Table S1. The primers for RT-qPCR assay

Genes	Forward	Reverse
AKR1B10P1	5'-GGTAATATGATCGGTGGAAAAGCA-3'	5'-TAGGGGGCTGTAGGCCATAAT-3'
SOX4	5'-GCACTAGGACGTCTGCCTTT-3'	5'-ACACGGCATATTGCACAGGA-3'
miR-138	5'-AACGGAGCTGGTGTTGTGAATC-3'	5'-GTGCAGGGTCCGAGGT-3'
ChIP assay Primer-1	5'-TGGGATGCAGCTAAAGCAGT-3'	5'-CTGCTGTGTCCAGAGTTGGT-3'
ChIP assay Primer-2	5'-GAGGCAGGAGCAAACAAAGC-3'	5'-TGGTGCAGACCTAGGAGTCA-3'

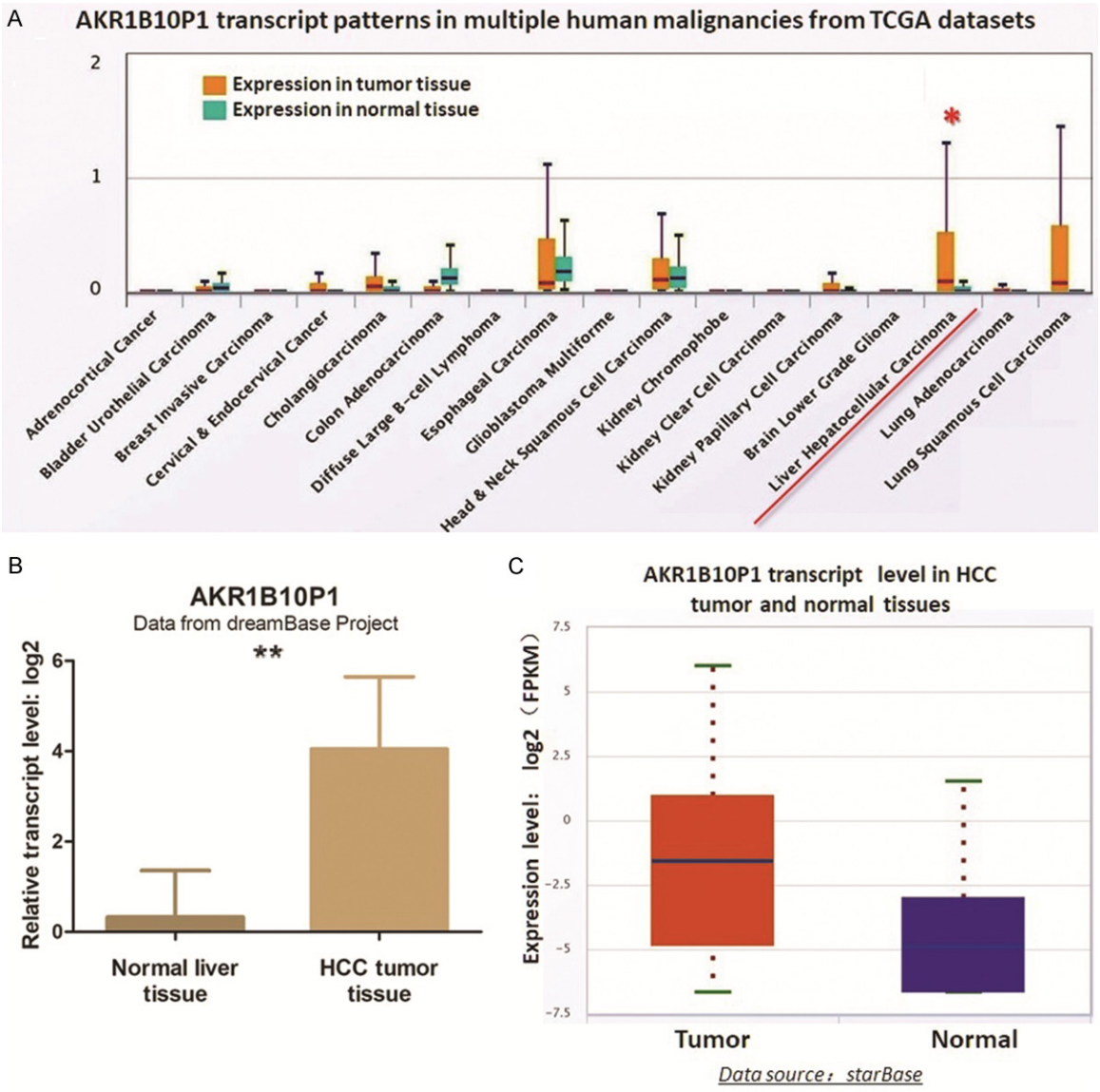
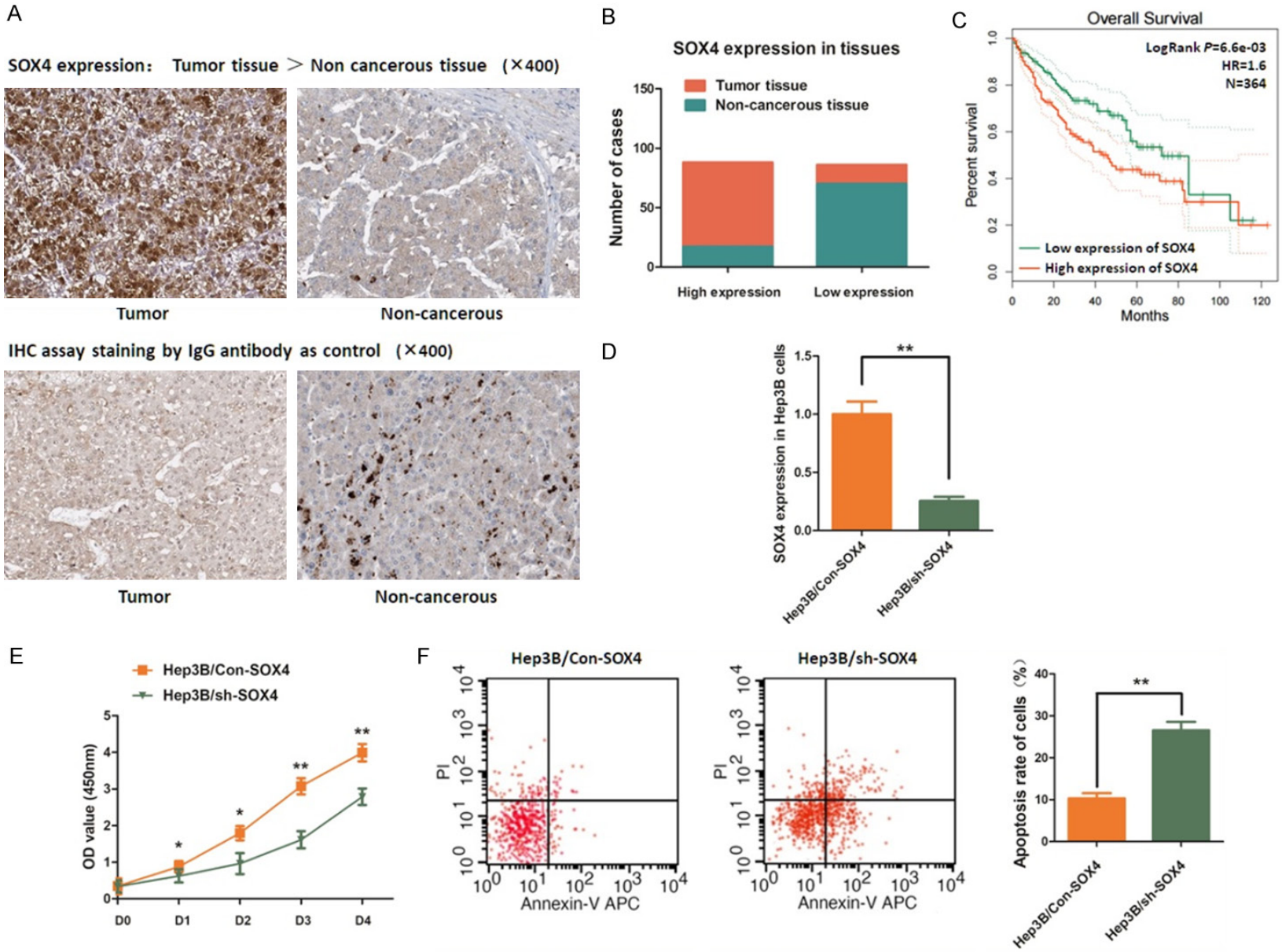


