CORRECTION

Correction: Hypoxia-induced TUFT1 promotes the growth and metastasis of hepatocellular carcinoma by activating the Ca^{2+}/PI3K/AKT pathway

Changwei Dou, Zhenyu Zhou, Qiuran Xu, Zhikui Liu, Yuqun Zeng, Yufeng Wang, Qing Li, Liang Wang, Wei Yang, Qingguang Liu and Kangsheng Tu

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After publication, the authors regret that the original version of this paper contained some incorrect representative images. The images of migrated and invaded HCCLM3 cells with NT shRNA transfection in Fig. 2F and the image of migrated Hep3B cells with LY294002 treatment in Fig. 5G were mis-pasted. A wrong image was placed in the TUFT1+Vehicle group in Fig. 5F when choosing a representative image from the countless image data. The authors confirm that the errors do not affect the results or conclusions of the study, and apologize for the mistake and any confusion this may have caused.

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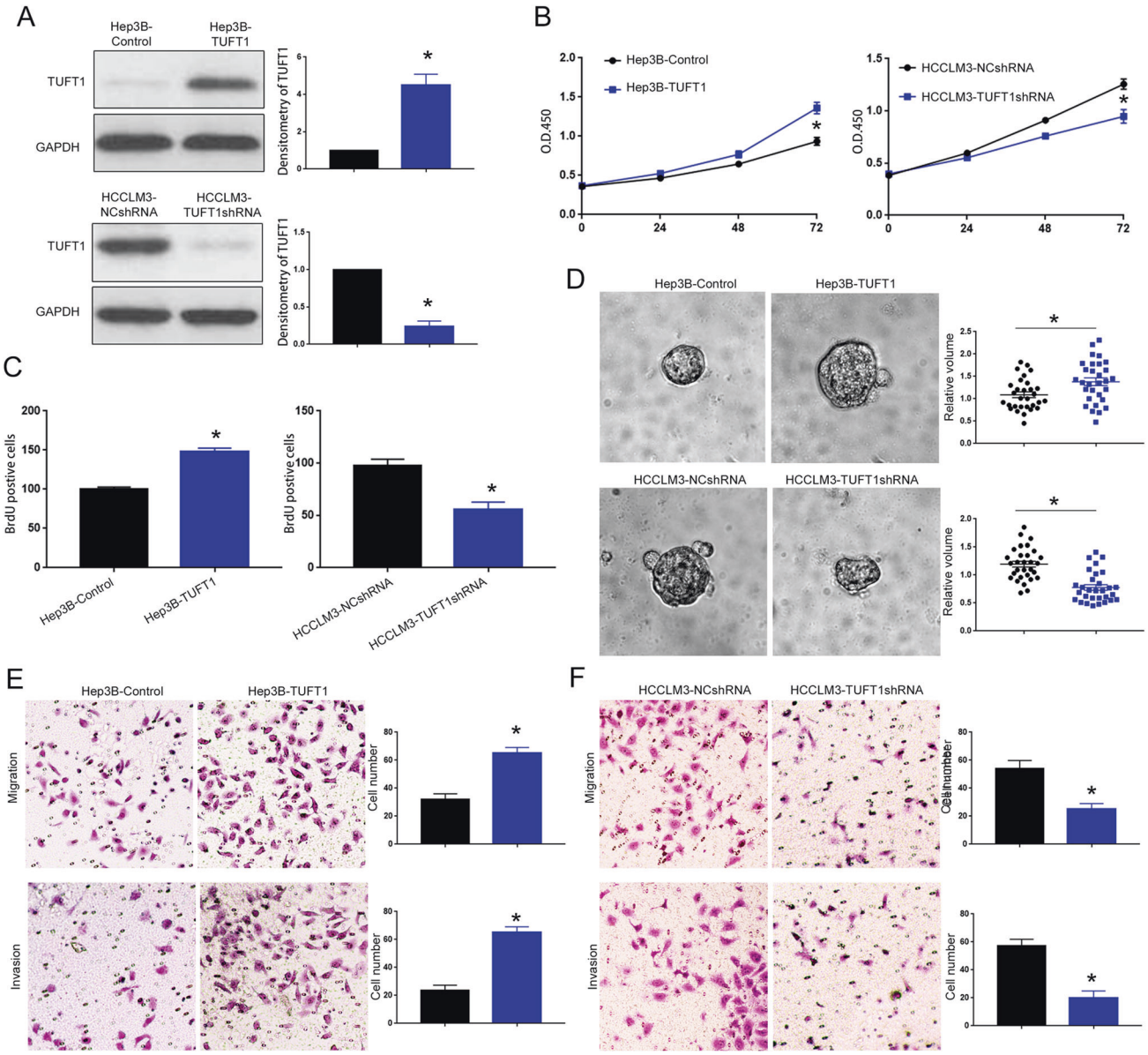

