

## Erratum

# MiR-1205 functions as a tumor suppressor by disconnecting the synergy between KRAS and MDM4/E2F1 in non-small cell lung cancer: Am J Cancer Res. 2019; 9(2): 312-329

Hong Yan<sup>1,2</sup>, Xiaoying Chen<sup>1</sup>, Yu Li<sup>1,2</sup>, Lei Fan<sup>1,2</sup>, Yusi Tai<sup>1,2</sup>, Yang Zhou<sup>1,2</sup>, Yuxiang Chen<sup>1</sup>, Xinming Qi<sup>1,2</sup>, Ruimin Huang<sup>2,3</sup>, Jin Ren<sup>1,2</sup>

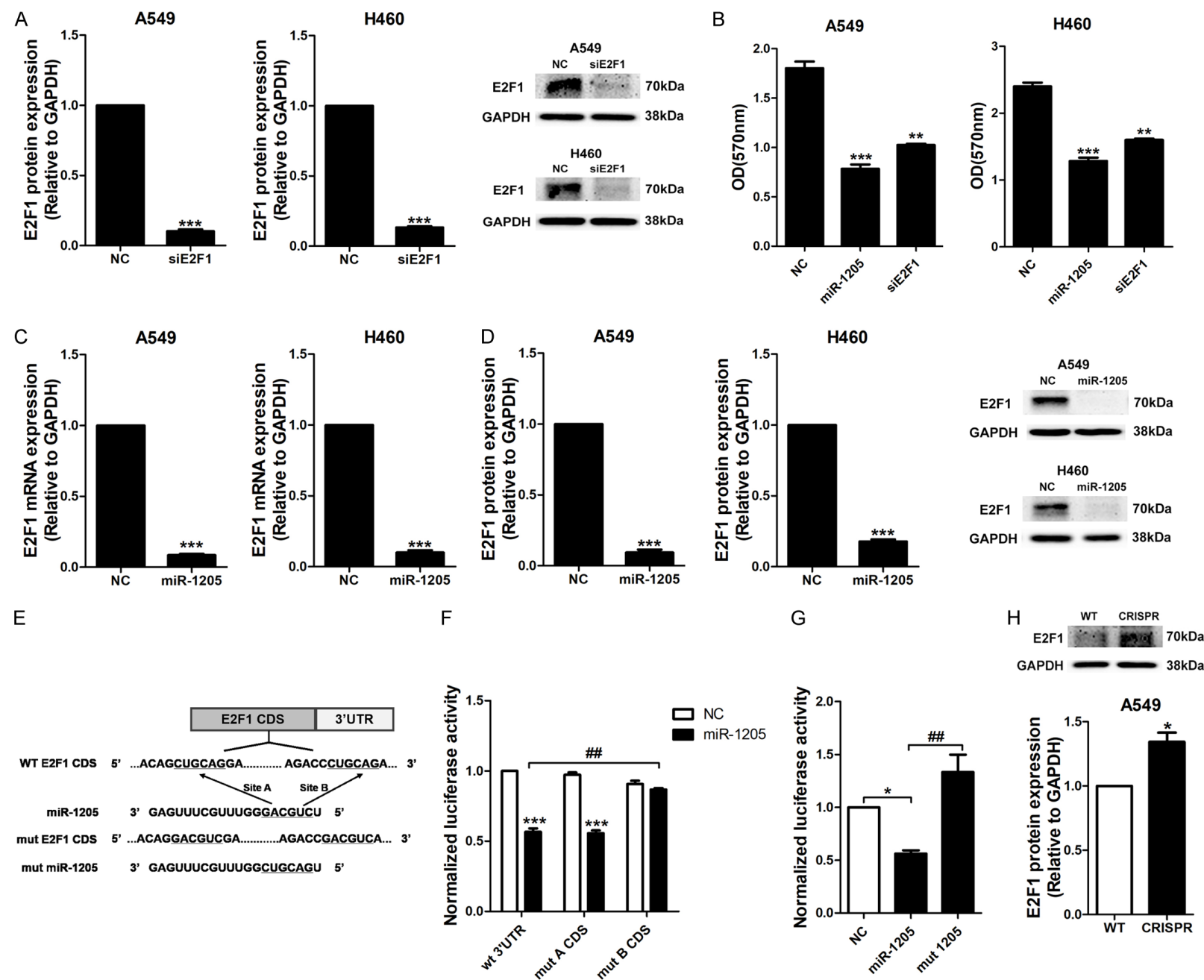
<sup>1</sup>Center for Drug Safety Evaluation and Research, State Key Laboratory of Drug Research, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Shanghai 201203, China; <sup>2</sup>University of Chinese Academy of Sciences, Beijing 100049, China; <sup>3</sup>Shanghai Institute of Materia Medica, Chinese Academy of Sciences, Shanghai 201203, China

