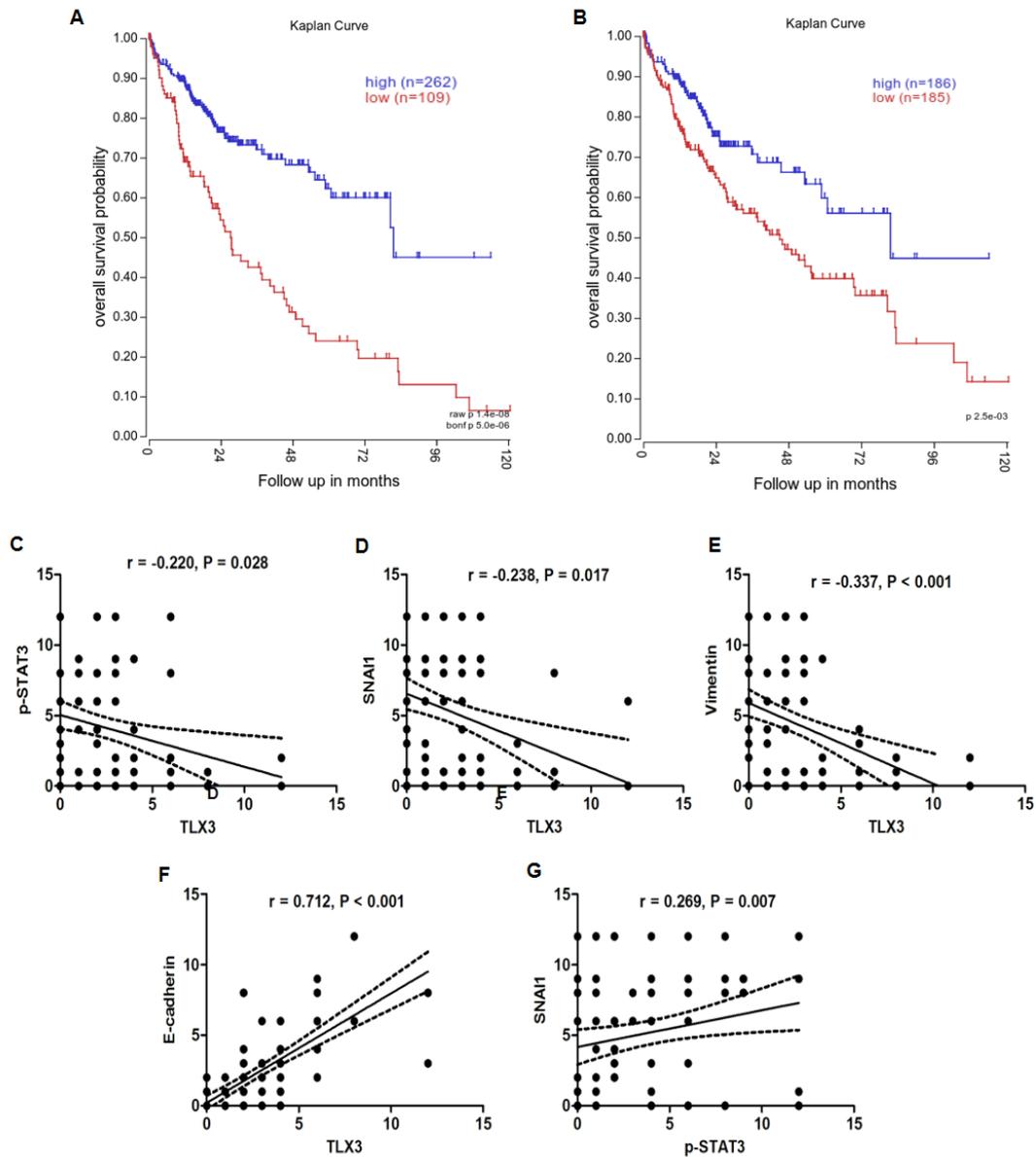


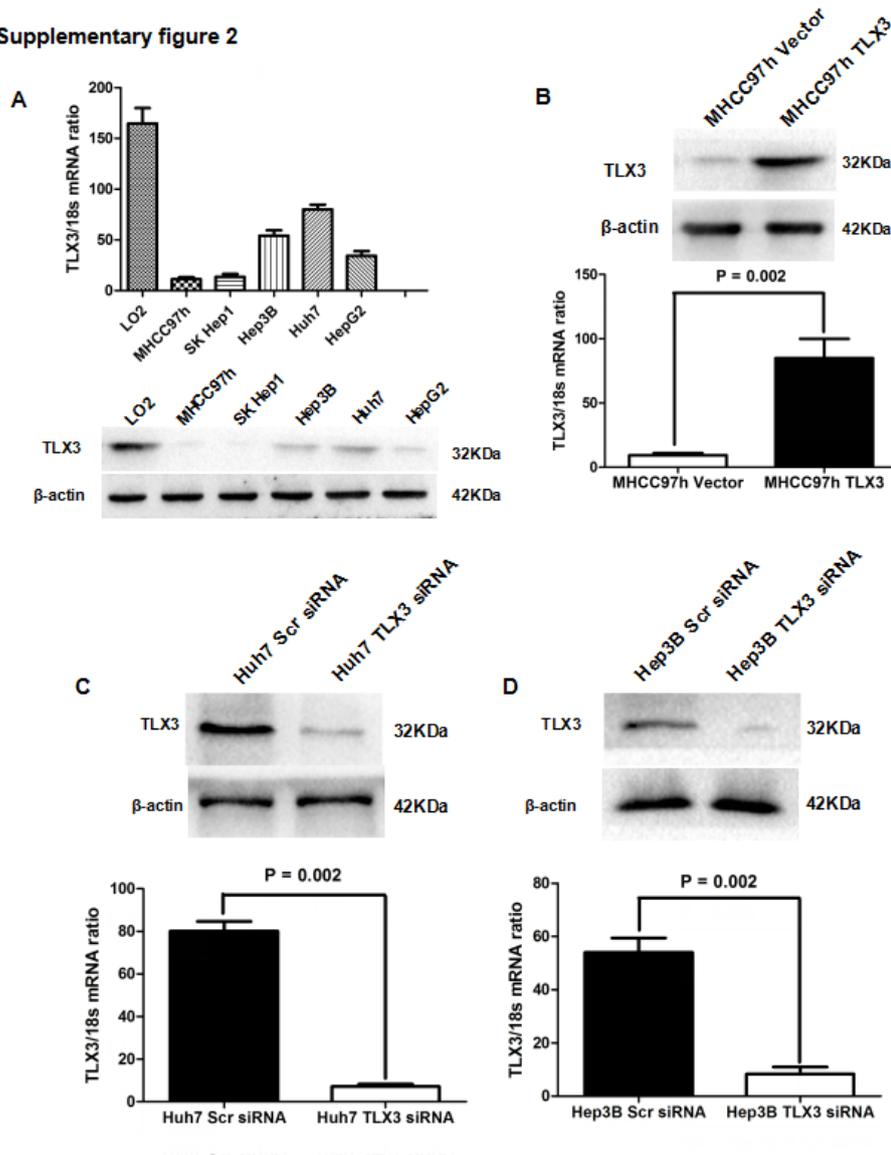
Supplementary fig 1



Supplementary fig.1 The relationship between TLX3 expression and survival in HCC was analyzed in TCGA database. (A) Using the ratio of TLX3 expression in HCC/adjacent liver tissues as the cut-off value, HCC patients with lower TLX3 expression in HCC tissues than adjacent liver tissues suffered from the unfavorable survival compared to those with higher TLX3 expression ($P = 1.4 \text{ e-}8$). (B) Using the median value of TLX3 expression in HCC tissues as the cut-off value, HCCs with higher TLX3 in HCC tissues had the better

survival ($P = 2.5 \times 10^{-3}$). The results of IHC staining on HCC specimens were analyzed by Spearman test and it was found that TLX3 expression was associated negatively with the expression of p-STAT3 (C), SNAI1 (D) and Vimentin (E) and positively with E-cadherin expression (F). And there was also positive correlation found between p-STAT3 and SNAI1 in HCC tissues (G).

