry analysis was performed to evaluate the percentage of apoptotic cells in 786-O A1CF overexpression stable cells. **(G)**. Flow cytometry analysis was performed to evaluate the percentage of apoptotic cells in 786-O A1CF knockdown stable cells. **(F, H)**. Statistical analysis of cell apoptosis in 786-O stable cell lines and histogram was drawn in GraphPad Prism 5. Values were presented as mean \pm SEM ($n = 3$). p-values were calculated by Student t-test, ** $p < 0.01$. **(I–L)**. Proliferation of stable renal cancer cells were measured by MTT assay. Results were expressed as mean \pm SEM from 6 replicate well. p-values were calculated by Student t-test, * $p < 0.05$; ** $p < 0.01$. **(I, J)**. 786-O stable cell lines indicated above were seeded on 96-well plates and cell viability was measured by MTT assays at 0, 24, 48 hours. **(K, L)**. OSRC-2 stable cell lines indicated above were seeded on 96-well plates and cell viability was measured by MTT assays at 0, 24, 48 hours.

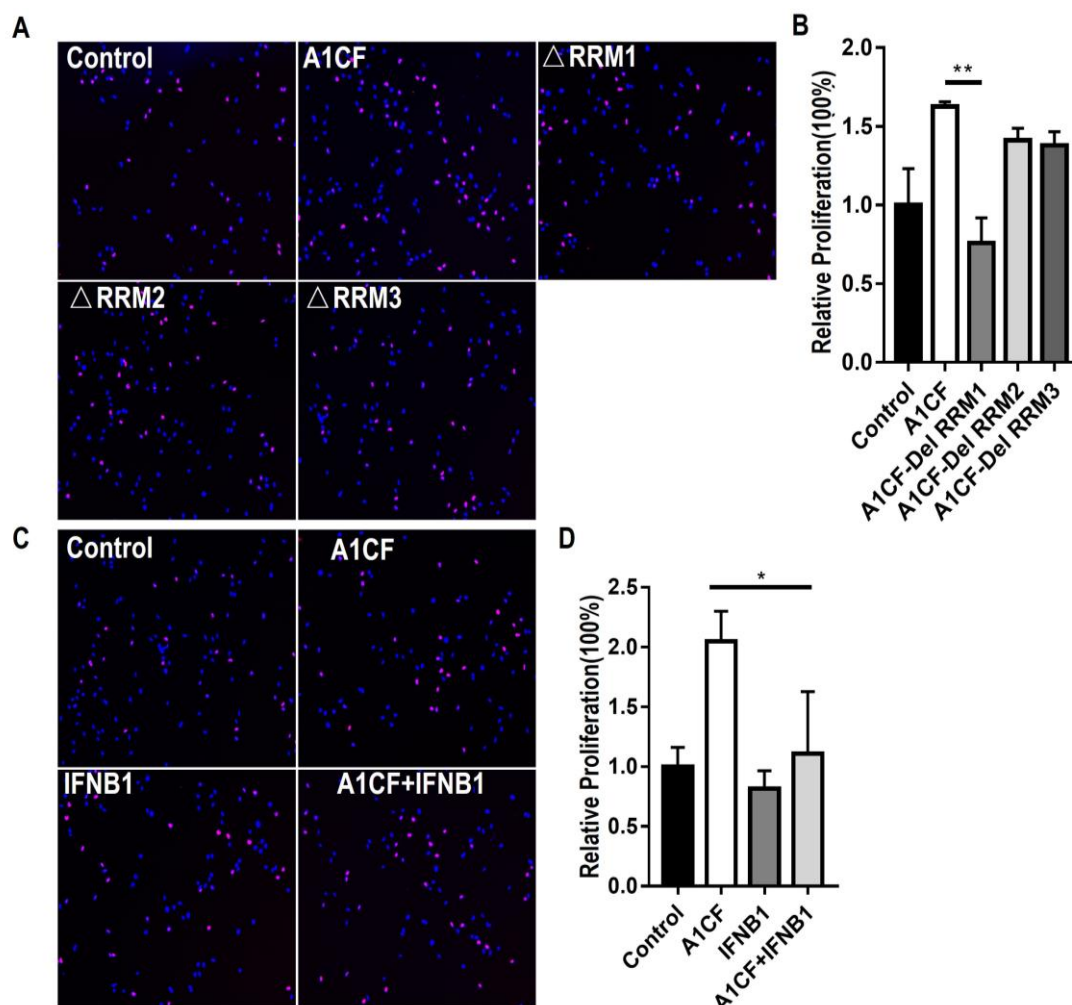


