frontiers | Frontiers in Pharmacology

TYPE Correction
PUBLISHED 17 May 2024
DOI 10.3389/fphar.2024.1392148

OPEN ACCESS

EDITED AND REVIEWED BY
Yidong Wang,
Xi'an Jiaotong University, China

*CORRESPONDENCE
Xianxi Tan,
drtxxwz@163.com
Li Lin,
linliwz@163.com
Ye Xiong,
xiongye2310@163.com

†These authors have contributed equally to this work and share first authorship

RECEIVED 14 March 2024
ACCEPTED 03 May 2024
PUBLISHED 17 May 2024

CITATION
Zhao Y, Ye S, Lin J, Liang F, Chen J, Hu J, Chen K, Fang Y, Chen X, Xiong Y, Lin L and Tan X (2024), Corrigendum: NmFGF1-regulated glucolipid metabolism and angiogenesis improves functional recovery in a mouse model of diabetic stroke and acts via the AMPK signaling pathway.
Front. Pharmacol. 15:1392148.
doi: 10.3389/fphar.2024.1392148

Corrigendum: NmFGF1-regulated glucolipid metabolism and angiogenesis improves functional recovery in a mouse model of diabetic stroke and acts via the AMPK signaling pathway

Yeli Zhao[1,2†], Shasha Ye[1,2†], Jingjing Lin[2], Fei Liang[2], Jun Chen[2], Jian Hu[2], Kun Chen[2], Yani Fang[2], Xiongjian Chen[2], Ye Xiong[1*], Li Lin[1,2,3*] and Xianxi Tan[1*]

[1]The First Affiliated Hospital of Wenzhou Medical University, Wenzhou, China, [2]School of Pharmaceutical Sciences, Wenzhou Medical University, Wenzhou, China, [3]Research Units of Clinical Translation of Cell Growth Factors and Diseases Research, Chinese Academy of Medical Science, Wenzhou, China

KEYWORDS

ischemic stroke, diabetes, nmFGF1, angiogenesis, AMPK

A Corrigendum on
NmFGF1-regulated glucolipid metabolism and angiogenesis improves functional recovery in a mouse model of diabetic stroke and acts via the AMPK signaling pathway

by Zhao Y, Ye S, Lin J, Liang F, Chen J, Hu J, Chen K, Fang Y, Chen X, Xiong Y, Lin L and Tan X (2021). Front. Pharmacol. 12:680351. doi: 10.3389/fphar.2021.680351

In the published article, there was an error in Figure 4 and Figure 5 as published. The representative images and quantified data in Figures 4B, C and Figures 5G, H have been presented incorrectly. Two pictures were taken from the same cell culture well with HG treatment in Figure 4B and HG + OGD + nmFGF1+A-769662 treatment in Figure 5G, respectively. Therefore, we reassembled and quantified all pictures in Figures 4B, C and Figures 5G, H. The corrected Figure 4 and Figure 5 and its captions appear below.

