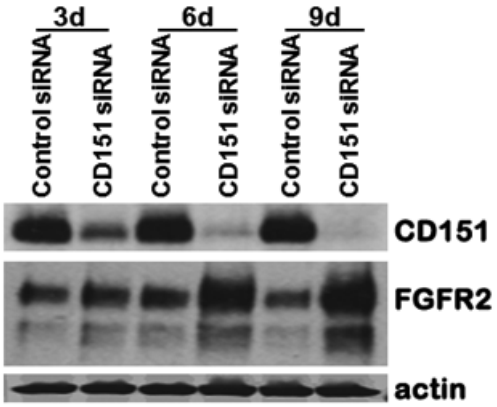
Supplementary Fig.1



Transient knockdown of CD151 in HB2 cells resulted in the increased level of FGFR2. Note, the correlation between the degree of CD151 knockdown and the increase in the expression level of FGFR2.

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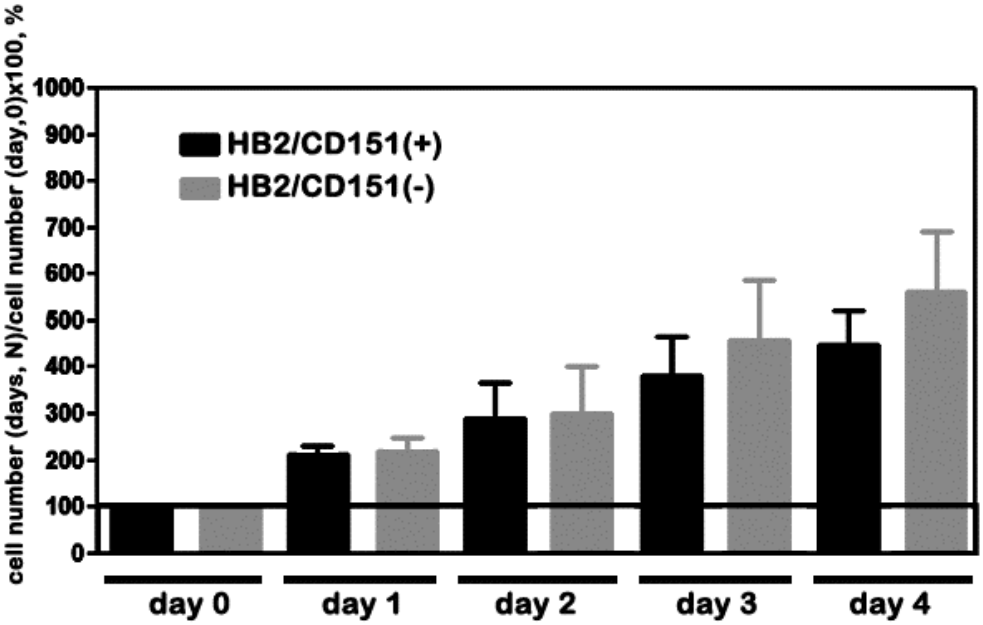
Supplementary Fig.2



Knockdown of CD151 does not affect proliferative responses of HB2 cells to FGF2. Cells were serum starved and subsequently incubated in serum-free growth media containing FGF2 (50ng/ml) for indicated time intervals. Number of cells on a given day was counted, and ratio to the number of cells plated ("day 0") was calculated and multiplied by 100. Cells were plated in triplicated for each of the time points.

