Molecular Cancer

CORRECTION

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Correction: CMTM6 overexpression confers trastuzumab resistance in HER2-positive breast cancer

Fei Xing[1], Hongli Gao[1], Guanglei Chen[1], Lisha Sun[1], Jiayi Sun[1], Xinbo Qiao[1], Jinqi Xue[1] and Caigang Liu[1*]

Correction: Mol Cancer 22, 6 (2023)
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Following publication of the original article [1], the authors sincerely acknowledge that an incorrect image panel was inadvertently included in Figure 3F and 4H. To ensure the accuracy and integrity of the article, the authors have carefully reviewed the original data and replaced the erroneous images. This correction does not affect the main findings or conclusions of the study. The revised figures and their corresponding correct captions are provided below.

The original article can be found online at https://doi.org/10.1186/s12943-023-01716-y.

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Incorrect Fig. 3 and incorrect caption:

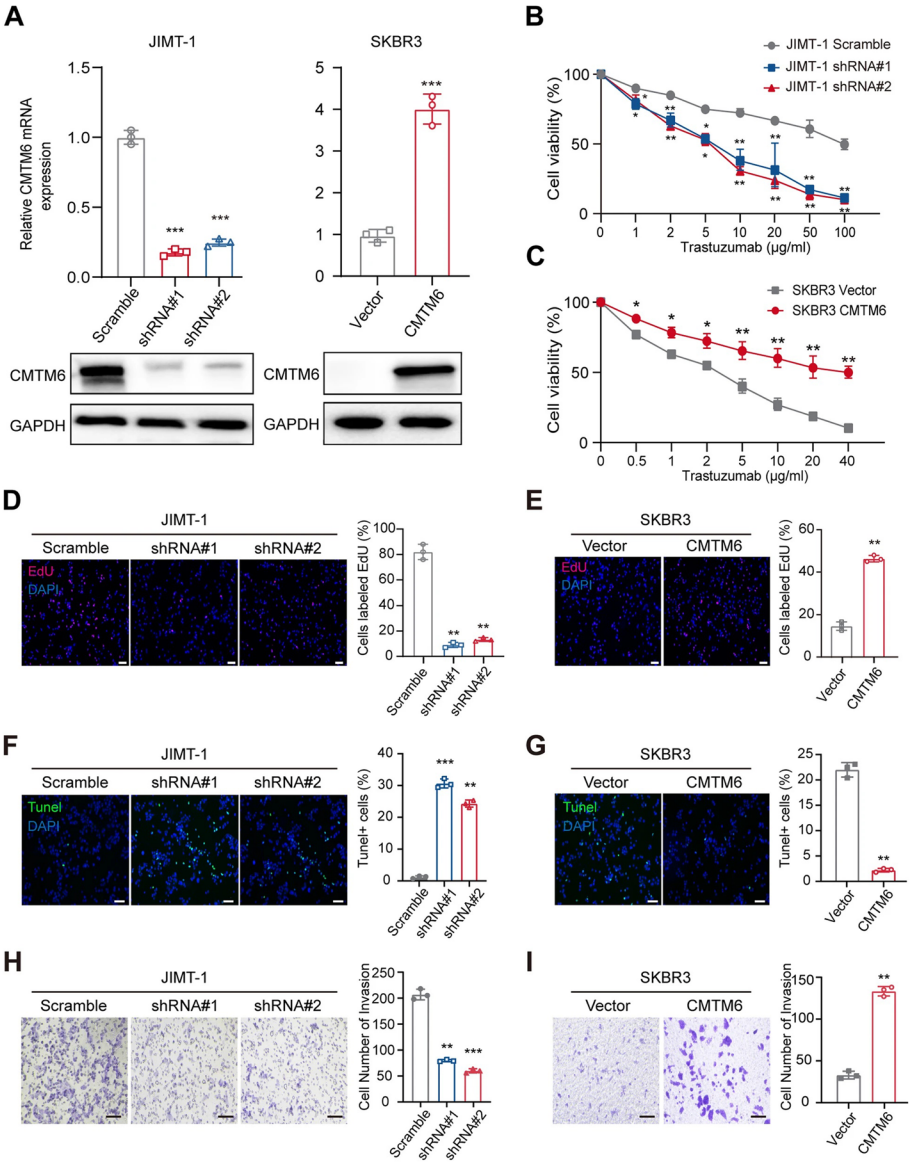