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

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

10.3389/fphar.2024.1392148

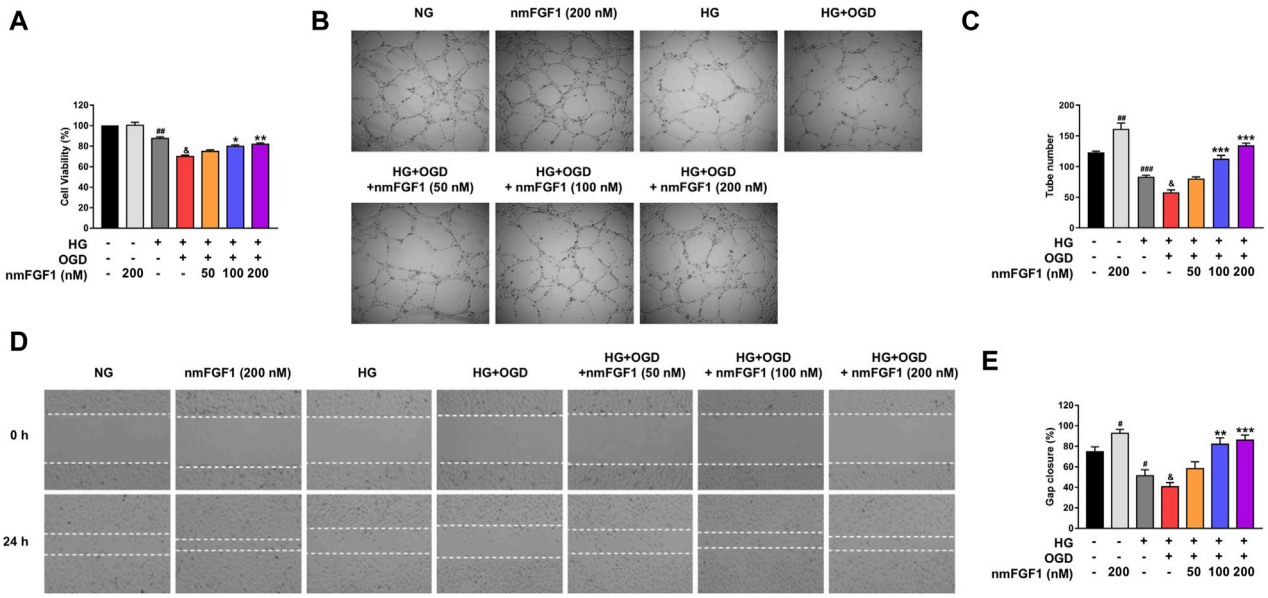

FIGURE 4
NmFGF1 reversed the reduction of tube formation and cell migration in HG + OGD-treated HBMEC cells. **(A)** The effect of nmFGF1 on the viability of HG + OGD-treated HBMEC cells. **(B)** The effects of nmFGF1 on tube formation, original magnification: ×40. **(C)** Quantitative analysis of the number of capillary-like tubes. **(D)** Wounding healing migration assay of HBMEC cells; images show wound areas as observed by phase-contrast microscopy, original magnification: ×40. **(E)** The migration ratio was calculated using Image Pro Plus software ($n = 3$). Date are presented as means ± SEM. $^{\#}p < 0.05$, $^{\#\#}p < 0.01$, $^{\#\#\#}p < 0.001$ vs. NG group; $^{\&}p < 0.05$ vs. HG group; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ vs. HG + OGD group.