Figure S3. Disrupting the A1CF-NKRF interaction changed the renal cancer cell proliferation measured by EdU assay through A1CF-NKRF- NF- κ B- IFN- β axis. (A-D). Growth of 786-O cells expressing the indicated plasmids were measured by EdU assay. Results were expressed as mean \pm SEM from 3 replicate well. p-values were calculated by Student t-test, *p < 0.05. (A). Merged image of EdU staining (red) and Hoechst staining (blue) for 786-O cells expressing indicated A1CF Δ RRM mutants. (C). Merged image of EdU staining (red) and Hoechst staining (blue) for 786-O cells expressing indicated plasmids. (B, D). Statistical analysis of 786-O cell proliferation. Values were presented as mean \pm SEM (n = 3). p-values were calculated by Student t-test, * p < 0.05.

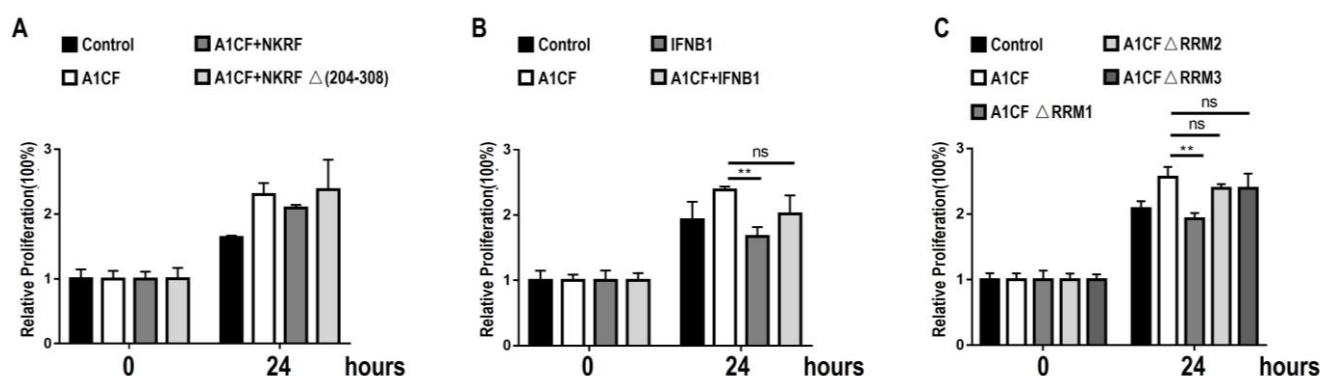


Figure S4. Disrupting the A1CF-NKRF interaction changed the renal cancer cell proliferation measured by MTT assay through A1CF-NKRF- NF-κB- IFN-β axis. (A-C). Proliferation of 786-O cells expressing the indicated plasmids were measured by MTT assay. Cells indicated above were seeded on 96-well plates and cell viability was measured at 0, 24 hours. Results were expressed as mean ± SEM from 6 replicate well. p-values were calculated by Student t-test, ns=no significance; **p < 0.01.