Fig. 3 CMTM6 promotes the survival, migration, invasion and trastuzumab resistance of BC cells in vitro. **A** qRT-PCR and Western blot validated CMTM6 silencing in JIMT-1 cells and CMTM6-overexpresstion in SKBR3 cells. Negative control (NC) JIMT-1 and SKBR3 cells were transduced with lentivirus for the control shRNA or transfected with the control plasmid, respectively. **B, C** CCK-8 assay determined the viability of the indicated BC cells following treatment with trastuzumab (0–100 μg/ml). **D, E** ethynyl-2′-deoxyuridine (EdU) analysis of the proliferation of CMTM6-silenced JIMT-1 cells, CMTM6 overexpressing SKBR3 cells, control JIMT-1 cells and SKBR3 cells following treatment with trastuzumab (10 μg/ml). (scale bar, 50 μM). **F, G** TUNEL analysis of apoptotic CMTM6-silenced JIMT-1 cells, CMTM6 overexpressing SKBR3 cells, control JIMT-1 and SKBR3 cells following treatment with trastuzumab (10 μg/ml). **H, I** Cell invasion assay revealed that CMTM6 silencing inhibited JIMT-1 cell invasion while CMTM6 overexpression enhanced SKBR3 cell invasion following treatment with trastuzumab (10 μg/ml). (scale bar, 50 μM). Data are representative images or expressed as the mean ± SD of each group from three independent experiments. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$

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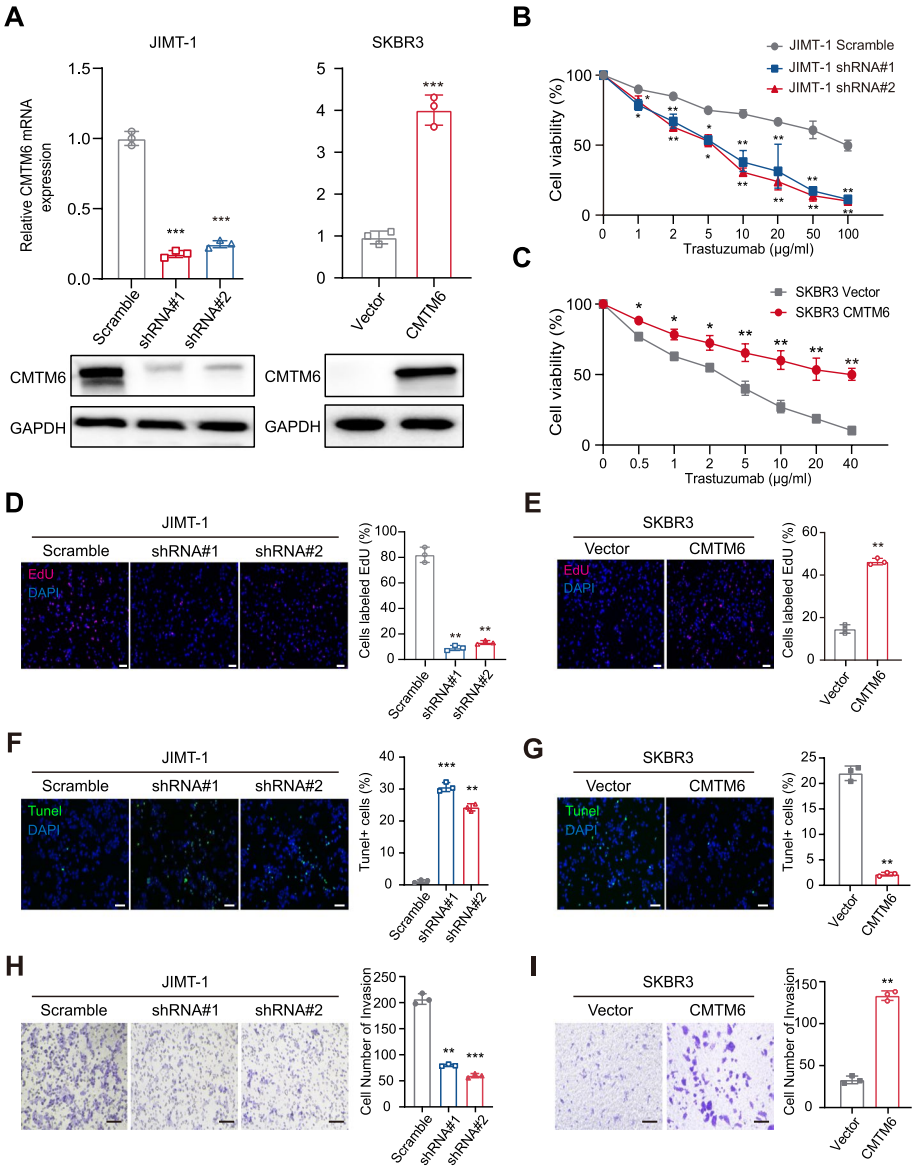

