CORRECTION

Open Access

Correction to: Activation of LncRNA TINCR by H3K27 acetylation promotes Trastuzumab resistance and epithelialmesenchymal transition by targeting MicroRNA-125b in breast Cancer



Huaying Dong^{1*}, Jianguo Hu², Kejian Zou¹, Mulin Ye¹, Yuanwen Chen³, Chengyi Wu⁴, Xin Chen^{4*} and Mingli Han^{5*}

Correction to: Mol Cancer 18, 3 (2019) https://doi.org/10.1186/s12943-018-0931-9

Following the publication of the original paper [1], the authors realized that there are errors in Fig. 3d and Fig. 6e, which caused duplications. The errors occurred during the preparation of figures and went unnoticed during the review process and during preparation of final publication. The revised Figures are shown below. This correction does not alter any of the findings or conclusions of the study. The authors regret this error.

The specific changes of figures are listed as follows:

1. Figure 3d (Replacement for 48h of SKBR-3TR cells),

2. Figure 6e (Replacement for 0h (sh-TINCR#1+p-Snail-1) of SKBR-3TR cells)

(insert figures here)

Author details

¹Department of General Surgery, Hainan General Hospital, Hainan Medical University, No.19 Xiu Hua Road, Xiuying District, Haikou City 570311, Hainan Province, China. ²Department of Obstetrics and Gynecology, The Second Affiliated Hospital, Chongqing Medical University, Chongqing 400010, China. ³Department of General Surgery, Chongqing Renji Hospital, University of Chinese Academy of Science, Chongqing 400062, China. ⁴Department of General Surgery, The Frist Affiliated Hospital, Chongqing Medical University, Chongqing 400016, China. ⁵Department of Breast Surgery, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China.

Published online: 11 June 2021

Reference

 Dong H, Hu J, Zou K, Ye M, Chen Y, Wu C, et al. Activation of LncRNA TINCR by H3K27 acetylation promotes Trastuzumab resistance and epithelialmesenchymal transition by targeting MicroRNA-125b in breast Cancer. Mol Cancer. 2019;18:3. https://doi.org/10.1186/s12943-018-0931-9.

The original article can be found online at https://doi.org/10.1186/s12943-018-0931-9.

* Correspondence: dr_dhy@163.com; chenxin1192@126.com; minglihan@126.com

¹Department of General Surgery, Hainan General Hospital, Hainan Medical University, No.19 Xiu Hua Road, Xiuying District, Haikou City 570311, Hainan Province, China

⁴Department of General Surgery, The Frist Affiliated Hospital, Chongqing Medical University, Chongqing 400016, China

⁵Department of Breast Surgery, The First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, China

Full list of author information is available at the end of the article

© The Author(s). 2021 **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.