10.3389/fphar.2024.1392148

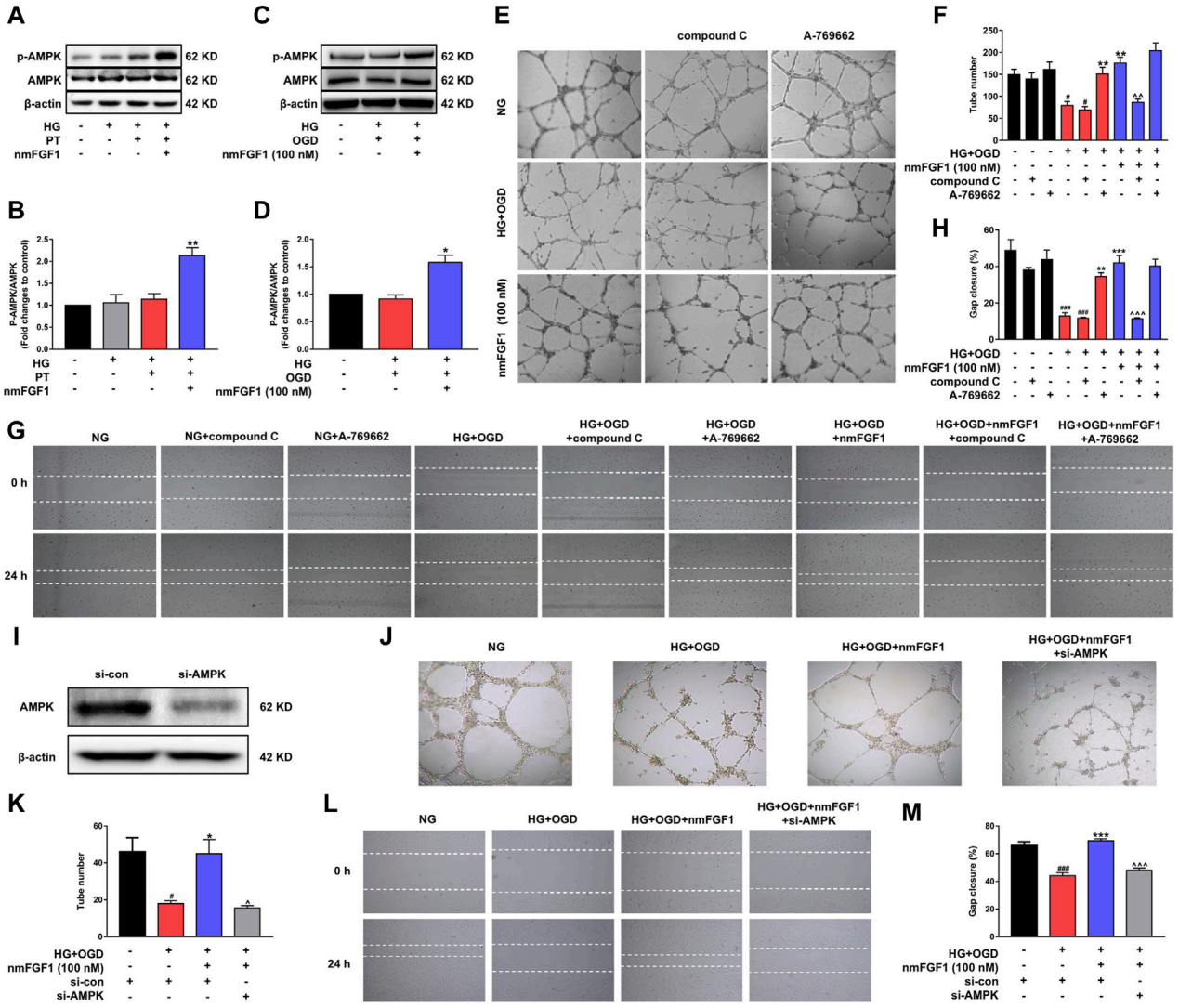

FIGURE 5
NmFGF1 promoted tube formation and migration of HBMEC in HG + OGD-treated HBMEC cells by activating the AMPK signaling pathway. **(A)** Western blotting analysis of *p*-AMPK and AMPK levels in a mouse model of diabetic stroke treated with or without nmFGF1. β-actin was used as a loading control. **(B)** The ratio of *p*-AMPK/AMPK. **(C)** The levels of *p*-AMPK and AMPK in HBMEC cells, as determined by western blotting. **(D)** The ratio of *p*-AMPK/AMPK in HBMECs, as quantified by image software. **(E)** Cells treated with AMPK agonist or inhibitor. AMPK agonist, A-769662, 10 nM; AMPK inhibitor, compound C, 5 μM. Tube formation was observed by phase contrast microscopy, original magnification: ×40. **(F)** The number of tubes formed in HBMECs treated with AMPK agonist or inhibitor. **(G)** The migration ability of HG + OGD-cultured HBMECs treated with an AMPK agonist or inhibitor. Images of the wound areas were observed by phase-contrast microscopy. A-769662, 10 nM, compound C, 5 μM; Original magnification: ×40. **(H)** The migration ratio was calculated using Image Pro Plus software. **(I)** AMPK expression was silenced by the transfection of siRNA. **(J)** The effect of AMPK silencing on tube formation. Original magnification: ×40. **(K)** Quantitative analysis of tube formation. **(L)** Wounding healing migration assay of cells in which AMPK had been silenced by siRNA. Original magnification: ×40. (M) Quantitative analysis of migration ratio ($n = 3$). Data are presented as means ± SEM. $^{\#}p < 0.05$, $^{\#\#}p < 0.01$, $^{\#\#\#}p < 0.001$ vs NG group; $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ vs HG + OGD or HG + PT group; $^{\wedge}p < 0.05$, $^{\wedge\wedge}p < 0.01$, $^{\wedge\wedge\wedge}p < 0.001$ vs HG + OGD + nmFGF1 group.