Journal of
Translational Medicine

CORRECTION

Open Access

Correction: Epithelial cell adhesion molecule (EpCAM) regulates HGFR signaling to promote colon cancer progression and metastasis

Chi-Chiu Lee[1], Chia-Jui Yu[1], Sushree Shankar Panda[1], Kai-Chi Chen[1], Kang-Hao Liang[1,2], Wan-Chen Huang[1], Yu-Shiuan Wang[1], Pei-Chin Ho[2] and Han-Chung Wu[1,2*]

Correction: Journal of Translational Medicine (2023) 21:530
https://doi.org/10.1186/s12967-023-04390-2

Following publication of the original article [1], we have been notified about the errors in Fig. 7A, B, G. In Fig. 7A, B, the label of NMIgG should be revise to "–" in the EpAb2-6 and Crizotinib combine treatment group. In Fig. 7G, the red curve is NMIgG + Crzotinib and the blue curve is EpAb2-6 + Vehicle.

The incorrect version of Fig. 7 is as per below:

The original article can be found online at https://doi.org/10.1186/s12967-023-04390-2.

*Correspondence:
Han-Chung Wu
hcw0928@gate.sinica.edu.tw
[1] Institute of Cellular and Organismic Biology, Academia Sinica, 128 Academia Road, Section 2, Nankang, Taipei 11529, Taiwan
[2] Biomedical Translation Research Center (BioTReC), Academia Sinica, Taipei 11529, Taiwan

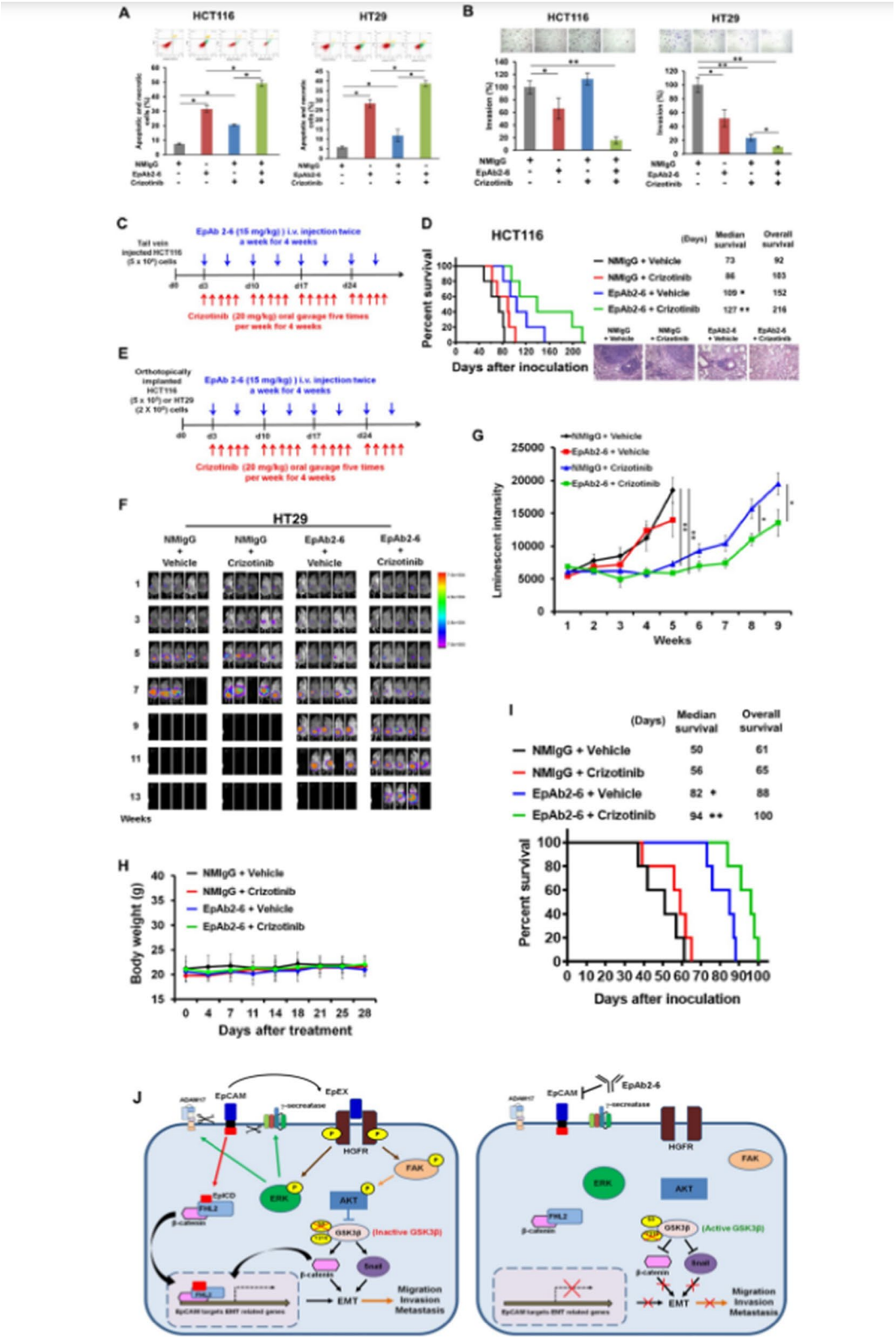
A
HCT116
HT29
B
HCT116
HT29
C
EpAb 2-6 (15 mg/kg)) i.v. injection twice a week for 4 weeks
Tail vein injected HCT116 (5 x 10⁶) cells
Crizotinib (20 mg/kg) oral gavage five times per week for 4 weeks
D
HCT116
Percent survival
Days after inoculation
NMIgG + Vehicle 73 92
NMIgG + Crizotinib 86 103
EpAb2-6 + Vehicle 109 * 152
EpAb2-6 + Crizotinib 127 ** 216
E
Orthotopically implanted HCT116 (5 x 10⁶) or HT29 (2 X 10⁶) cells
EpAb 2-6 (15 mg/kg)) i.v. injection twice a week for 4 weeks
Crizotinib (20 mg/kg) oral gavage five times per week for 4 weeks
F
HT29
NMIgG + Vehicle
NMIgG + Crizotinib
EpAb2-6 + Vehicle
EpAb2-6 + Crizotinib
Weeks
G
Lminescent intansity
Weeks
H
Body weight (g)
Days after treatment
I
(Days) Median survival Overall survival
NMIgG + Vehicle 50 61
NMIgG + Crizotinib 56 65
EpAb2-6 + Vehicle 82 * 88
EpAb2-6 + Crizotinib 94 ** 100
Percent survival
Days after inoculation
J
EpCAM
EpEX
HGFR
FAK
ERK
AKT
EpICD
FHL2
β-catenin
GSK3β
(Inactive GSK3β)
(Active GSK3β)
Snail
EMT
Migration
Invasion
Metastasis
EpAb2-6