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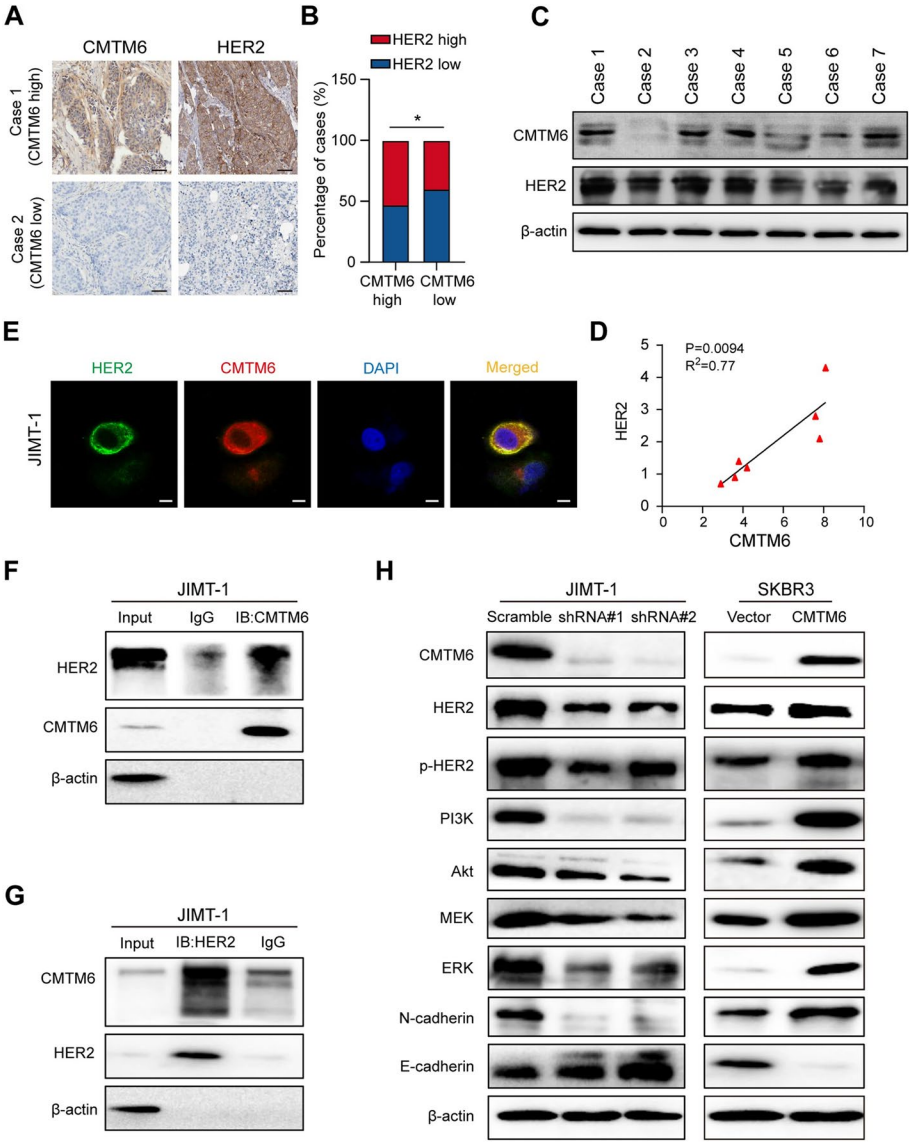