Fig. 2 TUFT1 promotes proliferation, migration and metastasis of HCC cells in vitro. A Retrovirus encoding TUFT1 vector were transduced into Hep3B cells to establish Hep3B cells stably overexpressing TUFT1 (Hep3B-TUFT1 cells). Lentivirus encoding TUFT1 shRNA were transduced into HCCLM3 cells to establish HCCCLM3 cells with TUFT1 knockdown (HCCLM3-TUFT1 shRNA cells). Western blot was performed to confirm the overexpression or knockdown of TUFT1 in corresponding cells. $n =$ three independent experiments, $^*P < 0.05$ by t test. **B** MTT assay was performed to evaluate the effect of TUFT1 on the viability of Hep3B and HCCLM3 cells. $n =$ three independent experiments, $^*P < 0.05$ by two-way ANOVA. **C** BrdU assay was performed to evaluate the effect of TUFT1 on the proliferation of Hep3B and HCCLM3 cells. $n =$ five randomly selected fields of three independent experiments, $^*P < 0.05$ by t test. **D** 3D culture was used to investigate the effect of TUFT1 on growth of Hep3B and HCCLM3 cells. $n = 30$, $^*P < 0.05$ by t test. **E**, **F** Transwell assay was used to investigate the effect of TUFT1 on the migration and invasion of Hep3B and HCCLM3 cells. $n =$ five randomly selected fields of three independent experiments, $^*P < 0.05$ by t test.