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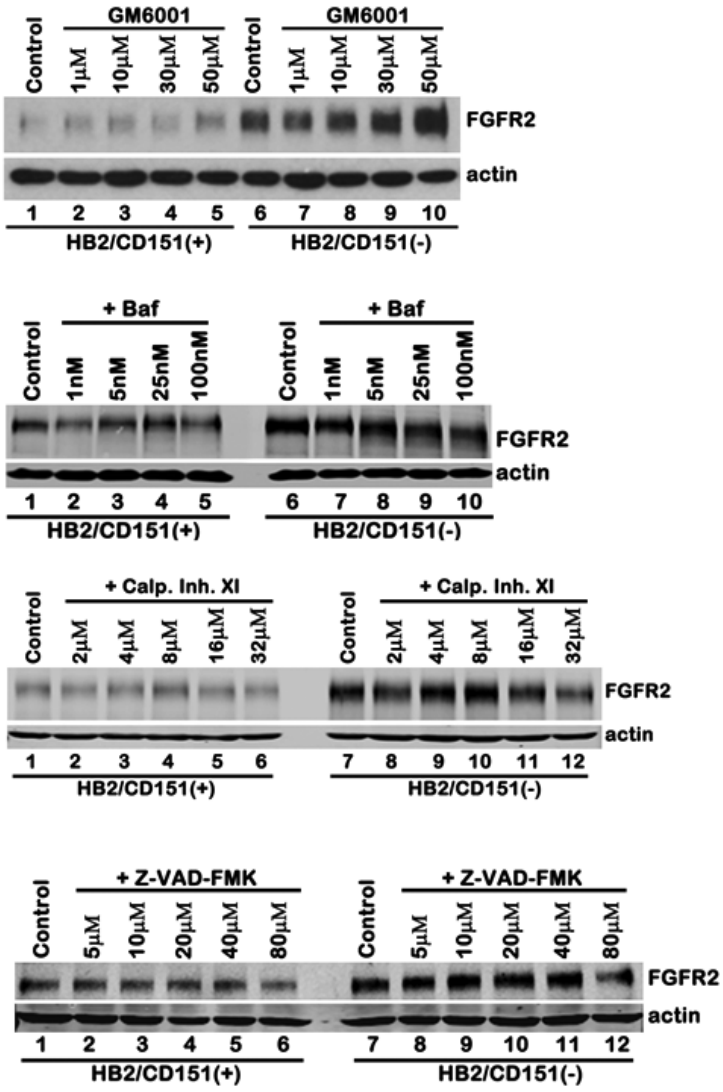
Supplementary Fig.3



The effect of proteolytic inhibitors on the expression levels of FGFR2 in HB2 cells. Cells were incubated with indicated concentrations of the inhibitors for 16 hours and the expression of FGFR2 was analysed by Western blotting.

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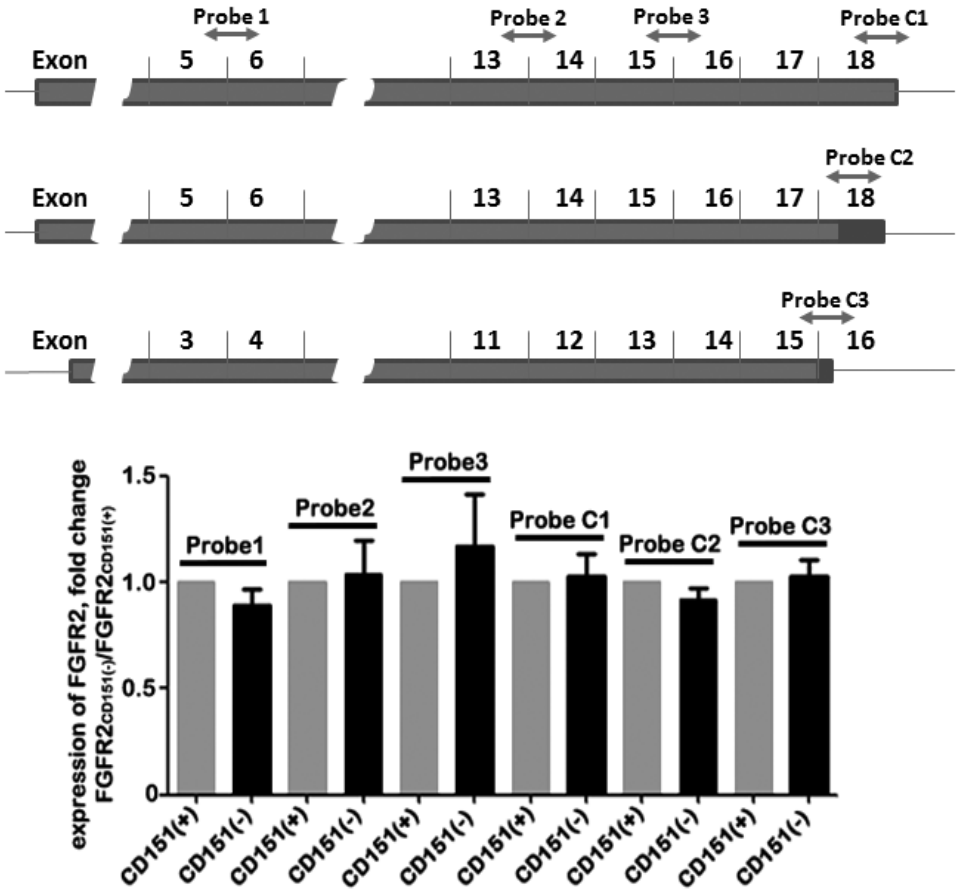
Supplementary Fig.4



Analysis of the mRNA FGFR2 expression in HB2 cells by RT-qPCR. Top diagram shown positions of the amplified regions in the mRNA FGFR2.