Figure S1. AKR1B10P1 expression profile from the databases. A. According to the analysis of TCGA database, pseudogene AKR1B10P1 presents detectable transcript products in normal liver tissues and cells in multiple human malignancies (*P < 0.05). B. According to the datasets from dreamBase Project database, AKR1B10P presents extremely low transcription in normal liver tissues, but was transcribed in HCC tumor tissues (**P < 0.01). C. According to the starBase database, AKR1B10P1 was transcribed in HCC tissues (P=1.0e-7).


A
SOX4 expression: Tumor tissue > Non cancerous tissue (×400)
Tumor
Non-cancerous
IHC assay staining by IgG antibody as control (×400)
Tumor
Non-cancerous
B
SOX4 expression in tissues
Tumor tissue
Non-cancerous tissue
Number of cases
High expression
Low expression
C
Overall Survival
LogRank P=6.6e-03
HR=1.6
N=364
Percent survival
Low expression of SOX4
High expression of SOX4
Months
D
SOX4 expression in Hep3B cells
Hep3B/Con-SOX4
Hep3B/sh-SOX4
E
Hep3B/Con-SOX4
Hep3B/sh-SOX4
OD value (450nm)
D0
D1
D2
D3
D4
F
Hep3B/Con-SOX4
Hep3B/sh-SOX4
PI
Annexin-V APC
Apoptosis rate of cells (%)
Hep3B/Con-SOX4
Hep3B/sh-SOX4