Fig. 7 EpAb2-6 and crizotinib coordinately inhibit tumor progression and metastasis. **A** HCT116 and HT29 cells were treated with 10 μg/ml NMIgG or EpAb2-6 and 4 μM HGFR inhibitor crizotinib for 24 h. The apoptotic and necrotic cells were quantified by fluorescein annexin V-FITC/PI double labeling. **B** HCT116 and HT29 cells were treated with 10 μg/ml NMIgG or EpAb2-6 and 10 μM HGFR inhibitor crizotinib. Cell invasion was assessed by a Transwell assay with matrigel after 24 h. **C** Timeline of the experiment to evaluate EpAb2-6 and/or crizotinib effects in the metastatic animal model. **D** NOD/SCID mice were intravenously injected with 5×10^6 HCT116 cells, followed by treatment with either control IgG, EpAb2-6 and/ or crizotinib. The survival curve, median survival days and representative H&E staining of lung tissues in metastatic animal models are shown. **E** Timeline of the experiment to evaluate EpAb2-6 and/or crizotinib in the orthotopic animal model. **F** NOD/SCID mice received orthotopic implantation of HT29-Luc cells and then were treated with control IgG (normal mouse IgG, NMIgG), crizotinib, EpAb2-6, or crizotinib combined with EpAb2-6 starting at 3 days after tumor inoculation. Tumor growth was monitored by examining bioluminescence with the IVIS 200 Imaging System. **G** HT29-Luc tumor cells monitored by bioluminescence quantification. **H** Body-weights of each treatment group in the HT29 orthotopic animal model after indicated treatments. **I** Survival curves and median survival days of each treatment group in the HT29 orthotopic animal model. J Summary illustration of the cell signaling events mediating EpCAM tumorigenic effects. In brief, EpEX binds to HGFR then stimulates HGFR to induce ERK and FAK-AKT activation, which promotes active β-catenin and Snail protein stabilization via reducing GSK3β activity that drives tumor progression, migration, and invasion. The EpCAM neutralizing antibody EpAb2-6 inhibits cancer cell invasion by blocking EpEX-HGFR axis mediated downstream signaling to promote reduction of active β-catenin and Snail protein stability. Statistical differences were determined by two-tailed Student t test. N = 5 independent experiments. All data are presented as mean ± SEM. *$p < 0.01$