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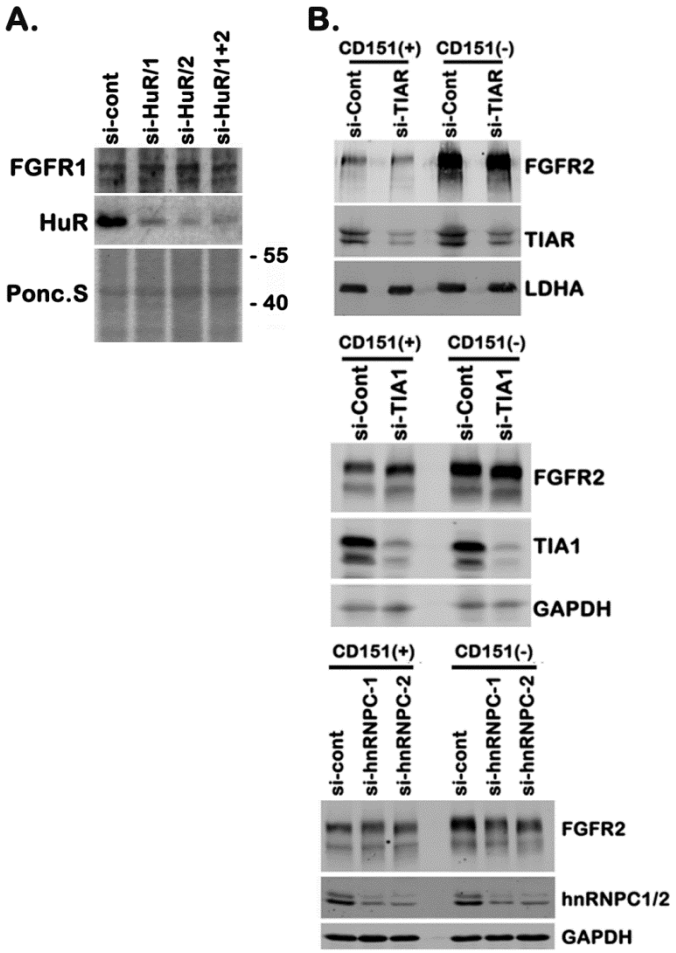
Supplementary Fig.5



A. Knockdown of HuR/ELAVL-1 does not affect expression of FGFR2. **B.** Knockdown of various RNA binding proteins has no or minimal effect on the FGFR2 expression in HB2/CD151(+) and HB2/CD151(-) cells, “CD151(+)” and “CD151(-)” respectively. The expression of FGFR2 was assessed by Western blotting 72 hours after transfection.

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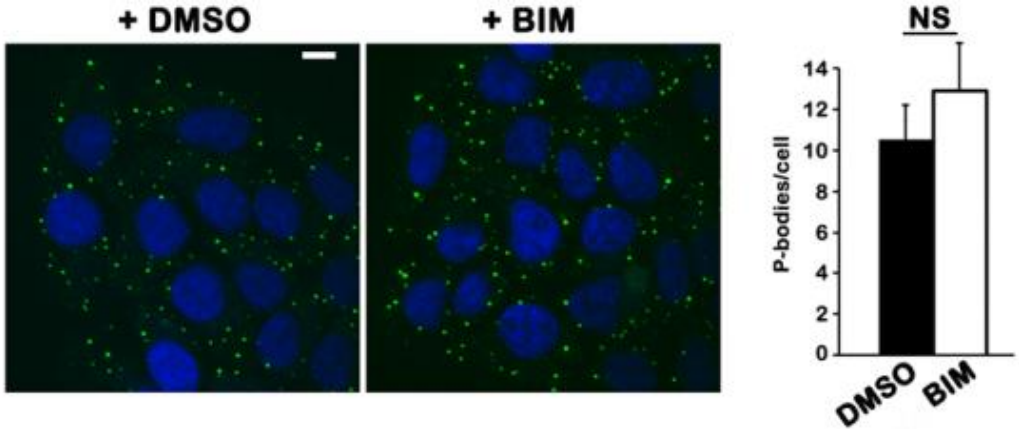
Supplementary Fig.6



Treatment of HB2/CD151(+) cells with a PKC inhibitor has no effect on the assembly of P-bodies. Cells were grown under standard culturing conditions in media supplemented with BIM I (2μM) for 72 hours with daily media change. P-bodies were visualised with anti-EDC4 Ab. Numbers of P-bodies was quantified in at least 30-50 cells. Scale bar, 10 μm.

