CORRIGENDUM

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MicroRNA-218 inhibits the proliferation and metastasis of esophageal squamous cell carcinoma cells by targeting BMI1

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Following the publication of the above article, an interested reader drew to the authors' attention that the 'Control' and 'miR-218 / BMI1' data panels for the Transwell invasion assay experiments shown in Figs. 4D and 5D on p. 100 and 101 respectively contained apparently overlapping data, albeit presented in different orientations, such that these data would have been derived from the same original source, even though they were intended to have shown the results from different experiments.

On re-examining their original data, the authors realized that they had inadvertently assembled the data from the Transwell assay experiments incorrectly in Figs. 2, 4 and 5. The authors elected to repeat these Transwell assay experiments in view of the errors made in assembling these figures, and the revised versions of Figs. 2, 4 and 5 (specificially, containing the replacement Transwell assay data in Figs. 2F, 4D and 5D) are shown on the next three pages. Note that the errors made in assembling these figures did not affect the overall conclusions reported in the paper. All the authors agree with the publication of this corrigendum, and are grateful to the Editor of *International Journal of Molecular Medicine* for granting them the opportunity to publish this. Furthermore, they apologize to the readership for any inconvenience caused.

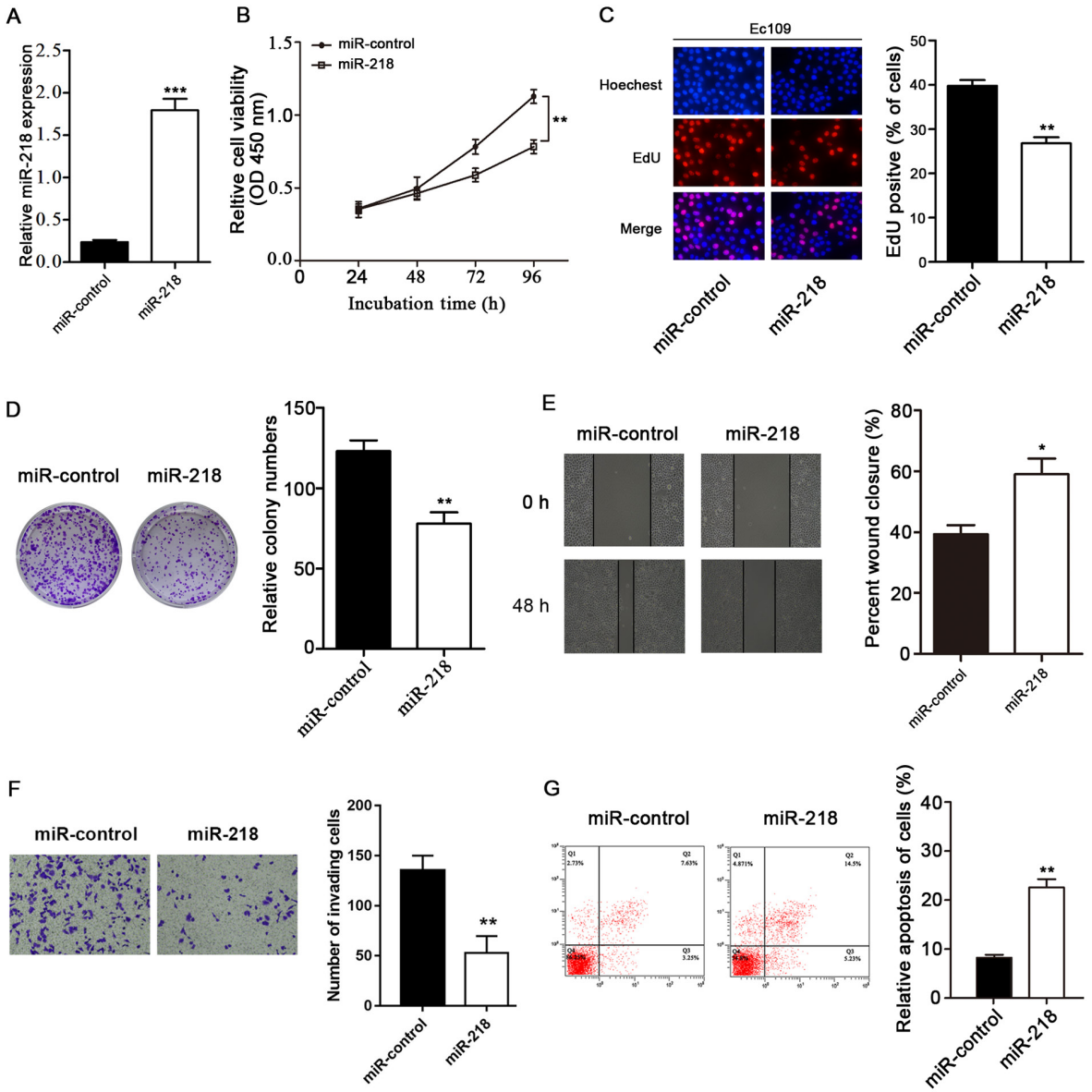

Figure 2. Inhibition of EC109 cell proliferation, migration and invasion by miR-218 and the increase in EC109 cell apoptosis induced by miR-218. (A) Expression of miR-218 measured by RT-qPCR in EC109 cells transfected with miR-218 mimics or miR-control. (B) MTT assay of relative EC109 cell viability at different time points (24, 48, 72 and 96 h) after transfection with miR-218 mimics or miR-control. (C) Effect of miR-218 on EC109 cell proliferation determined by EdU staining. Blue, Hoechst 33342 labeling of cell nuclei; red, EdU labeling of nuclei of proliferative cells. Quantitative data showed the percentage of EdU-positive cells (number of red vs. blue nuclei). (D) Effect of miR-218 overexpression on colony formation of EC109 cells. (E) Effect of miR-218 on migration of EC109 cells by wound healing assay. (F) Transwell invasion assay of EC109 cells treated with miR-218 mimics. (G) Effect of miR-218 on EC109 cell apoptosis determined by flow cytometry. Q2 + Q3 represent the total apoptotic rate. Data are presented as the means ± SD from 3 replicate samples. *P<0.05 and **P<0.01 vs. control.

