DOI: 10.1002/cac2.12210

CANCER COMMUNICATIONS Open Access

ERRATUM

Correction to: Overexpression of amplified in breast cancer 1 (*AIB1*) gene promotes lung adenocarcinoma aggressiveness in vitro and in vivo by upregulating c-x-c motif chemokine receptor 4

Following publication of this article [1], the authors noticed incorrect use of images in Figure 4: Western blot for β-actin in Figure 4b, wound-healing assays of A549-vector and A549-AIB1+CXCR4-siRNA-1 cells in Figure 4c, and transwell assays of A549 cells in Figure 4d and H1993 cells in Figure 4f. These images were chosen mistakenly during the figure typesetting process. The corrected Figure 4 is provided below.

The original data has been submitted to the editorial board of *Cancer Communications* and has been approved. The corrections do not change the results and conclusions drawn in this paper.

Author information
Liru He and Haixia Deng contributed equally to this work.

Affiliations
[1]The State Key Laboratory of Oncology in South China, Collaborative Innovation Center for Cancer Medicine, Sun Yat-Sen University Cancer Center, No. 651, Dongfeng Road East, Guangzhou, 510060, China.
Liru He, Haixia Deng, Shiliang Liu, Jiewei Chen, Binkui Li, Chenyuan Wang, Xin Wang, Mengzhong Liu & Dan Xie
[2]Department of Radiation Oncology, Sun Yat-Sen University Cancer Center, Guangzhou, China.
Liru He, Shiliang Liu & Mengzhong Liu
[3]Department of Thoracic Oncology, Sun Yat-Sen University Cancer Center, Guangzhou, China.
Xin Wang
[4]The State Key Laboratory of Respiratory Disease, Guangzhou Medical University, Guangzhou, China.
Yiguo Jiang
[5]Key Laboratory of Protein Modification and Degradation, School of Basic Medical Sciences, Affiliated Cancer Hospital & Institute of Guangzhou Medical University, Guangzhou, China.
Ningfang Ma

Corresponding author
Correspondence to *Menzhong Liu* and *Dan Xie*.

REFERENCE

1. He L, Deng H, Liu S, Chen J, Li B, Wang C, et al. Overexpression of amplified in breast cancer 1 (*AIB1*) gene promotes lung adenocarcinoma aggressiveness in vitro and in vivo by upregulating C-X-C motif chemokine receptor 4. Cancer Communications (London, England). 2018;38(1):53. https://doi.org/10.1186/s40880-018-0320-1.

