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CORRECTION

Correction to: MYO1D binds with kinase domain of the EGFR family to anchor them to plasma membrane before their activation and contributes carcinogenesis

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Following the publication of this article, the authors noted three invasion images in Fig. 7 were repeated; the two images in Fig. 7a (EV, WT) and one image in Fig. 7b (TH) were repeated in Fig. 7c (EV, WT, TH). The corrected Fig. 7 is shown below. The authors would like to apologize for any inconvenience caused.

The original article has been corrected.

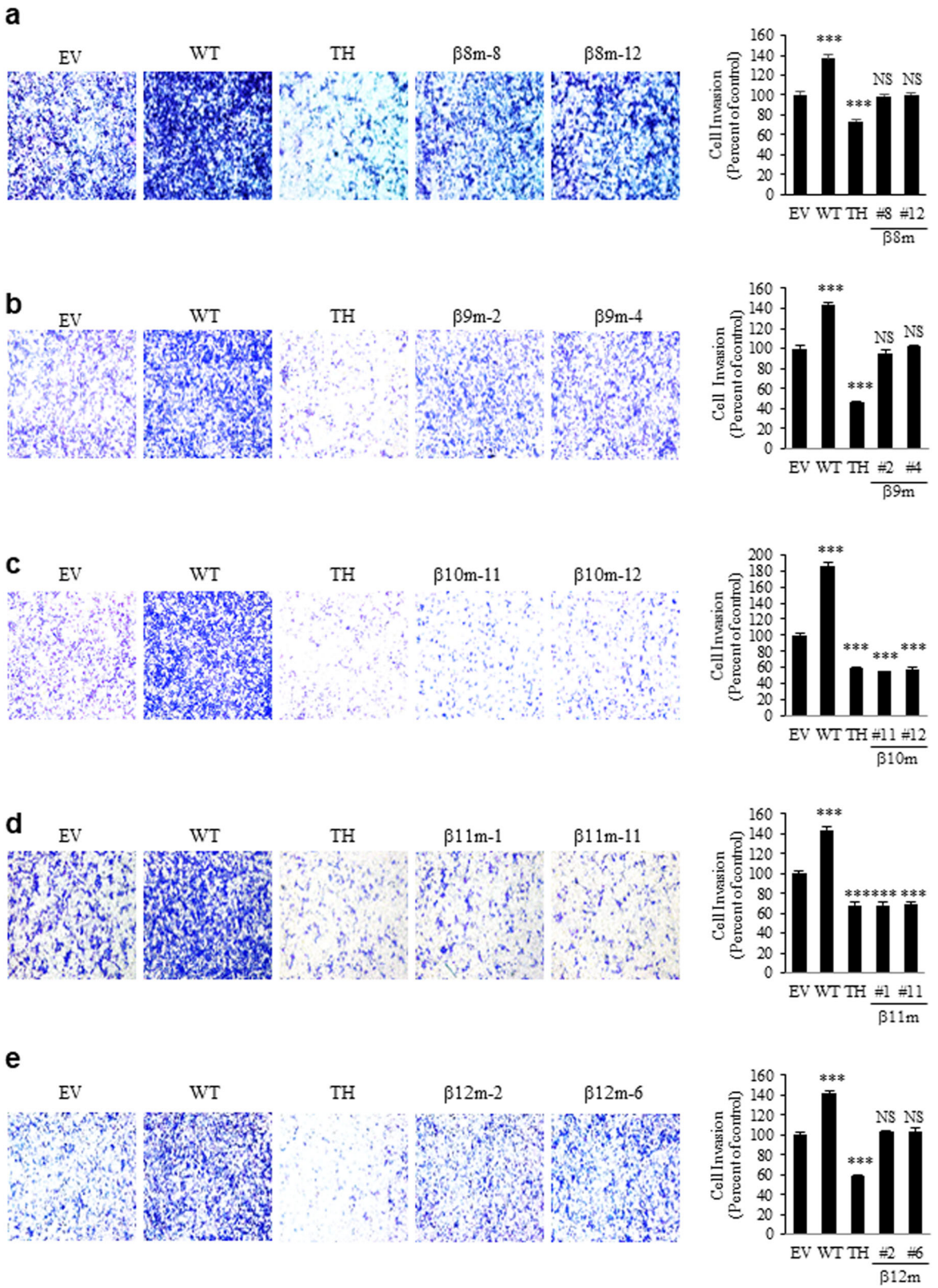

Fig. 7 Three β-sheets in the C-terminal end of MYO1D (except for β10 and β11) are essential to the RTK-binding site. Caco2 cells were co-transfected with empty vector, MYO1D WT, MYO1D TH1 domain (TH), or the mutated TH1 construct (β8m, β9m, β10m, β11m, or β12m) for 48 h and subjected to the transwell invasion assay. β8m was made by substitution the residues of 8th β-sheet at a time by alanine (Table S2), leaving the remaining β-sheets (β9, β10, β11, and β12) intact. The expression of each mutated TH1 construct was confirmed in lysates from the same cells used for the invasion assay (right side). The pictures and histogram of the invasion assay were obtained as in Fig. 1c.