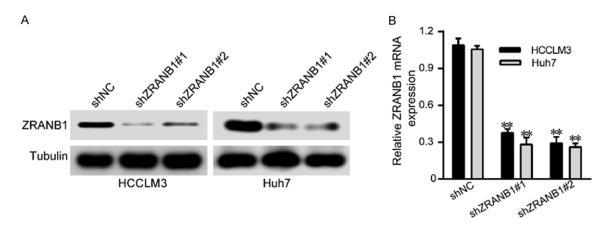
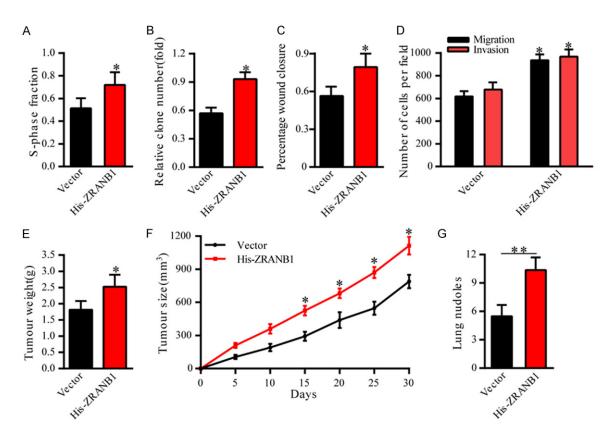
	•	
name	sequence	accession number
ZRANB1 forward	5'-ACAGCCTGCATGACTGTTCA-3'	NM_017580.3
ZRANB1 reverse	5'-CCCAGTCTTCTTGCCACTGT-3'	NM_017580.3
LOXL2 forward	5'-CCAGTGTGGTCTGCAGAGAG-3'	NM_002318.3
LOXL2 reverse	5'-CCTGTGCACTGGATCTCGTT-3'	NM_002318.3
SP1 forward	5'-CCCTTGAGCTTGTCCCTCAG-3'	NM_138473.3
SP1 reverse	5'-TGAAAAGGCACCACCACCAT-3'	NM_138473.3
GAPDH forward	5'-CCATGGGGAAGGTGAAGGTC-3'	NM_002046.7
GAPDH reverse	5'-TGAAGGGGTCATTGATGGCA-3'	NM_002046.7

Supplementary Table 1. Primers sequences

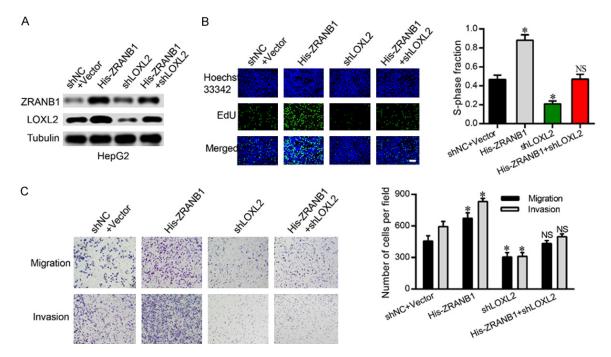


Supplementary Figure 1. ZRANB1 shRNA sequences transfection efficiency. A and B. The ZRANB1 knockdown efficiency was confirmed by western blotting and qRT-PCR analysis (*p<0.05).

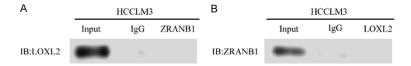


Deubiquitinase promotes ZRANB1 HCC tumorigenesis through SP1-LOXL2 axis

Supplementary Figure 2. ZRANB1 upregulation facilitates HCC cells progression *in vitro* and *in vivo*. A and B. Cells proliferation capacities were detected by EdU assay and cell colony formation assay in HepG2 cells transfected with Vector or His-ZRANB1 (*p<0.05). C and D. The scratching assay and Transwell migration and invasion assays were performed to detect the metastasis ability of ZRANB1 overexpressing HepG2 cells. E and F. Tumor weights and tumor sizes of HepG2-Vector or HepG2-His-ZRANB1 groups of nude mice were measured, and corresponding tumor growth curves were obtained (means \pm SEM; *p<0.05). Tumor weight obtained during 30 days after inoculation (*p<0.05, as determined by Student's t-test). G. Incidence of lung metastasis of each group (*p<0.01).



Supplementary Figure 3. LOXL2 is key for ZRANB1-mediated HCC cells progression. A. Western blotting was used to detect the expression of ZRANB1 and LOXL2 in HepG2 cells. The upregulation of ZRANB1 abated the decrease of LOXL2 expression in HepG2-shLOXL2 cells. B. EdU assays showed that the upregulation of ZRANB1 significantly rescued the cell proliferation in HepG2-shLOXL2 cells (*p<0.05; NS, not significant; scale bar, 100 µm). C. The upregulation of ZRANB1 significantly rescued the cell migration and invasion suppression in HepG2-shLOXL2 cells (*p<0.05; NS, not significant).



Supplementary Figure 4. There is no interaction between ZRANB1 and LOXL2. A and B. ZRANB1 can not bind with LOXL2 directly through co-IP experiments in HCCLM3 cells.