Fig. 4 CMTM6 directly interacts with HER2 and enhancing the HER2 signaling in BC cells. **A** IHC analysis of HER2 protein expression in BC tissues with low or high CMTM6 protein expression (scale bar, 50 μM). **B** Association between CMTM6 and HER2 protein expression in BC tissues. **C** Western blot analysis of CMTM6 and HER2 protein levels in BC tissues. **D** Correlation between CMTM6 and HER2 protein levels in BC tissues. Data are mean ± SEM. **E** Confocal microscopy analysis of the subcellular co-localization of CMTM6 (green) and HER2 (red) in JIMT-1 cells, with DAPI nuclear staining (blue) (scale bar, 10 μM). **F, G** Co-immunoprecipitation revealed the direct interaction between endogenous CMTM6 and HER2 proteins in JIMT-1 cells. **H** Western blot analysis of CMTM6, HER2, p-HER2, PI3K, AKT, MEK, ERK, N-cadherin and E-cadherin protein levels in CMTM6-silenced JIMT-1, CMTM6 overexpressing SKBR3, control JIMT-1 and SKBR3 cells. Data are representative images of each group of cells from three separate experiments

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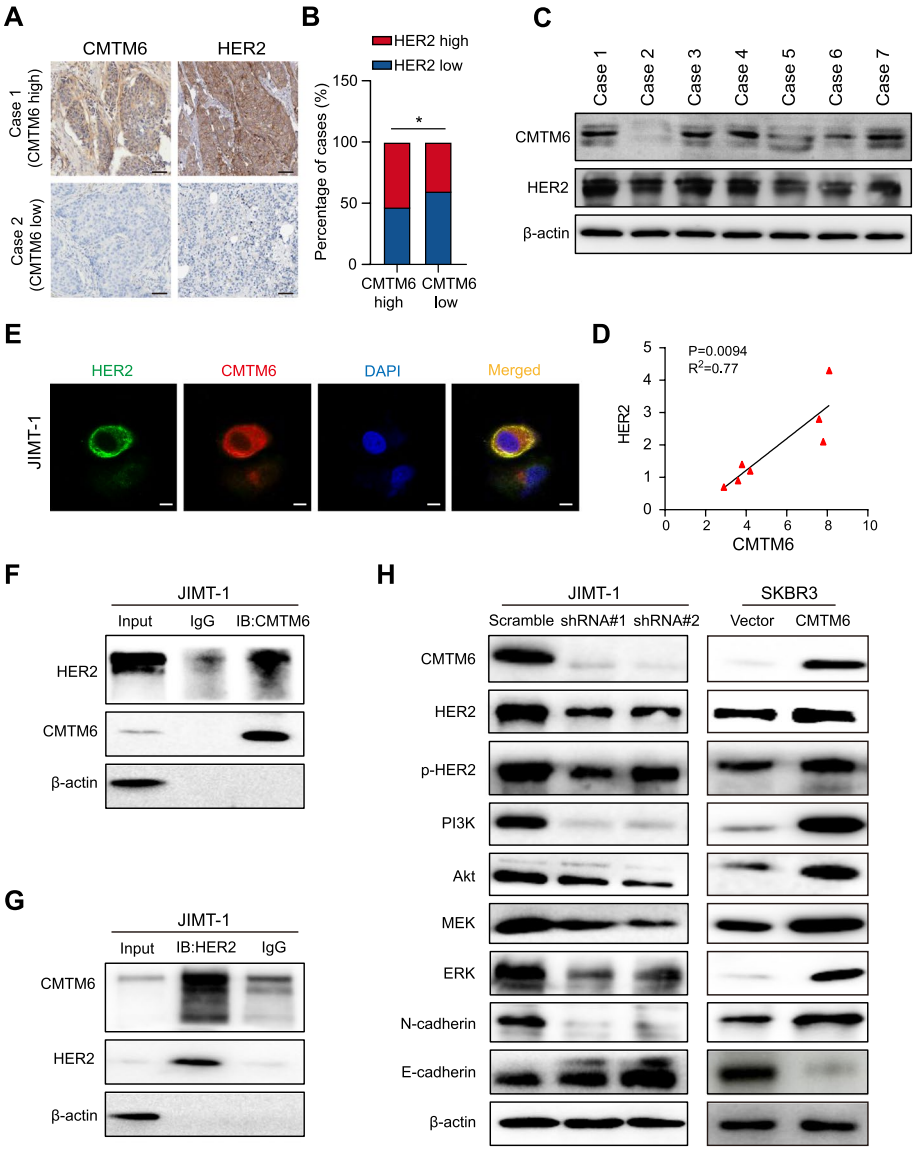

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Reference

1. Xing F, Gao H, Chen G, et al. CMTM6 overexpression confers trastuzumab resistance in HER2-positive breast cancer. Mol Cancer. 2023;22:6. https://doi.org/10.1186/s12943-023-01716-y.