CORRECTION Open Access

Correction to: miR-3928v is induced by HBx via NF-kB/EGR1 and contributes to hepatocellular carcinoma malignancy by down-regulating VDAC3



Qiaoge Zhang^{1†}, Ge Song^{1†}, Lili Yao¹, Yankun Liu^{1,2}, Min Liu¹, Shengping Li³ and Hua Tang^{1*†}

Correction to: J Exp Clin Cancer Res 37, 14 (2018) https://doi.org/10.1186/s13046-018-0681-y

Following publication of the original article [1], the authors identified some errors in Figs. 4, 5 and 6; specifically panels Fig. 4d, Fig. 5e, and Fig. 6h. The corrections do not change the results and conclusions of this paper.

The correct figures are given below.

Author details

¹Tianjin Life Science Research Center and Department of Pathogen Biology, Collaborative Innovation Center of Tianjin for Medical Epigenetics, School of Basic Medical Sciences, Tianjin Medical University, No. 22 Qi-Xiang-Tai Road, Tianjin 300070, China. ²The Cancer Institute, Tangshan People's Hospital, Tangshan 063001, China. ³Department of Hepatobiliary Oncology, State Key Laboratory of Oncology in Southern China, Cancer Center, Sun Yat-sen University, Guangzhou 510060, China.

Published online: 21 August 2020

Reference

 Zhang, et al. miR-3928v is induced by HBx via NF-kB/EGR1 and contributes to hepatocellular carcinoma malignancy by down-regulating VDAC3. J Exp Clin Cancer Res. 2018;37:14.

The original article can be found online at https://doi.org/10.1186/s13046-018-0681-y.

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



© The Author(s). 2020 **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: htang2002@yahoo.com; tangh@tmu.edu.cn

[†]Qiaoge Zhang, Ge Song and Hua Tang contributed equally to this work.

¹Tianjin Life Science Research Center and Department of Pathogen Biology,
Collaborative Innovation Center of Tianjin for Medical Epigenetics, School of
Basic Medical Sciences, Tianjin Medical University, No. 22 Qi-Xiang-Tai Road,
Tianjin 300070. China

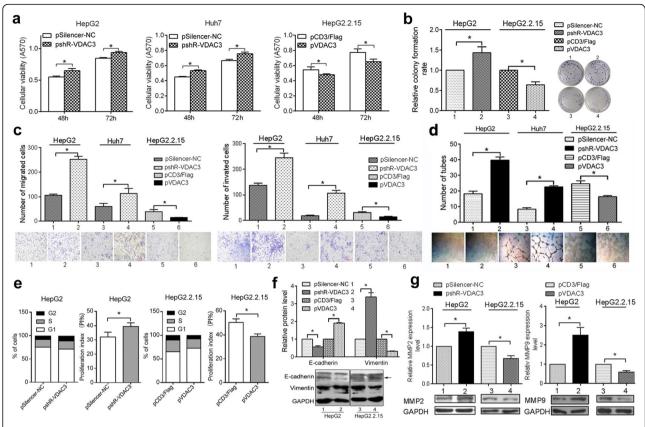


Fig. 4 VDAC3 acts as a tumor suppressor. Huh7 and HepG2 cells were transfected with pshR-VDAC3 or controls. HepG2.2.15 cells were transfected with pVDAC3 or controls. Twenty-four hours after transfection, cells were seeded into cell culture inserts. **a** MTT assays were performed to evaluate cell viability. **b** Colony formation assays were used to estimate cell proliferation. **c** Migration/invasion assays and **f** Western blot analyses were used to evaluate EMT. **d** VM and **g** molecular markers of VM were examined to assess tumor angiogenesis. **e** FACS assays were used to examine changes in cell cycle distribution. (*: *P* < 0.05)