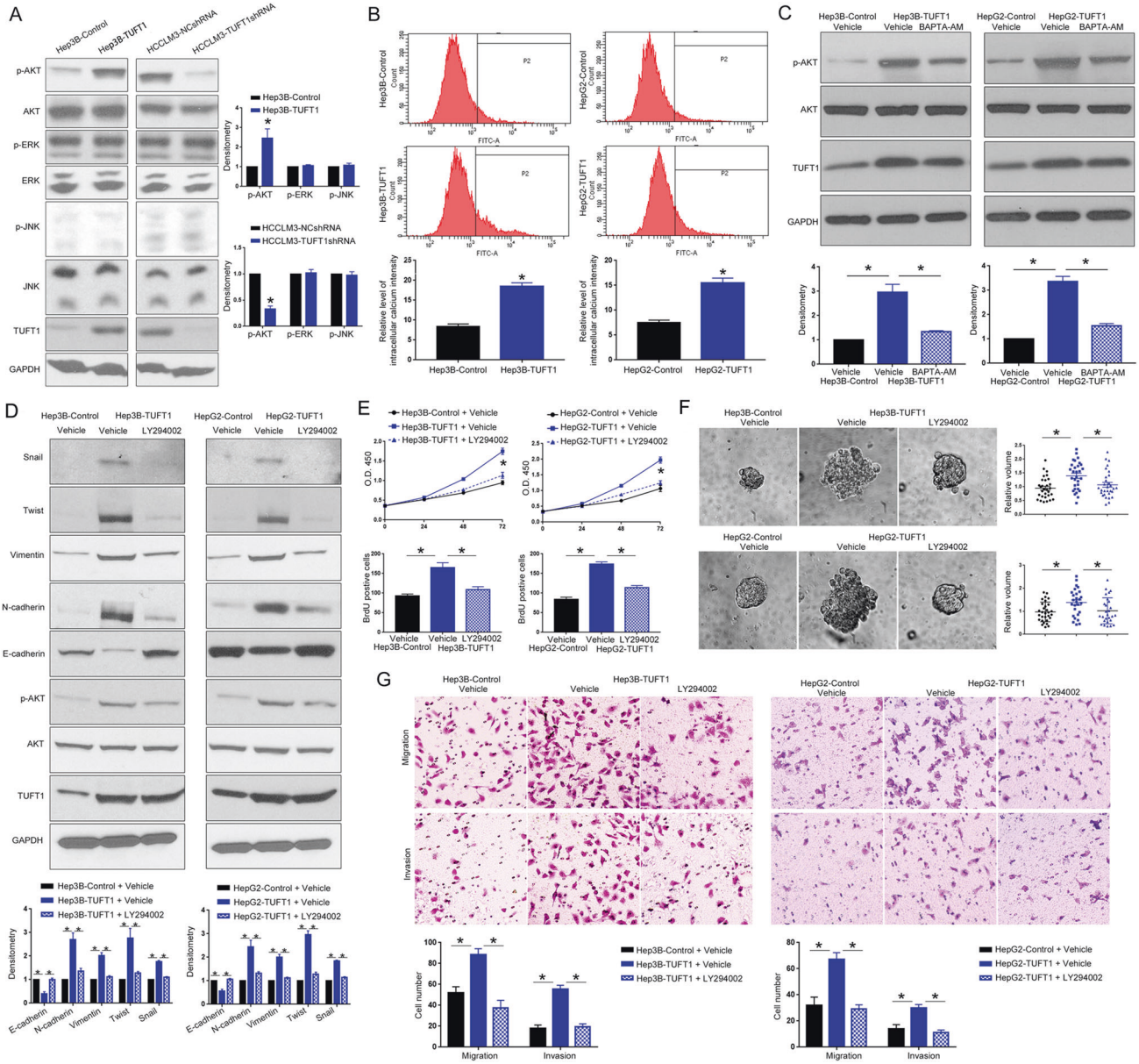

Fig. 5 TUFT1 exerts oncogenic effects on HCC cells through activating Ca^{2+}/PI3K/AKT pathway. A Western blot was performed to investigate the influence of TUFT1 on AKT, ERK and JNK pathways in Hep3B and HCCLM3 cells. The levels of p-AKT, p-ERK and p-JNK, activation markers of AKT, ERK and JNK pathways, were examined. GAPDH was used as internal control. *$P < 0.05$ by *t* test. **B** Cytosolic calcium immunofluorescence was evaluated by flow cytometry. Vertical lines were set at the same fluorescence intensity level and cells on the right proportion were considered to be $[Ca^{2+}]_{cyto}$ positive. Overexpression of TUFT1 significantly increased intracellular calcium level of Hep3B and HepG2 cells. *$P < 0.05$ by *t* test. **C** Hep3B and HepG2 cells overexpressing TUFT1 and corresponding cells in control group were treated with or without BAPTA-AM (10 μM) and subjected to western blot. Intracellular calcium chelation blocked the phosphorylation of AKT induced by TUFT1 overexpression. *$P < 0.05$ by *t* test. **D**–**G** Hep3B and HepG2 cells overexpressing TUFT1 and corresponding cells in control group were treated with LY294002 (an inhibitor of PI3K) for 24 h, and were subjected to **D** western blot, **E** MTT and BrdU assay, **F** 3D culture assay, and **G** Transwell assay for migration and invasion. PI3K inhibition by LY294002 blocked the phosphorylation of AKT and thus abrogated the promoting effects of TUFT1 overexpression on EMT, cell viability and proliferation, growth, migration and invasion. *$P < 0.05$ by ANOVA. $n =$ three independent experiments.