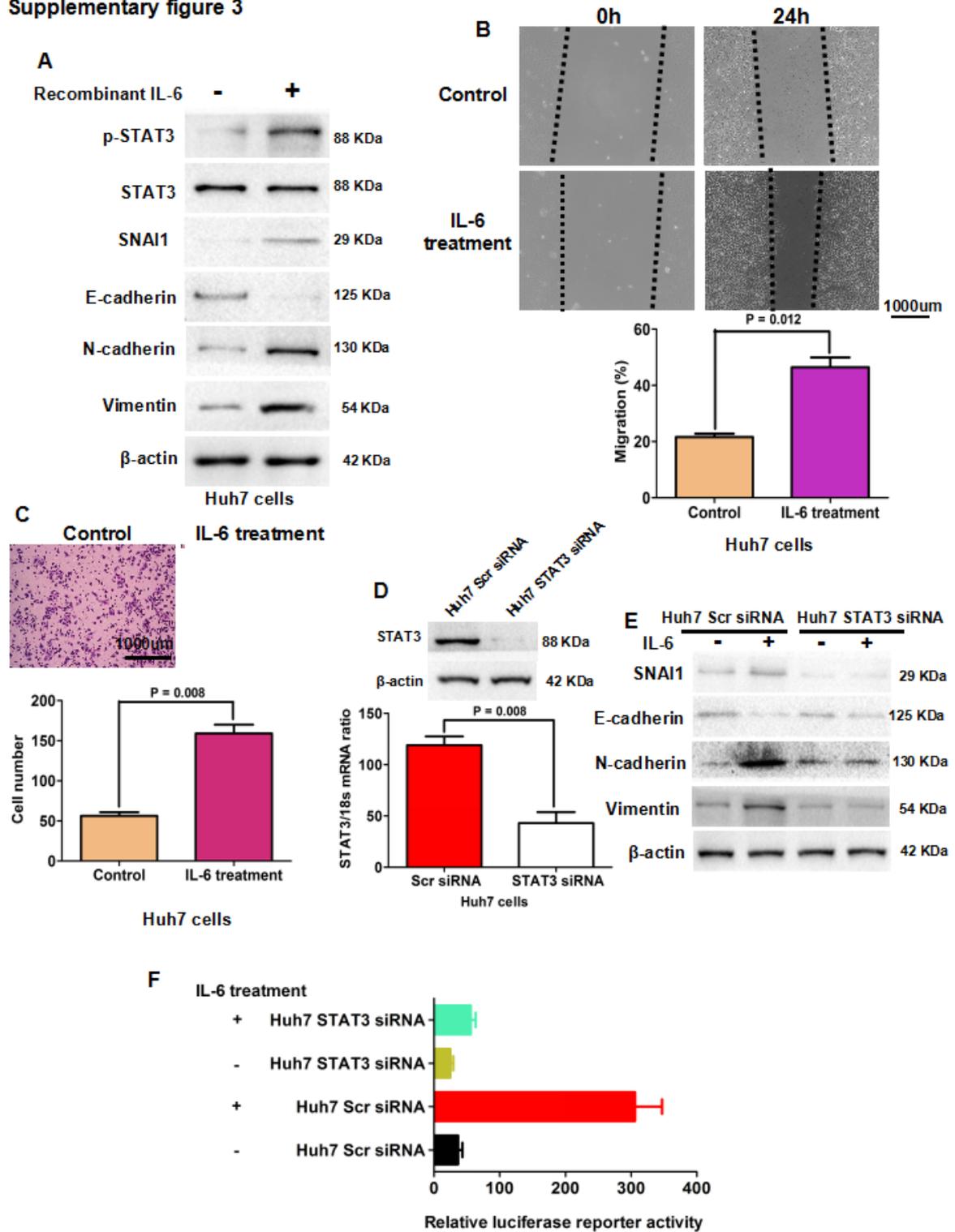
Supplementary figure 2



Supplementary fig.2 By both qRT-PCR and Western immunoblotting assays, it was displayed that TLX3 expression in 5 kinds of HCC cell lines was significantly less than normal liver cell line (LO2) (A); Transfection with TLX3 expressing plasmid was verified to result in increase of TLX3 expression in MHCC97h cells by both qRT-PCR and Western immunoblotting assays (B); Additionally, qRT-PCR and Western immunoblotting assays

also confirmed that transfection of siRNA sequences against TLX3 abolished TLX3 expression in both Huh7 (C) and Hep3B cells (D) successfully.

Supplementary figure 3



Supplementary fig.3 Western immunoblotting assay showed that IL-6 treatment led to increased expression of SNAI1, N-cadherin and Vimentin and repression of E-cadherin accompanied with increased phosphorylation of STAT3 (A); As assessed by wound

healing assay, IL-6 treatment amplified migration ability of Huh7 cells (B); Transwell assay also demonstrated that IL-6 resulted in up-regulation of invasion capacity of Huh7 cells (C); Transfection of siRNAs targeting STAT3 abrogated STAT3 expression in Huh7 cells successfully (D); Western immunoblotting assay revealed that IL-6 treatment did not impact the expression of EMT bio-markers including SNAI1, E-cadherin, N-cadherin and Vimentin any more (E); The luciferase reporter assay confirmed that IL-6 treatment gave rise to notable up-regulation of SNAI1 promoter activity, which was revoked by knockdown of STAT3 in Huh7 cells (F).