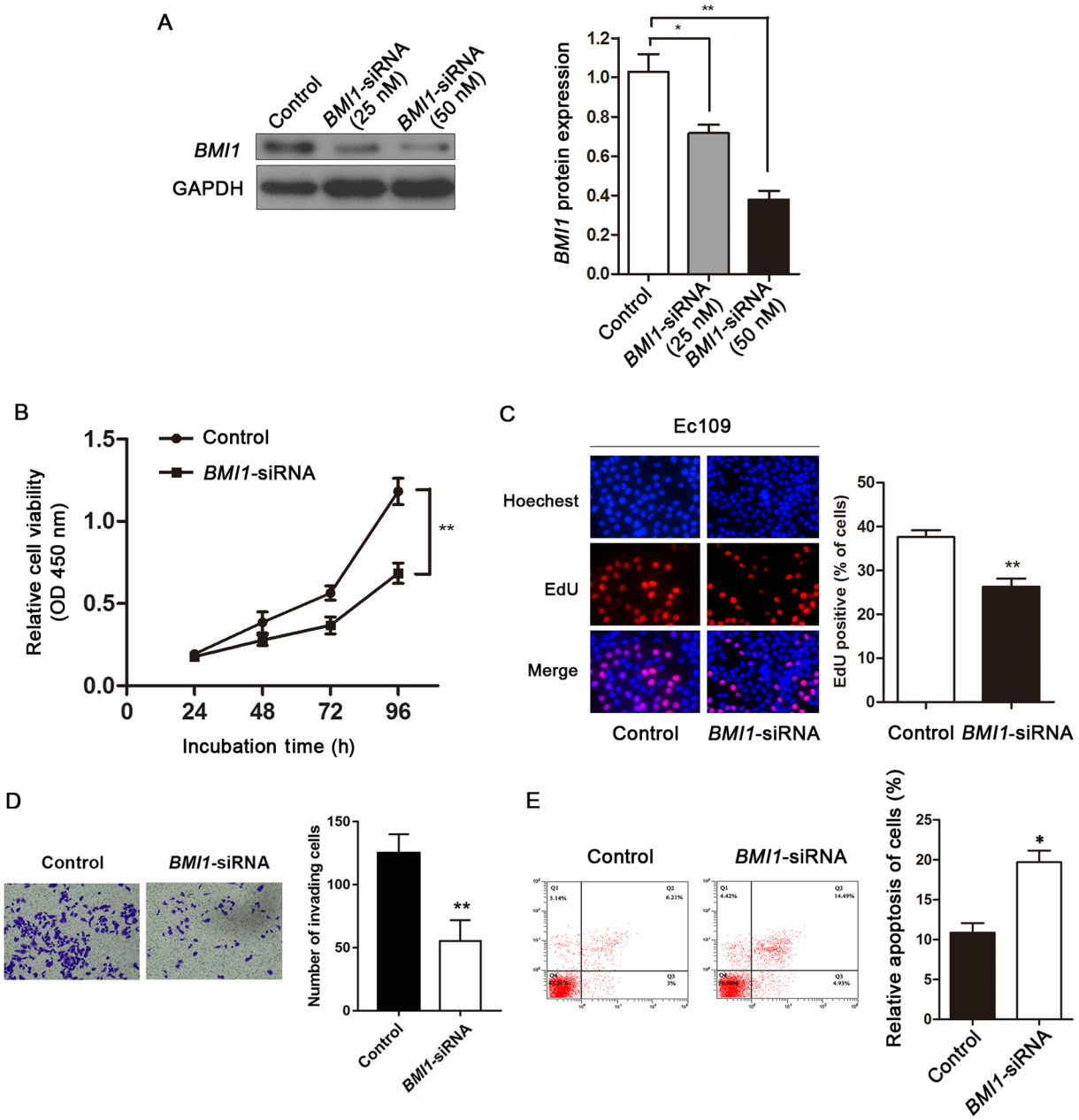

Figure 4. Effects of the downregulation of BMI1 expression on the phenotype of EC109 cells. (A) Determination of BMI1 protein levels by western blot analysis. EC109 cells were transfected with 25 nM BMI1-siRNA, 50 nM BMI1-siRNA or control oligo. GAPDH was used as a loading control. The relative values of BMI1 expression in the cells were calculated following normalization to GAPDH using ImageJ software. (B) MTT assay of EC109 cell growth at different time points (24, 48, 72 and 96 h) following transfection with BMI1-siRNA or control oligo. (C) Effect of BMI1 on EC109 cell proliferation determined by EdU staining. (D) Transwell invasion assay of EC109 cells transfected with BMI1-siRNA. (E) Effect of BMI1 on EC109 cell apoptosis determined by flow cytometry. Data are presented as the means ± SD from 3 replicate samples. *P<0.05 and **P<0.01 vs. control.