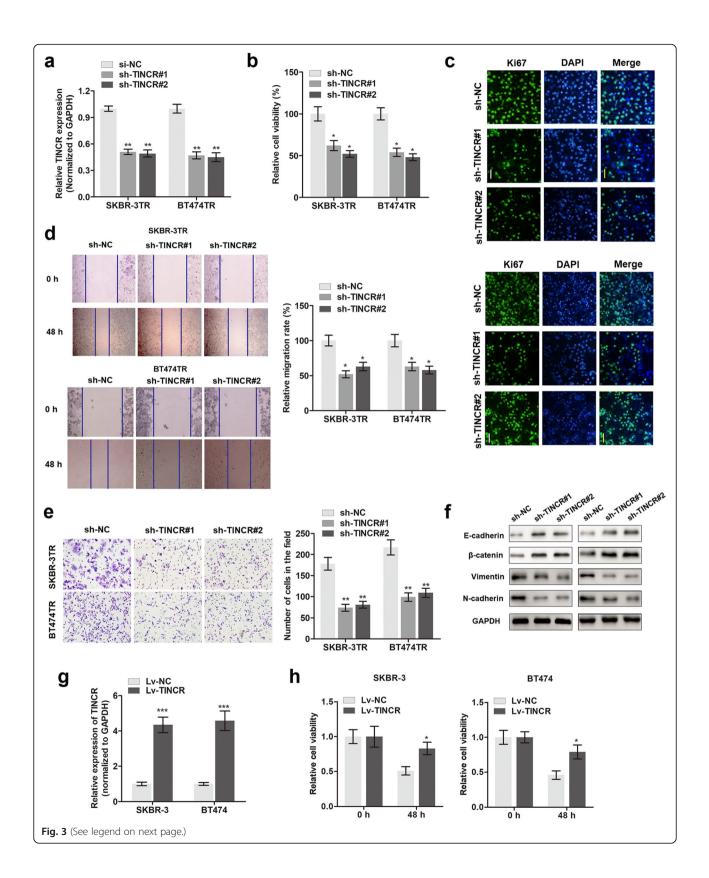
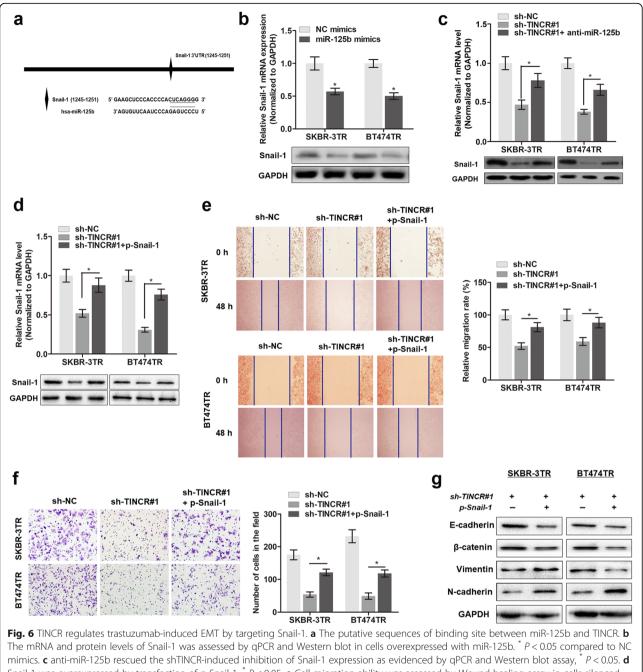


Fig. 3 Knockdown of TINCR abrogated trastuzumab resistance of breast cancer cells. **a** qPCR determination of the silencing effect of TINCR after infection with sh-TINCR#1 and sh-TINCR#2. ^{**}P < 0.01 compared to sh-NC group. **b** Cell viability was measured by MTT assay in cells silenced with TINCR. ^{*}P < 0.05 compared to sh-NC group. **c** In site Ki-67 expression was detected by performing immunofluorescence assay (Images were magnified at 20×). **d** Migration ability was assessed by using Wound-healing assay in cells silenced with TINCR. ^{*}P < 0.05 compared to sh-NC group. **e** Invasion ability was assessed by using Matrigel transwell assay. ^{**}P < 0.01 compared to sh-NC group. **f** The expression levels of Ecadherin, β -catenin, vimentin and N-cadherin were determined by Western blot assay. **g** TINCR expression was assessed via q-PCR in cells infected with Lv-TINCR. ^{**}P < 0.001 compared to Lv-NC group. **h** Cell viability was determined via MTT assay in breast cancer cells infected with Lv-TINCR. ^{**}P < 0.05 compared to Lv-NC group.



Snail-1 was overexpressed by transfection of p-Snail-1, * P < 0.05. **e** Cell migration ability was assessed by Wound-healing assay in cells silenced with TINCR or (and) overexpressed with Snail-1, * P < 0.05. **f** Cell invasion ability was assessed by Transwell assay in cells silenced with TINCR or (and) overexpressed with Snail-1, * P < 0.05. **f** Cell invasion ability was assessed by Transwell assay in cells silenced with TINCR or (and) overexpressed with Snail-1, * P < 0.05. **g** The expression levels of E-cadherin, β -catenin, vimentin and N-cadherin were determined by Western blot assay