Received May 29, 2025; Accepted June 3, 2025; Epub June 25, 2025; Published June 30, 2025

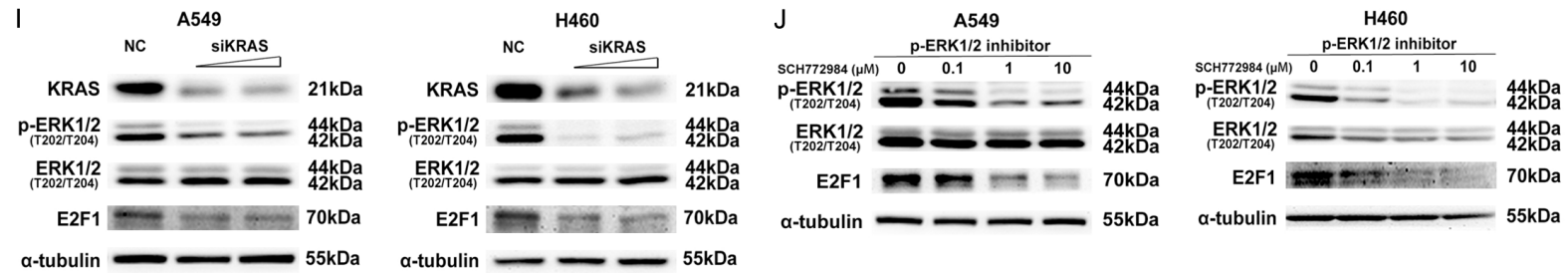
We identified an inadvertent error in **Figure 4J**, where the band for ERK1/2 (T202/T204) and the corresponding  $\alpha$ -tubulin loading control were mistakenly used from an image at a similar position from another experimental group due to the high similarity between the bands during figure preparation. This error does not affect the overall conclusion of the study, but we believe it is very important to correct it for accuracy and clarity. We sincerely apologize for this oversight and any confusion it may have caused. The corrected **Figure 4** is shown below.

**Address correspondence to:** Xinming Qi and Jin Ren, Center for Drug Safety Evaluation and Research, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, 501 Haik Road, Shanghai 201203, China. Tel: +86-18616608790; E-mail: xmqi@cdser.simm.ac.cn (XMQ); Tel: +86-1881770-1167; E-mail: jren@cdser.simm.ac.cn (JR); Ruimin Huang, Shanghai Institute of Materia Medica, Chinese Academy of Sciences, 555 Zuchongzhi Road, Shanghai 201203, China. Tel: +86-13512183680; E-mail: rmhuang@simm.ac.cn

Tumor suppressor role of miR-1205 via multiple targets



## Tumor suppressor role of miR-1205 via multiple targets



**Figure 4.** E2F1 expression was regulated by miR-1205 via direct CDS binding and indirect KRAS signaling inhibition. A. E2F1 protein levels in A549 and H460 cells after transfected with siE2F1 or NC for 72 h, 10 nM. B. MTT analysis of A549 and H460 cells transfected with miR-1205 mimics or siE2F1 or NC for 72 h, 10 nM. C and D. QRT-PCR and WB analysis of E2F1 mRNA and protein levels in A549 and H460 cells after transfected with miR-1205. E. Predicted binding between miR-1205 and matched sequence in the CDS of E2F1. Mutant sequence of miR-1205 (mut miR-1205) and E2F1 CDS (mut E2F1 CDS with mut A and mut B) were shown. F. Luciferase activity in cells transfected with miR-1205 and reporter plasmids containing wt or mut E2F1 CDS. G. Luciferase activity in cells transfected with miR-1205 or mut miR-1205 and reporter plasmids containing wt E2F1 CDS. H. E2F1 protein level in miR-1205 deleted A549 cells by CRISPR/CAS9. I. A549 or H460 cells were transfected with different dose of siKRAS (5, 10 nM) or NC for 72 h, KRAS signaling and E2F1 protein level were analyzed. J. A549 or H460 cells were treated with SCH772984 (0, 0.1, 1, 10 μM) for 24 h, and analyzed for the expression of p-ERK1/2 and E2F1. Data are presented as the mean ± SEM of three independent experiments (\* or #P<0.05, \*\* or ##P<0.01, \*\*\* or ###P<0.001).