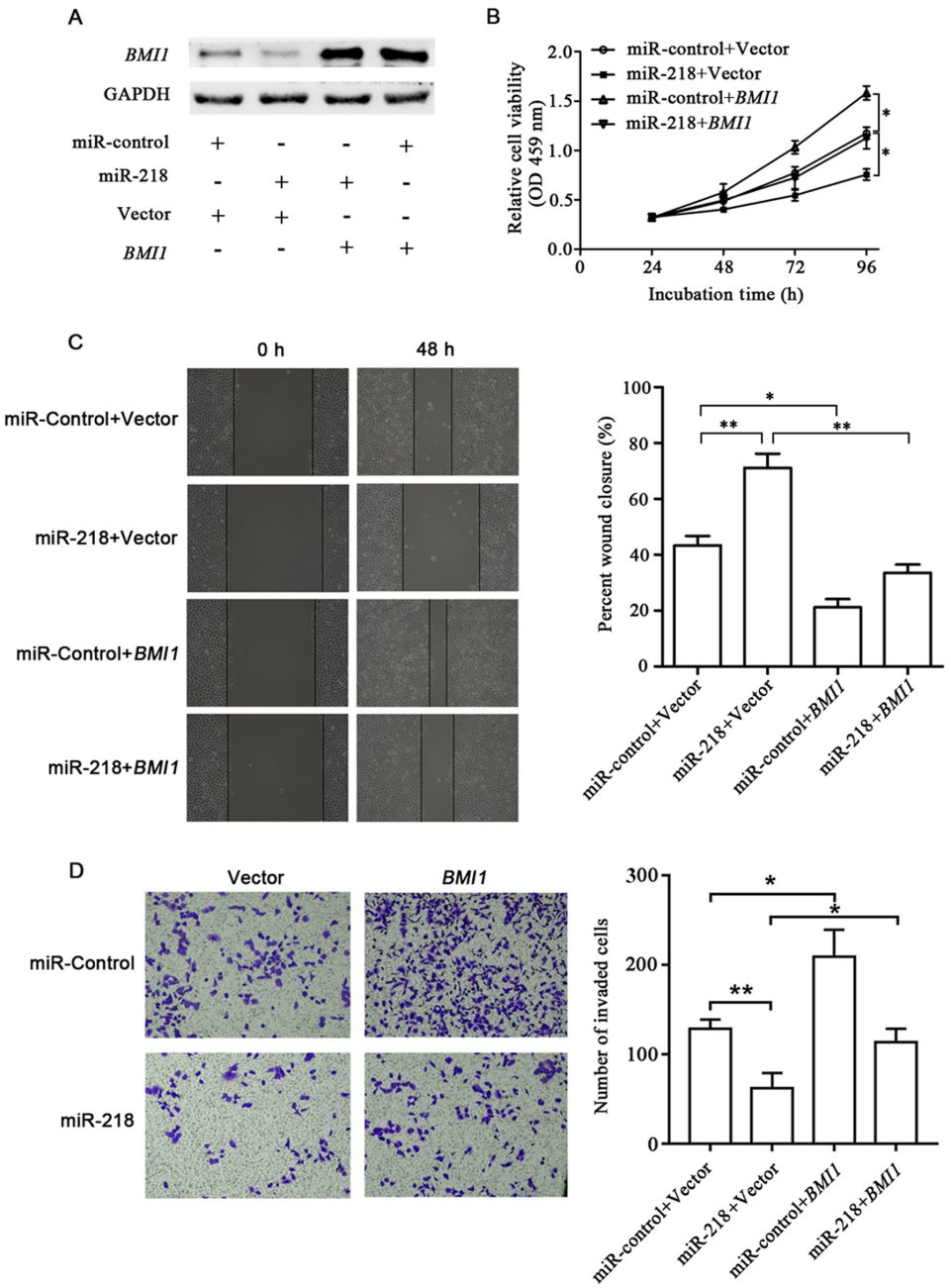

Figure 5. Partial reversal of the inhibitory effects of miR-218 on the ESCC cell phenotype by BMI1. (A) Western blot analysis of BMI1 protein levels in EC109 cells which were co-transfected with miR-218 mimics or miR-control and the BMI1 plasmid (without 3'-UTR) or vector control. (B) MTT assay of relative EC109 cell viability. (C) Wound healing assay. (D) Transwell invasion assay. Data are presented as the means ± SD from 3 replicate samples. $^{*}P<0.05$ and $^{**}P<0.01$.