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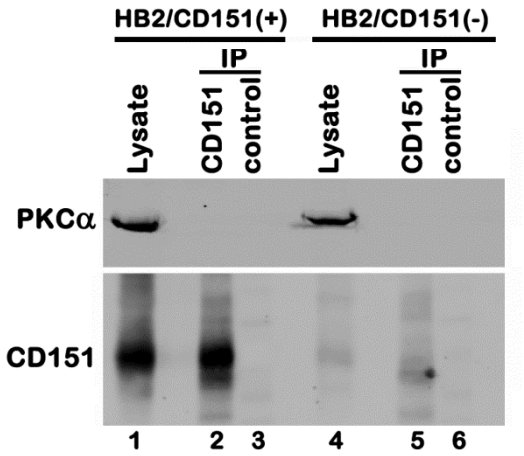
Supplementary Fig. 7



Cells grown under standard culturing conditions were lysed in 1%Brij98 and CD151-containing complexes were precipitated using mouse anti-CD151 mAb (5C11). The complexes were resolved in SDS-PAGE and the presence of PKCα in the complex was examined using rabbit anti-PKCα Ab. Mouse anti-GFP mAb were used as isotype-specific negative control.

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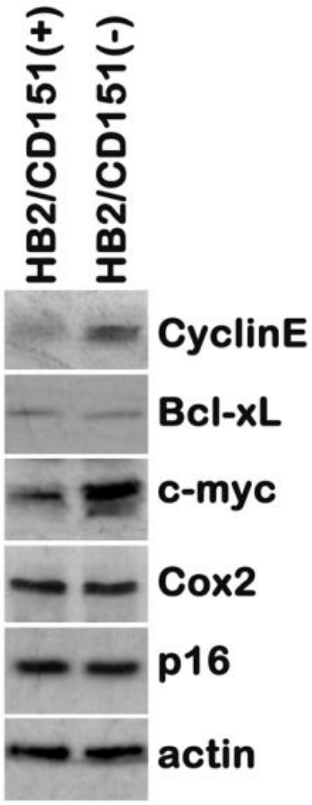
Supplementary Fig. 8



The effect of CD151 knockdown on the expression of cellular proteins that were identified as HuR/ELAVL-1 targets.

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Supplementary Table 1

gene	siRNA sequence
PatL1	GGACCUUUCUGAACGAGCA
PatL1	CGUCGACUCUUGCAUCAGA
TIA-1	CGCUCCAAAGAGUACAUAU
TIA-1	GAUAUUCAUUUGUUCGGUU
TIAL	GGAUUUGGAGUAGAUCAAU
TIAL	GUAAACCACCUGCACCUAA
EDC4	GGUGUCUGCACGAGUGGAA
EDC4	GGAGAUGAUAGCUCCACCU
hnRNPC	AACGUCAGCGUGUAUCAGGAA
hnRNPC	UUGGUGAUACCUCAUCCUUGA
ELAVL1/HuR	AAGUAGCAGGACACAGCUUGG
ELAVL1/HuR	ACCAGUUUCAAUGGUCAUAAA
α6 integrin subunit	GGUCGUGACAUGUGCUCAC
α6 integrin subunit	CAAGACAGCUCAUAUUGAUUU
CD151	CAUGUGGCACCGUUUGCCU

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Supplementary Table 2.

Patient characteristics.

Number of patients	166
Age (years)	
< 50	52
≥ 50	114
Disease stage	
I	39
II	84
III	43
T status	
T1	55
T2	103
T3	1
T4	7
Grade	
1-2	95
3	71
Nodal status	
Negative	82
Positive	84
Steroid receptor status	
Negative	73
Positive	93
HER2 status	
Negative	138
Positive	28

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