

CORRECTION

Open Access



Correction: LncRNA APCDD1L-AS1 induces icotinib resistance by inhibition of EGFR autophagic degradation via the miR-1322/ miR-1972/ miR-324-3p-SIRT5 axis in lung adenocarcinoma

Jie Wu^{1,2†}, Chunlei Zheng^{3,4,5†}, Yizhe Wang¹, Zichang Yang^{3,4,5}, Ce Li^{3,4,5}, Wanxia Fang^{3,4,5}, Yue Jin^{3,4,5}, Kezuo Hou^{3,4,5}, Yang Cheng¹, Jianfei Qi⁶, Xiujuan Qu^{3,4,5}, Yunpeng Liu^{3,4,5}, Xiaofang Che^{3,4,5*} and Xuejun Hu^{1*}

Correction: Biomark Res 9, 9 (2021)
<https://doi.org/10.1186/s40364-021-00262-3>

In the original article [1], there was a mistake in Fig. 1e as published. The pictures of GAPDH of PC9 and HCC827 cells in Fig. 1e were misused by accident during

figure assembly. The corrected Fig. 1e appears below with new histograms.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way.

[†]Jie Wu and Chunlei Zheng contributed equally to this work.

The original article can be found online at <https://doi.org/10.1186/s40364-021-00262-3>.

*Correspondence:

Xiaofang Che
xfche@cmu.edu.cn
Xuejun Hu
xjhu@cmu.edu.cn

¹ Department of Respiratory and Infectious Disease of Geriatrics, The First Hospital of China Medical University, No.155 Nanjing North Street, Heping District, Shenyang 110001, Liaoning, China

² Department of Oncology, The First Affiliated Hospital of Jinzhou Medical University, Jinzhou 121000, Liaoning, China

³ Department of Medical Oncology, The First Hospital of China Medical University, No.155, North Nanjing Street, Heping District, Shenyang 110001, Liaoning, China

⁴ Key Laboratory of Anticancer Drugs and Biotherapy of Liaoning Province, The First Hospital of China Medical University, Shenyang 110001, Liaoning, China

⁵ Liaoning Province Clinical Research Center for Cancer, Shenyang 110001, Liaoning, China

⁶ Marlene and Stewart Greenebaum Comprehensive Cancer Center, University of Maryland, Baltimore, MD, USA

Published online: 12 May 2023

Reference

1. Jie Wu, et al. LncRNA APCDD1L-AS1 induces icotinib resistance by inhibition of EGFR autophagic degradation via the miR-1322/miR-1972/ miR-324-3p-SIRT5 axis in lung adenocarcinoma. *Biomark Res.* 2021;9:9. <https://doi.org/10.1186/s40364-021-00262-3>.



© The Author(s) 2023. **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.

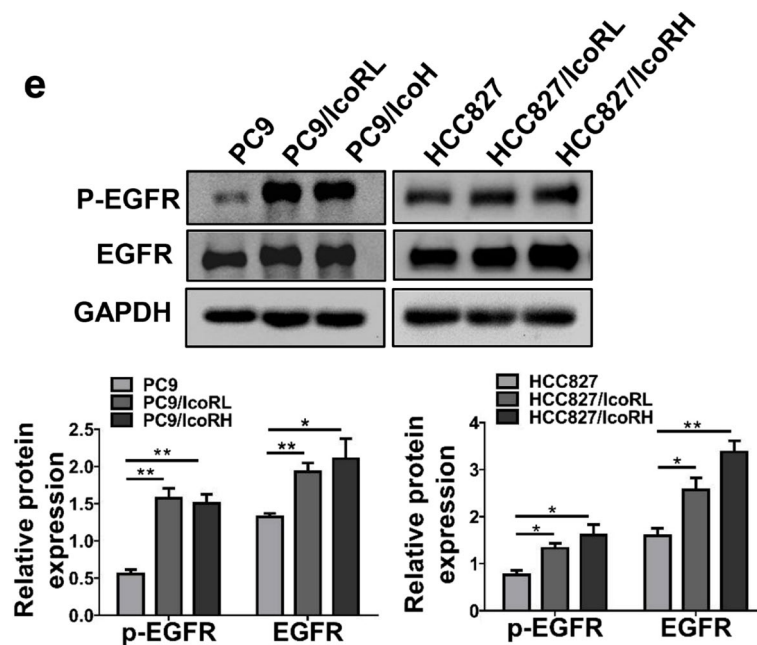


Fig. 1 Significant upregulation of APCDD1L-AS1 in icotinib-resistant LUAD cells. **a** The icotinib sensitivity in icotinib-resistant LUAD cells and their parental cells treated with different concentrations of icotinib for 96 h was determined by MTT assay. PC9/IcoRL: PC9 low-dose icotinib-resistant cells; PC9/IcoRH: PC9 high-dose icotinib-resistant cells; HCC827/IcoRL: HCC827 low-dose icotinib-resistant cells; HCC827/IcoRH: HCC827 high-dose icotinib-resistant cells. **b** The cell viability of both the parental cells and their icotinib-resistant cells after treated with icotinib (10 μ M) for 24, 48, 72 and 96 h was detected by MTT assay. **c** The colony formation ability of the parental cells and their icotinib-resistant cells under different concentrations of icotinib was analyzed using colony formation assay. **d** The subcutaneous tumor mouse models of icotinib-resistant cells and their parental cells were treated with or without icotinib. Average tumor volume for each group was measured ($n = 3$). **e** The level of EGFR expression and phosphorylation in the parental cells and their icotinib-resistant cells was evaluated by western blot. **f** Four upregulated lncRNAs identified by volcano plots in PC9/IcoRL cells and PC9/IcoRH cells comparing with PC9 cells. **g** The list of top four upregulated lncRNAs in PC9/IcoRL cells and PC9/IcoRH cells comparing with PC9 cells by transcriptome sequencing. **h** The expression level of lncRNAs, APCDD1L-AS1, PAX8-AS1, GASS and lnc-GSDMD, was determined in the parental cells and their icotinib-resistant cells by qRT-PCR. The mean \pm SD of triplicate experiments were plotted, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, n.s., not statistically significant