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Erratum

The following was originally published in Volume 25, No. 8, pages 1329–1340, 2017 (doi: https://doi.org/10.3727/096504017X14876227286564). Recently, it was discovered that Figure 5 displayed overlapping images, which unfortunately caused the images to be incorrect. To correct the problem, the experiment was repeated so the errors in the images could be corrected. The figure has also been replaced with the corrected version in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2017/00000025/00000008/art00012). The results of the repeated experiments were in adherence to our original conclusion.

TRAF4 Regulates Migration, Invasion, and Epithelial–Mesenchymal Transition via PI3K/AKT Signaling in Hepatocellular Carcinoma

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Overexpression of the tumor necrosis factor receptor-associated factor 4 (TRAF4) has been detected in many cancer types and is considered to foster tumor progression. However, the role of TRAF4 in hepatocellular carcinoma (HCC) remains elusive. In this study, we found that TRAF4 was highly expressed in HCC cell lines and HCC tissues compared with normal liver cell lines and adjacent noncancerous tissues. TRAF4 overexpression in HCC tissues was correlated with tumor quantity and vascular invasion. In vitro studies showed that TRAF4 was associated with HCC cell migration and invasion. An in vivo study verified that TRAF4 overexpression facilitated metastasis in nude mice. In addition, overexpressed TRAF4 promoted the phosphorylation of Akt and induced Slug overexpression, leading to downregulated E-cadherin and upregulated vimentin, while silencing TRAF4 moderated the phosphorylation of Akt and repressed the expression of Slug, which resulted in upregulated E-cadherin and downregulated vimentin. These effects were inversed after pretreatment of the PI3K/Akt inhibitor LY294002 or overexpression of constitutively active Akt1. Our study demonstrated that TRAF4 was involved in promoting HCC cell migration and invasion. The process was induced by the EMT through activation of the PI3K/Akt signaling pathway.

Key words: Tumor necrosis factor receptor-associated factor 4 (TRAF4); Hepatocellular carcinoma (HCC); Epithelial–mesenchymal transition (EMT); PI3K/Akt signaling pathway

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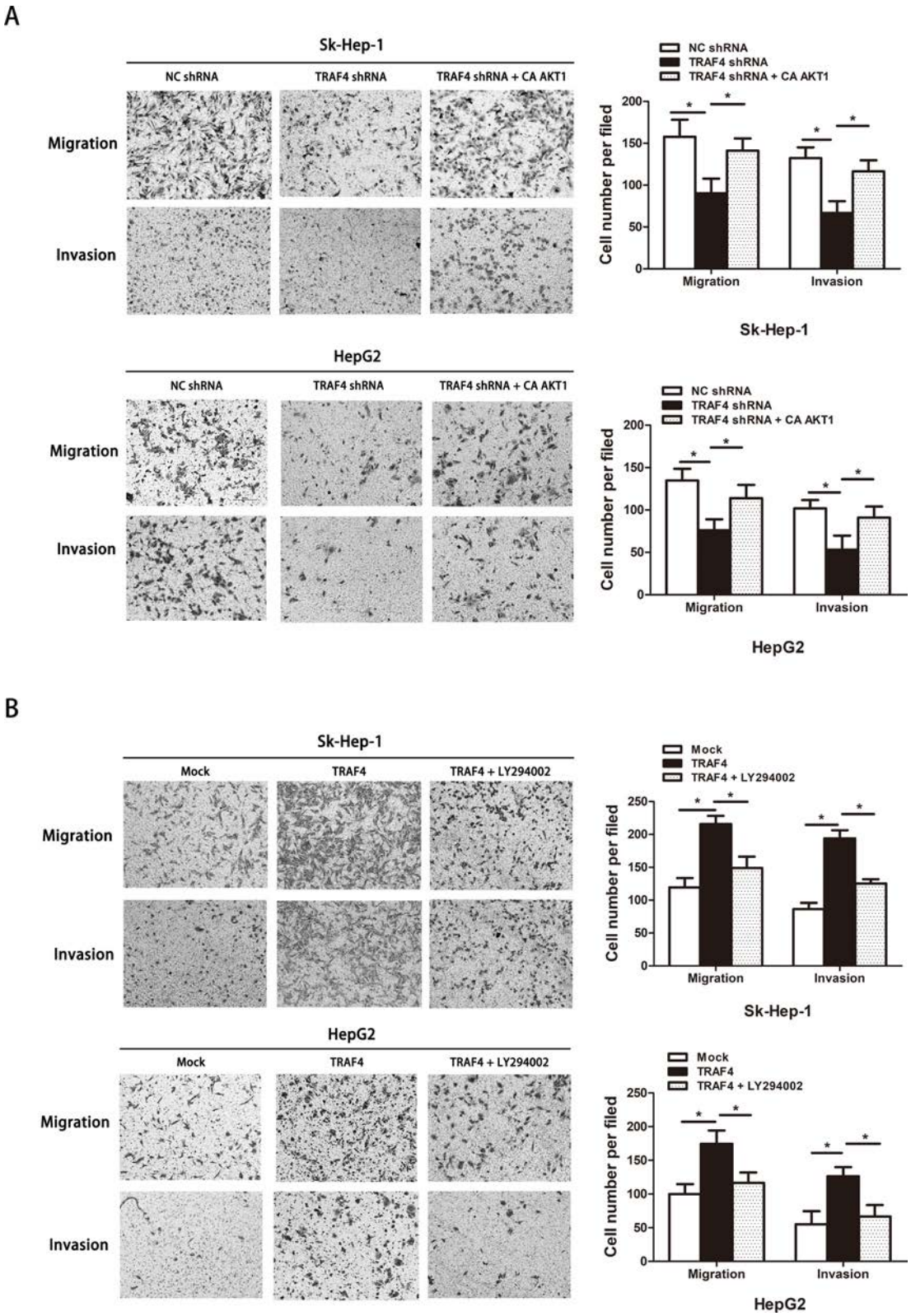

Figure 5. Regulatory effect of PI3K/Akt signaling pathways on TRAF4-induced cell mobility. (A) Constitutive activation of Akt1 recovered cell migration and invasion impaired by TRAF4 downregulation ($p < 0.05$). (B) PI3K/Akt inhibitor LY294002 hampered the TRAF4-induced cell migration and invasion ($p < 0.05$). Data are presented as the mean ± SD; $n = 6$. *$p < 0.05$. All experiments were repeated independently three times; significant difference between groups: *$p < 0.05$.