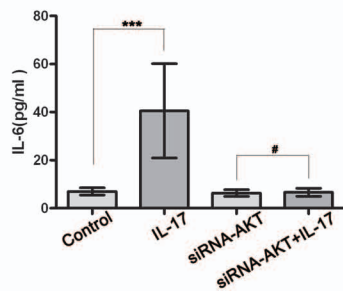
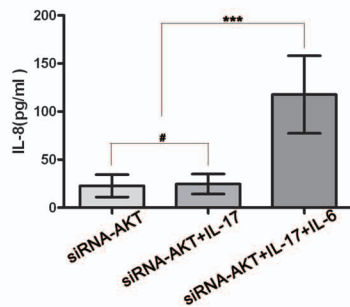
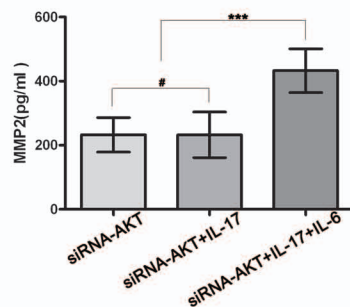
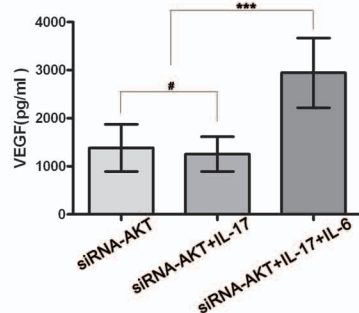
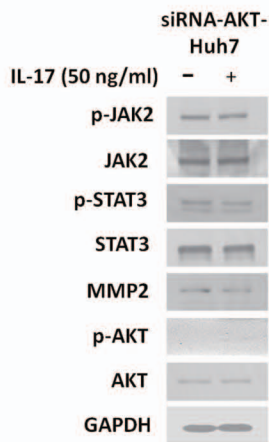
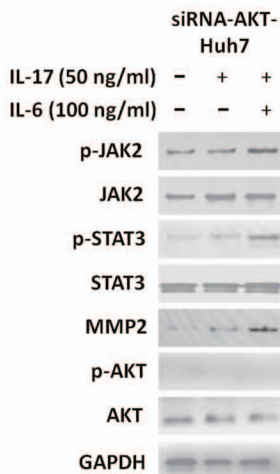


Additional file 4

Figure S4 IL-17 promotes HCC invasion via AKT-dependent IL-6/STAT3 activation. Huh7 cells were exposed to AKT-targeted or STAT3-targeted siRNA, and then cultured with IL-17 (50 ng/ml) and/or IL-6 (100 ng/ml) for 24 h. **(A)** As assessed by ELISA, the expression of IL-6, IL-8, MMP2 and VEGF showed no significant change after IL-17 stimulation in siRNA-AKT-Huh7 cells, while expression of IL-8, MMP2 and VEGF were significantly increased after addition of IL-17 plus IL-6. **(B)** AKT-siRNA significantly downregulated JAK2/STAT3 phosphorylation induced by IL-17. **(C)** IL-6 also recovered IL-17-stimulated JAK2/STAT3 phosphorylation in siRNA-AKT-Huh7 cells. **(D)** As shown by Matrigel invasion assay, STAT3-siRNA significantly reversed tumor invasion by IL-17 stimulation. IL-6 mAb (10 ng/ml for 36 h) completely reversed IL-17-induced HCC invasion, while IL-6 (100 ng/ml for 36 h) completely recovered IL-17-stimulated invasion of siRNA-AKT-Huh7 cells. Three separate experiments were performed in each study. Data are expressed as mean \pm SD; Student's *t* test; # $p > 0.05$; * $p < 0.05$, ** $p < 0.01$, and *** $p < 0.001$.

A**IL-6****IL-8****MMP2****VEGF****B****C****D**