CORRECTION Open Access

Correction to: Direct inhibition of ACTN4 by ellagic acid limits breast cancer metastasis via regulation of β -catenin stabilization in cancer stem cells



Neng Wang^{1,2}, Qi Wang², Hailin Tang¹, Fengxue Zhang², Yifeng Zheng², Shengqi Wang², Jin Zhang², Zhiyu Wang^{2*} and Xiaoming Xie^{1*}

Correction to: J Exp Clin Cancer Res 36, 172 (2017) https://doi.org/10.1186/s13046-017-0635-9

Following publication of the original article [1], minor errors we identified in Fig. 6d and Fig. S4; specifically:

- Figure 6d: incorrect image used for shCtrl scratch image (24 h); correct image now used
- Fig. S4: incorrect images used for the invasive MDA-MB-231 cells (25 μ M EA); correct images now used

The corrected figures, produced using the original data, are given here. The correction does not have any effect on the final conclusions of the paper.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13046-022-02341-1.

Additional file 4. The wound healing and chamber invasive assay revealed that breast cancer cell migration and invasion were inhibited by EA in a time- and dose-dependent manner.

Published online: 31 March 2022

Reference

 Wang N, Wang Q, Tang H, et al. Direct inhibition of ACTN4 by ellagic acid limits breast cancer metastasis via regulation of β-catenin stabilization in cancer stem cells. J Exp Clin Cancer Res. 2017;36:172. https://doi.org/10. 1186/s13046-017-0635-9.

The original article can be found online at https://doi.org/10.1186/s13046-017-0635-9.

² Guangdong Provincial Hospital of Chinese Medicine, The Second Clinical Medical College & The Research Center of Integrative Medicine, Guangzhou University of Chinese Medicine, Guangzhou Shi, China



© The Author(s) 2022, corrected publication 2022. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

^{*}Correspondence: wangzhiyu976@126.com; xiexm@sysucc.org.cn

Department of Breast Oncology, Sun Yat-sen University Cancer
Center; State Key Laboratory of Oncology in South China; Collaborative
Innovation Center of Cancer Medicine, Guangzhou, People's Republic
of China

Wang et al. J Exp Clin Cancer Res (2022) 41:118 Page 2 of 2

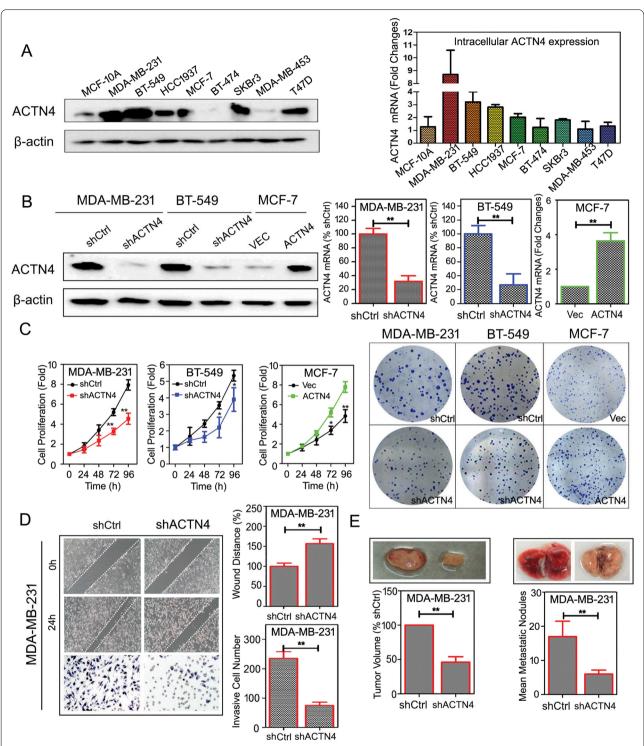


Fig. 6 ACTN4 promotes breast cancer proliferation and metastasis *in vitro* and *in vivo*. **a** Intracellular expression of ACTN4 was determined by Western blot (left) and real-time PCR (right) analysis, respectively; **b** ACTN4 expression was modified by transfecting recombinant plasmid or its shRNA in breast cancer cells and subjected to Western blotting (left) and real-time PCR (right) validation; **c** MTT and colony formation assay showed that ACTN4 silencing abrogated breast cancer cell proliferation while its overexpression promoted cell growth; **d** ACTN4 silencing inhibited the migration and invasion abilities of MDA-MB-231 cells; **e** ACTN4 silencing inhibited breast cancer growth and lung metastasis *in vivo* (*P < 0.05, *P < 0.01 versus control, values represented as the mean \pm SD, n = 3)