The correct version of Fig. 7 is as per below:

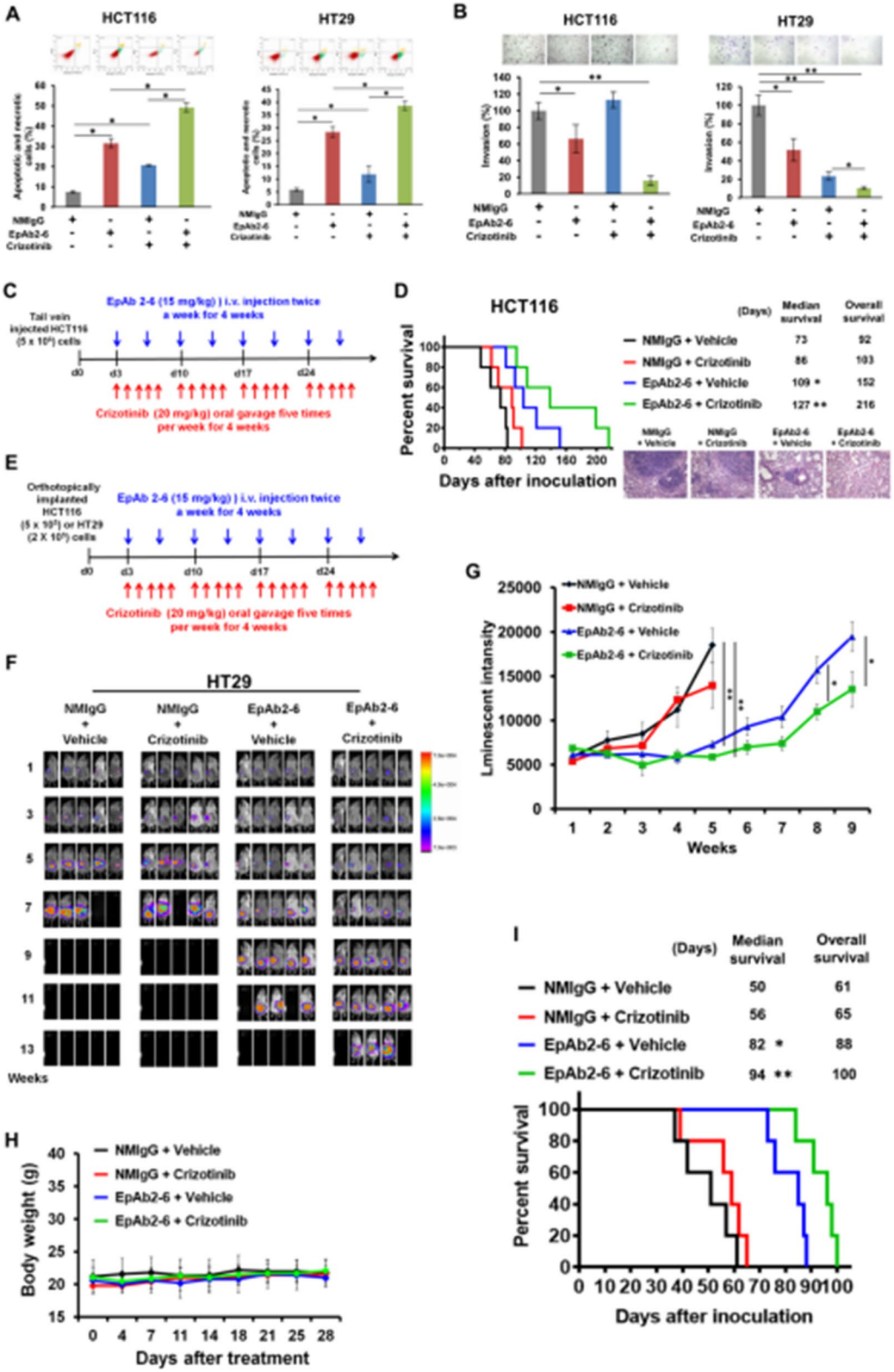
A
HCT116
HT29
Apoptotic and necrotic cells (%)
NMIgG
EpAb2-6
Crizotinib
B
HCT116
HT29
Invasion (%)
NMIgG
EpAb2-6
Crizotinib
C
Tail vein injected HCT116 (5 x 10⁵) cells
EpAb 2-6 (15 mg/kg)) i.v. injection twice a week for 4 weeks
d0 d3 d10 d17 d24
Crizotinib (20 mg/kg) oral gavage five times per week for 4 weeks
D
HCT116
Percent survival
Days after inoculation
(Days) Median survival Overall survival
NMIgG + Vehicle 73 92
NMIgG + Crizotinib 86 103
EpAb2-6 + Vehicle 109 * 152
EpAb2-6 + Crizotinib 127 ** 216
NMIgG + Vehicle
NMIgG + Crizotinib
EpAb2-6 + Vehicle
EpAb2-6 + Crizotinib
E
Orthotopically implanted HCT116 (5 x 10⁵) or HT29 (2 X 10⁶) cells
EpAb 2-6 (15 mg/kg)) i.v. injection twice a week for 4 weeks
d0 d3 d10 d17 d24
Crizotinib (20 mg/kg) oral gavage five times per week for 4 weeks
F
HT29
NMIgG + Vehicle
NMIgG + Crizotinib
EpAb2-6 + Vehicle
EpAb2-6 + Crizotinib
1 3 5 7 9 11 13
Weeks
G
NMIgG + Vehicle
NMIgG + Crizotinib
EpAb2-6 + Vehicle
EpAb2-6 + Crizotinib
Lminescent intansity
Weeks
H
NMIgG + Vehicle
NMIgG + Crizotinib
EpAb2-6 + Vehicle
EpAb2-6 + Crizotinib
Body weight (g)
Days after treatment
I
(Days) Median survival Overall survival
NMIgG + Vehicle 50 61
NMIgG + Crizotinib 56 65
EpAb2-6 + Vehicle 82 * 88
EpAb2-6 + Crizotinib 94 ** 100
Percent survival
Days after inoculation

