# CORRECTION

**Open Access** 

# Correction: miR-29c plays a suppressive role in breast cancer by targeting the TIMP3/STAT1/ FOXO1 pathway

Wan Li<sup>1,2†</sup>, Jie Yi<sup>3†</sup>, Xiangjin Zheng<sup>1,2</sup>, Shiwei Liu<sup>4</sup>, Weiqi Fu<sup>1,2</sup>, Liwen Ren<sup>1,2</sup>, Li Li<sup>2</sup>, Dave S. B. Hoon<sup>5</sup>, Jinhua Wang<sup>1,2\*</sup> and Guanhua Du<sup>1,2\*</sup>

## Correction: Clinical Epigenetics (2018) 10:64

https://doi.org/10.1186/s13148-018-0495-y Following publication of the original article [1], the authors noticed the errors in Fig. 4b and Fig. S4A in the supplementary material. The revised Fig. 4 has been presented with this erratum and the revised supplementary material with the inclusion of new Fig. S4A has been uploaded.

The original article can be found online at https://doi.org/10.1186/s13148-018-0495-y.

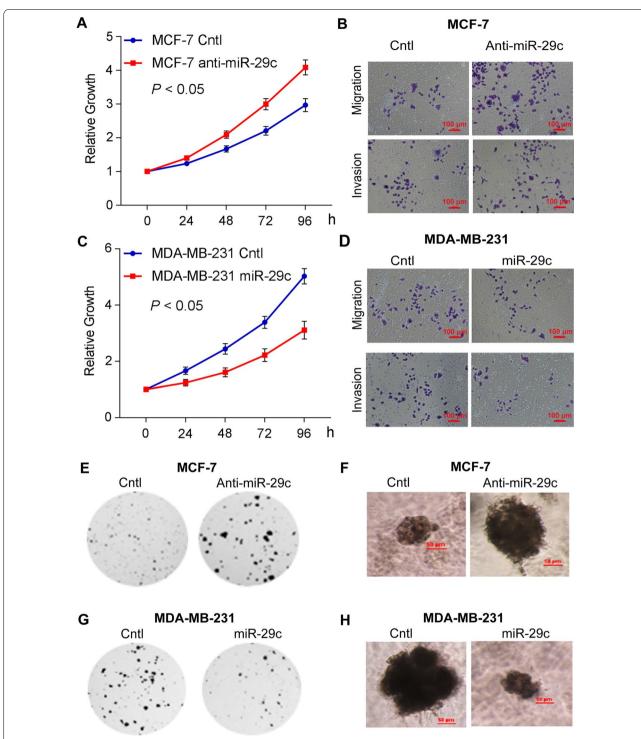
<sup>†</sup>Wan Li and Jie Yi contributed equally to this work.\*Correspondence: wjh@imm.ac.cn; dugh@imm.ac.cn

<sup>1</sup> The State Key Laboratory of Bioactive Substance and Function of Natural Medicines, Beijing, China

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



© The Author(s) 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 use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.gr/licenses/by/4.0/. The Creative Commons Public Domain Dedication waiver (http://creativecommons.gr/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated in a credit line to the data.



**Fig. 4** miR-29c inhibited the proliferation, migration and invasion, colony formation, and growth in 3D Matrigel of breast cancer cells. **a** Proliferation of MCF-7 anti-miR-29c is higher than that of MCF-7 Cntl by CCK8 proliferation assay. **b** Migration and invasion of MCF-7 anti-miR-29c is higher than that of MCF-7 Cntl. **c** Proliferation of MDA-MB-231 miR-29c mimic is lower than that MDA-MB-231 Cntl by CCK8 proliferation assay. **d** Migration and invasion assays of MDA-MB-231 miR-29c mimic are lower than that MDA-MB-231 Cntl. **e** Colony formations of MCF-7 anti-miR-29c are more than that of MCF-7Cntl in Soft agar assays. **f** Growth of MCF-7 anti-miR-29c is more than that of MCF-7 Cntl in 3D Matrigel culture. **g** Colony formations of MDA-MB-231 miR-29c mimic are less than that of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 Cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 miR-29c mimic is less than that of MDA-MB-231 cntl in soft agar assays. **h** Growth of MDA-MB-231 cntl in 3D Matrigel culture. Data are presented as mean ± SD from three independent experiments, and every experiment was repeated three times, \*P<0.05

### **Supplementary Information**

The online version contains supplementary material available at https://doi.org/10.1186/s13148-022-01317-4.

Additional file 1: Table S1. Sequence of DNMT3B siRNA. Table S2. Primers of miR-29c and DNMT3B. Table S3. Primers of TIMP3 for methylation specific PCR and unmethylation PCR. Figure S1. Quantification of protein expression level of DNMT3B in human breastcancer tissues and the paired adjacent non-tumor tissues. Figure S2. Migration and invasion of cells. Figure S3. miR-29c inhibited proliferation, migration and invasion, colony formation and growth in 3D Matrigel of MDA-MB-436 cells. Figure S4. DNMT3B promoted migration, invasion, colony formation and growth in 3D Matrigel of MDA-MB-436 cells. Figure S4. DNMT3B promoted migration, invasion, colony formation and growth in 3D Matrigel of MDA-MB-29C rells. Figure S5. Colony formation of cells. Figure S6. miR-29c reduced luciferase activity of wild type 3'UTR of DNMT3B-luciferase reporter, and not the mutant type 3'UTR of DNMT3B reporter in MCF-7 cells. Figure S7. Expression of DNMT3B, TIMP3, STAT1 and FOXO1. Figure S8. Migration and invasion of cells.

#### Author details

<sup>1</sup>The State Key Laboratory of Bioactive Substance and Function of Natural Medicines, Beijing, China. <sup>2</sup>Key Laboratory of Drug Target Research and Drug Screen, Institute of Materia Medica, Chinese Academy of Medical Science and Peking Union Medical College, Beijing 100050, China. <sup>3</sup>Department of Clinical Laboratory, Peking Union Medical College Hospital, Beijing 100730, China. <sup>4</sup>Department of Endocrinology, Shanxi DAYI Hospital, Shanxi Medical Cul University, Taiyuan 030002, Shanxi, China. <sup>5</sup>Department of Translational Molecular Medicine, John Wayne Cancer Institute (JWCI) at Providence Saint John's Health Center, Santa Monica, CA 90404, USA.

#### Published online: 29 July 2022

#### Reference

 Li W, et al. miR-29c plays a suppressive role in breastcancer by targeting the TIMP3/STAT1/FOXO1 pathway. Clin Epigenet. 2018;10:64. https://doi. org/10.1186/s13148-018-0495-y.

#### **Publisher's Note**

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.