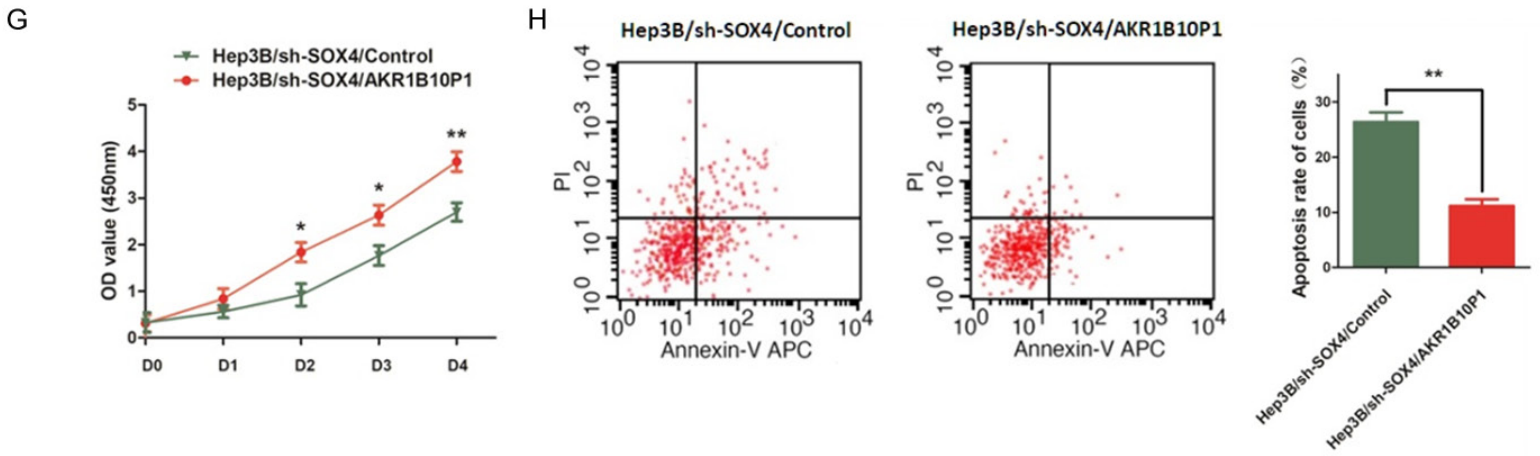


Figure S2. SOX4 expression profile and the impact on cell proliferation in HCC. A. Representative graph of immunohistochemistry analysis (400Î) of the HCC cases. Specimens stained IgG anti-body were regarded as control. SOX4 expression in tumor specimens was significantly higher than in adjacent non-cancerous tissues. B. Statistic of number of cases with higher or lower expression of SOX4 in specimens. SOX4 was up-regulated in most of the tumor tissues (74/93), and was expressed at a lower level in most of the adjacent non-cancerous tissues (17/93) ($P < 0.01$). C. Overall survival (OS) analysis according to GC patients' follow-up information was conducted and presented by Kaplan-Meier plot. High SOX4 level in HCC tissue is correlated poor OS (364 cases, P=6.63-03). D. SOX4 was knocked-down in Hep3B cells by using shRNA transfection. RT-qPCR assay was used fot validating the transfection effect. SOX4 was significant defected ($^{**}P < 0.01$). E. The CCK8 assay showed a significant inhibition of cell proliferation by knocking-down SOX4 ($^{*}P < 0.01$, $^{**}P < 0.01$). F. The representative histograms describing cell apoptosis status was generated according to the result from the flow cytometry detection. The apoptosis rate of Hep3B cells was significantly increased from 10.28% to 26.54% ($^{**}P < 0.01$) through knocking-down AKR1B10P1. The results are means of three independent experiments ± SD. ($^{**}P < 0.01$). G. According to the CCK8 assay result, re-introducing AKR1B10P1 significantly promoted cell proliferation in Hep3B cells knocked down SOX4 ($^{*}P < 0.01$, $^{**}P < 0.01$). H. Re-introducing AKR1B10P1 significantly rescued the increase of Hep3B cell apoptosis induced by knocking down SOX4 from 26.39% to 11.12% ($^{**}P < 0.01$).

Table S2. Selected sequence of the predicted miR-138 binding site of AKR1B10P1 transcript and the 3'-UTR of SOX4 mRNA, along with the relative mutated sequence

Genes	Sequence including the binding site (202 bp)	Relative mutated sequence
AKR1B10P1 transcrpit	5'-agattaaggagattgctgcaaggcacaaaaaaccacagcccaggttctgatccatttccatatccagaggaatgtgactgtgatccccaagtctgtgacaccagcacgcattgttgagaacattcaggcctttgactttacattgaatgatgaggagatggcaaccacactcagcttcaacagaaactggagggcccgtaac-3'	5'-acaattacgtgttaggtcctacggaaatatatcgagaccgctgctacagttgcttatgcttttgctgtgcattctcagtctcaacgcgatgacagagtgtggtcggcccttagatcacatcttactgccgtatcagtattcttagtaagttcacgtgttcggatcgagagtgaccatgatctgtatcagcacgccgccttag-3'
3'-UTR of SOX4 mRNA	5'-agatttctgtataagactgttgagcagtttttaaaatagtgtaggataatataaaaagcagatagatggcgctatgtttgattcctacaacgaaattatcaccagctttttttcattcttaactctttaaaggattcaaacgcaactcaaatctgtgctggactttaaaaaaacaattcaggaccaaatttttttctcagtgtgtg-3'	5'-acaatacagtaaatgtctgatcacctgatataatatttgagaacgtttaaaaatatacctgtttgttcggggtttctatcaatgcaagatccataatttgtggtcgtatatatgaatgtaatcacataatacgttactatccctagtgataacagaggtcgtcataatatatagattactgcagctattatatacactgagagag-3'