Fig. 7. EpAb2-6 and crizotinib coordinately inhibit tumor progression and metastasis. **A** HCT116 and HT29 cells were treated with 10 μg/ml NMIgG or EpAb2-6 and 4 μM HGFR inhibitor crizotinib for 24 h. The apoptotic and necrotic cells were quantified by fluorescein annexin V-FITC/PI double labeling. **B** HCT116 and HT29 cells were treated with 10 μg/ml NMIgG or EpAb2-6 and 10 μM HGFR inhibitor crizotinib. Cell invasion was assessed by a Transwell assay with matrigel after 24 h. **C** Timeline of the experiment to evaluate EpAb2-6 and/or crizotinib effects in the metastatic animal model. **D** NOD/SCID mice were intravenously injected with 5×10^6HCT116 cells, followed by treatment with either control IgG, EpAb2-6 and/ or crizotinib. The survival curve, median survival days and representative H&E staining of lung tissues in metastatic animal models are shown. **E** Timeline of the experiment to evaluate EpAb2-6 and/or crizotinib in the orthotopic animal model. **F** NOD/SCID mice received orthotopic implantation of HT29-Luc cells and then were treated with control IgG (normal mouse IgG, NMIgG), crizotinib, EpAb2-6, or crizotinib combined with EpAb2-6 starting at 3 days after tumor inoculation. Tumor growth was monitored by examining bioluminescence with the IVIS 200 Imaging System. **G** HT29-Luc tumor cells monitored by bioluminescence quantification. **H** Body-weights of each treatment group in the HT29 orthotopic animal model after indicated treatments. **I** Survival curves and median survival days of each treatment group in the HT29 orthotopic animal model. **J** Summary illustration of the cell signaling events mediating EpCAM tumorigenic effects. In brief, EpEX binds to HGFR then stimulates HGFR to induce ERK and FAK-AKT activation, which promotes active β-catenin and Snail protein stabilization via reducing GSK3β activity that drives tumor progression, migration, and invasion. The EpCAM neutralizing antibody EpAb2-6 inhibits cancer cell invasion by blocking EpEX-HGFR axis mediated downstream signaling to promote reduction of active β-catenin and Snail protein stability. Statistical differences were determined by two-tailed Student t test. N = 5 independent experiments. All data are presented as mean ± SEM. *$p < 0.01$

We were also notified that there was incorrectly description in the text body of the article ('Material and methods' section).

It is now:

To observe the Wnt HGFR-EpEX interaction, the cells were fixed and co-stained with HGFR or EpEX antibodies as described earlier in the IFS with cell lines section.

It should be:

To observe the HGFR-EpEX interaction, the cells were fixed and co-stained with HGFR or EpEX antibodies as described earlier in the IFS with cell lines section.

The original article was updated.

Published online: 08 January 2024

Reference

1. Lee C-C, Yu C-J, Panda SS, Chen K-C, Liang K-H, Huang W-C, Wang Y-S, Ho P-C, Wu H-C. Epithelial cell adhesion molecule (EpCAM) regulates HGFR signaling to promote colon cancer progression and metastasis. J Transl Med. 2023;21:530. https://doi.org/10.1186/s12967-023-04390-2.

Publisher's Note