Table S1: Primers used for plasmid constructs in this paper

Primer Name	Sequence
F-h.A1CF CDS	5'-GGATCCGAATTCATGGAATCAAATCACAAATCCG-3'
R-h.A1CF CDS	5'-ATGGTGGTGCTCGAGTCAGAAGGTGCCATATCCATC-3'
F-h.A1CF-truncation(1-293aa)	5'-GGATCCGAATTCATGGAATCAAATCACAAATCCGGGGA-3'
R-h.A1CF-truncation(1-293aa)	5'-ATGGTGATGGTGGTGTATTATAACCATCCAGCACCTTGCCAT-3'
F-h.A1CF-truncation(294-586aa)	5'-GGATCCGAATTCCTCCCCATTGAAGTCACCCTAGCAAA-3'
R-h.A1CF-truncation(294-586aa)	5'-ATGGTGATGGTGGTGTGTCAGAAGGTGCCATATCCATCCCCTC-3'
R-H.A1CF(1-130aa)	5'-ATGGTGATGGTGGTGTATTATAACAAACCCCTAAGAGGCGCCCATTT-3'
R-H.A1CF(131-220aa)	5'-GGATCCGAATTCGCCAGTGTGGACAACCTGCCGATTAT-3'
R-H.A1CF(131-220aa)	5'-ATGGTGATGGTGGTGTATTAACTTCTGGCTCTGCCAGTCTCCTG-3'
F-h.A1CF(221-293aa)	5'-GGATCCGAATTCGAAGTTGATGAAGATACAATGTCT-3'
F-h.NKRF CDS	5'-GGATCCGAATTCATGGGCTTTATGTTACCTCTCATCT-3'
R-h.NKRF CDS	5'-ATGGTGATGGTGGTGTTC AATTTGCTTGAGGCATAACAAGC-3'
R-h.NKRF(1-204aa overlap)	5'-ACACACCACGAGGTCTTCTGGATTAGAAAGGTTCTTCCAG-3'
F-h.NKRF(308aa-end)	5'-GACCTCGTGGTGTGTCAGATTGGCA-3'
F-h.IFNβ1 CDS	5'-GGATCCGAATTCAGGCGACACTGTTTCGTGTTGTCAAC-3'
R-h.IFNβ1 CDS	5'-ATGGTGATGGTGGTGGCCAGAGGCACAGGCTAGGAGATCT-3'
F-A1CF shRNA#1	5'-CCGGCCATGCTGCAAGGAGAGTATACTCGAGTATACTCTCCTTGCAGCATGGTTTTG-3'
R-A1CF shRNA#1	5'-AATTCAAAAACCATGCTGCAAGGAGAGTATACTCGAGATACTCTCCTTG CAGCATGG-3'
F-A1CF shRNA#2	5'-CCGGGCTGCTGCTGCTACTGCTTTC CTCGAG GAAAGCAGTAGCAGCAGCAGCTTTTTG-3'
R-A1CF shRNA#2	5'-AATTCAAAAAGCTGCTGCTGCTACTGCTTTC CTCGAG GAAAGCAGTAGCAGCAGCAGC-3'

Table S2: Primers used for qPCR in this paper

Primer Name	Sequence
A1CF-s	5'-TGTCATCGTCTACCCAAGCG-3'
A1CF-as	5'-GCTCTGCCCAGTCTACTGC-3'
IFNB1-s	5'-TCTTTCCATGAGCTACAACCTTGCT-3'
IFNB1-as	5'-GCAGTATTCAAGCCTCCCATTTC-3'
RELA-s	5'-TTTCTCCTCAATCCGGTGAC-3'
RELA-as	5'-ACCCCTCCCTACGCAGAC-3'
NKRF-s	5'-CCAAACCTTCCAAAGGTCAA-3'
NKRF-as	5'-CAGGGTTCCCACTGTCAAAA-3'
18s-s	5'-GTAACCCGTTGAACCCCATTC-3'
18s-as	5'-CCATCCAA TCGGTAGTAGCG-3'