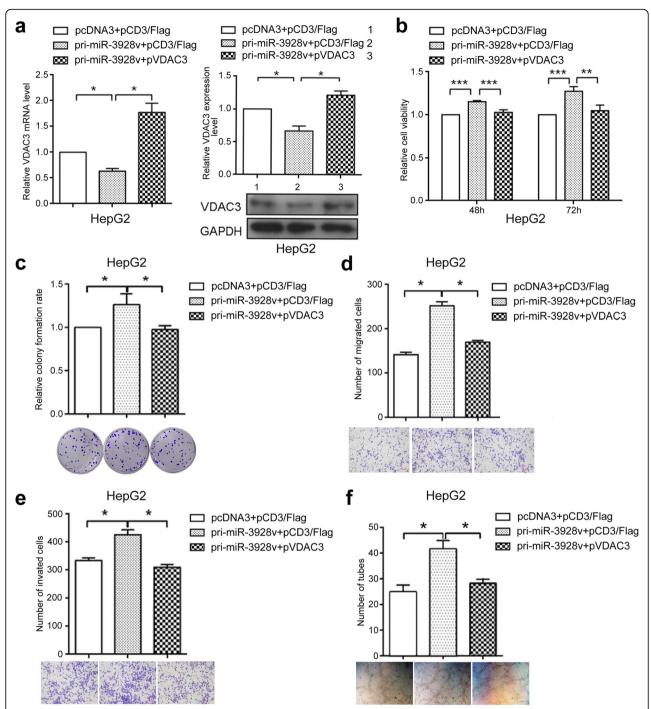


Fig. 5 VDAC3 is a functional target of miR-3928v. HepG2 cells were co-transfected with pCD3/Flag or pVDAC3 and pri-miR-3928v. RT-qPCR and Western blot analyses (**a**) and MTT (**b**), colony formation (**c**), migration (**d**), invasion (**e**) and tube formation assays (**f**) were performed to analyze whether VDAC3 could functionally rescue the miR-3928v-induced phenotype. (*: P < 0.05, **: P < 0.01, ***: P < 0.001)

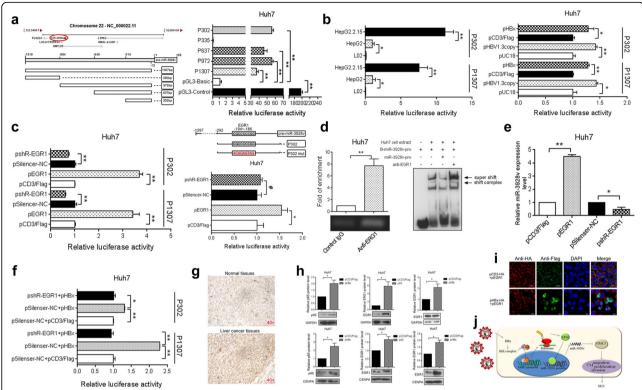


Fig. 6 HBx up-regulates miR-3928v expression via EGR1. **a** Schematic diagram of the location of miR-3928v in the human genome; the relative positions of the miR-3928v promoter constructs are shown (left). Luciferase reporter assays were used to examine miR-3928v promoter activity in Huh7 cells (right). **b** Promoter activity in HBV (+) and HBV (-) cells (left). Then, the promoter activity in Huh7 cells was examined after HBV or HBx overexpression (right). **c** The effect of EGR1 on promoter activity (left). The EGR1 binding site in the miR-3928v promoter was mutated, and the luciferase activity induced by EGR1 was examined (right). **d** ChIP analysis (left) and EMSAs (right) revealed that EGR1 directly binds to the miR-3928v promoter. **e** The miR-3928v expression level was measured by RT-qPCR after EGR1 overexpression or knockdown. **f** Blocking EGR1 inhibited HBx-induced miR-3928v promoter activity. **g** Representative IHC images showing the magnitude of EGR1 expression in tissues isolated from liver cancer patients. **h** Total and nuclear expression of EGR1 and P65 in Huh7 cells after transfection with HBx was analyzed by Western blot. **i** An immunofluorescence assay was used to detect the induction of EGR1 expression in the nucleus by HBx in Huh7 cells. **j** Mechanism by which HBx-induced miR-3928v expression contributes to HCC malignancy. (*: P < 0.05, **: P < 0.01)