Table 1 The relationship between TLX3 expression in tumor tissues and clinical characteristics in 100 HCC cases.

| Clinicopathological features | | No. | No. of Patients | | χ^2 | P |
|---|----------|-----|-------------------|------------------|----------|-------|
| | | | Lower TLX3 in HCC | High TLX3 in HCC | | |
| Age (years) | < 50 | 44 | 30 | 14 | 0.568 | 0.451 |
| | ≥ 50 | 56 | 42 | 14 | | |
| Gender | Male | 54 | 39 | 15 | 0.003 | 0.957 |
| | Female | 46 | 33 | 13 | | |
| HBV infection | Present | 87 | 67 | 20 | 8.337 | 0.004 |
| | Absent | 13 | 5 | 8 | | |
| Serum AFP level (ng/mL) | < 400 | 26 | 17 | 9 | 0.763 | 0.383 |
| | ≥ 400 | 74 | 55 | 19 | | |
| Tumor diameter (cm) | < 5 | 55 | 44 | 11 | 3.880 | 0.049 |
| | ≥ 5 | 45 | 28 | 17 | | |
| Liver cirrhosis | Present | 86 | 66 | 20 | 6.858 | 0.009 |
| | Absent | 14 | 6 | 8 | | |
| Edmondson-St einer Classification | I + II | 30 | 17 | 13 | 4.998 | 0.025 |
| | III + IV | 70 | 55 | 15 | | |
| TNM stage | I + II | 69 | 45 | 24 | 5.079 | 0.024 |
| | III + IV | 31 | 27 | 4 | | |

| | | | | | | |
|---------------|---------|----|----|----|-------|-------|
| Portal vein | Present | 21 | 20 | 1 | 7.120 | 0.008 |
| invasion | Absent | 79 | 52 | 27 | | |
| Intra-hepatic | Present | 16 | 15 | 1 | 4.470 | 0.035 |
| metastases | Absent | 84 | 57 | 27 | | |