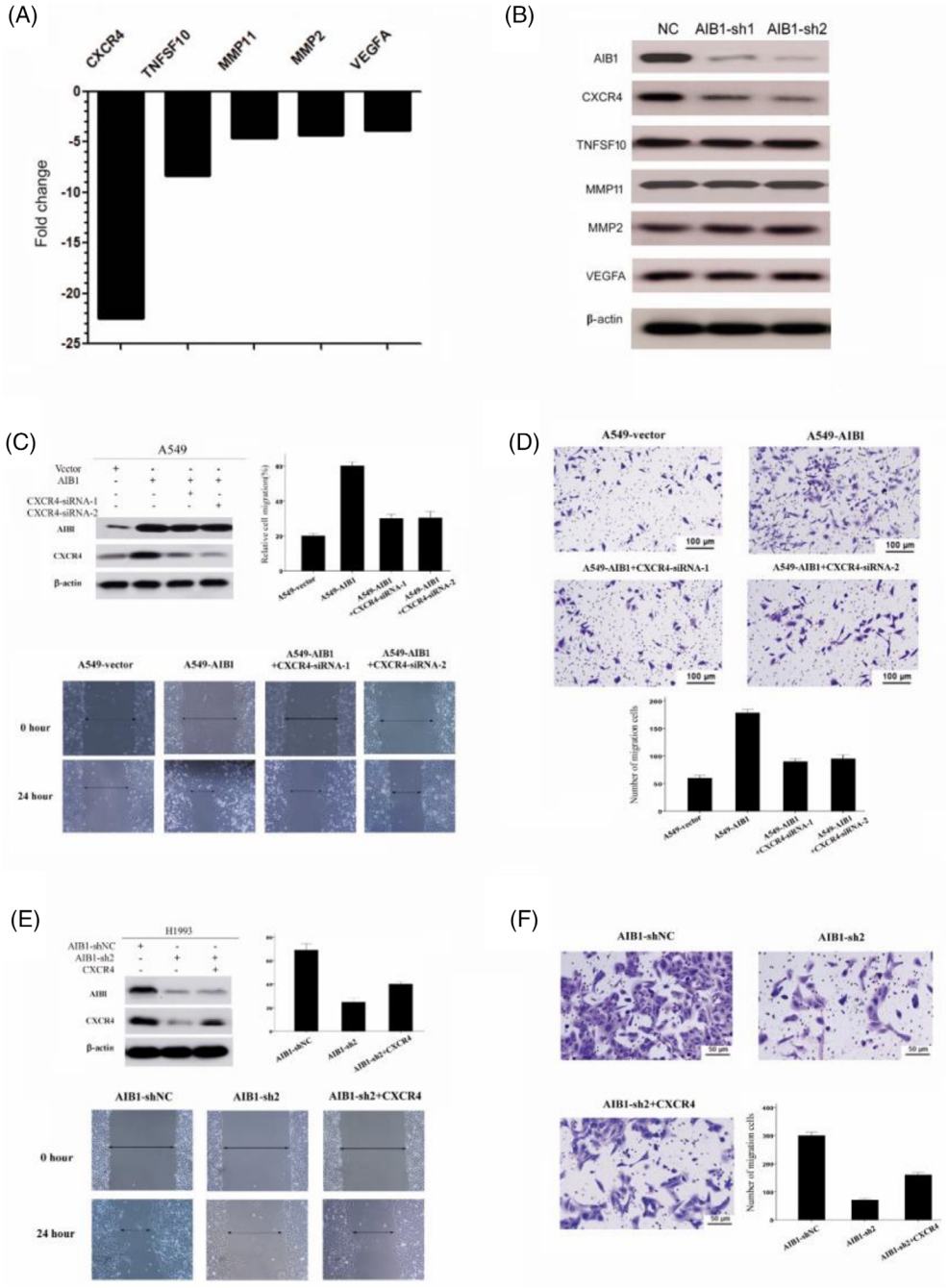

FIGURE 4 The associations of AIB1 and C-X-C motif chemokine receptor 4 (CXCR4) expression in lung adenocarcinoma cells. (**A**) The five genes, CXCR4, tumor necrosis factor (ligand) superfamily member 10 (TNFSF10), matrix metallopeptidase 11 (MMP11), matrix metallopeptidase 2 (MMP2), and vascular endothelial growth factor A (VEGFA), showed more than a 3.5-fold mRNA differential expression in shAIB1-transfected H1993 cells compared with that in control H1993 cells, as shown by using a human tumor metastasis RT2 profiler PCR array. (**B**) Silencing of AIB1 by two shRNAs down-regulated CXCR4 expression in shAIB1 H1993 cells, as detected by Western blotting. (**C**) Upper left: treatment of 2 CXCR4-shRNAs in A549-AIB1cells efficiently decreased the expression levels of CXCR4 as detected by Western blotting. Upper right and down: wound-healing assay showed that the enhanced migrative ability in A549-AIB1 cells was inhibited by silencing CXCR4. (**D**) Transwell assay demonstrated that the increased invasive capacity of A549-AIB1 cells was suppressed by CXCR4 silencing. Data are the mean±SE of three independent experiments. $^{**}P < 0.01$, $^{*}P < 0.05$ versus cells transfected with A549-AIB1 by Student's *t* test. (**E**) Upper left: the level of CXCR4 decreased by silence of AIB1, and then increased after the treatment of CXCR4 as detected by Western blotting. Upper right and down: Wound-healing assay showed that the attenuated migrative ability in H1993-shAIB1 cells was enhanced by the overexpression of CXCR4. (**F**) Transwell assay demonstrated that the attenuated invasive capacity of H1993-shAIB1 cells was enhanced by the overexpression of CXCR4. Data are the mean±SE of three independent experiments. $^{**}P < 0.01$, $^{*}P < 0.05$ versus cells transfected with AIB1-shNC by Student's *t* test