

ERRATUM

Erratum to: CXCL13–CXCR5 co-expression regulates epithelial to mesenchymal transition of breast cancer cells during lymph node metastasis

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In the original publication of the article, the Fig. 6C-i and ii were published incorrectly. The correct immunoblot images of Vimentin (for ROE+LT MDA-MB-231) and N-Cadherin (for ROE+LT T-47D) against Src and

PI3Kp110a inhibitors SU6656 and PI-103 are now given in Fig. 6C-i and ii with reanalyzed densitometry. These corrections do not change the interpretation of the results or the conclusions of this work.

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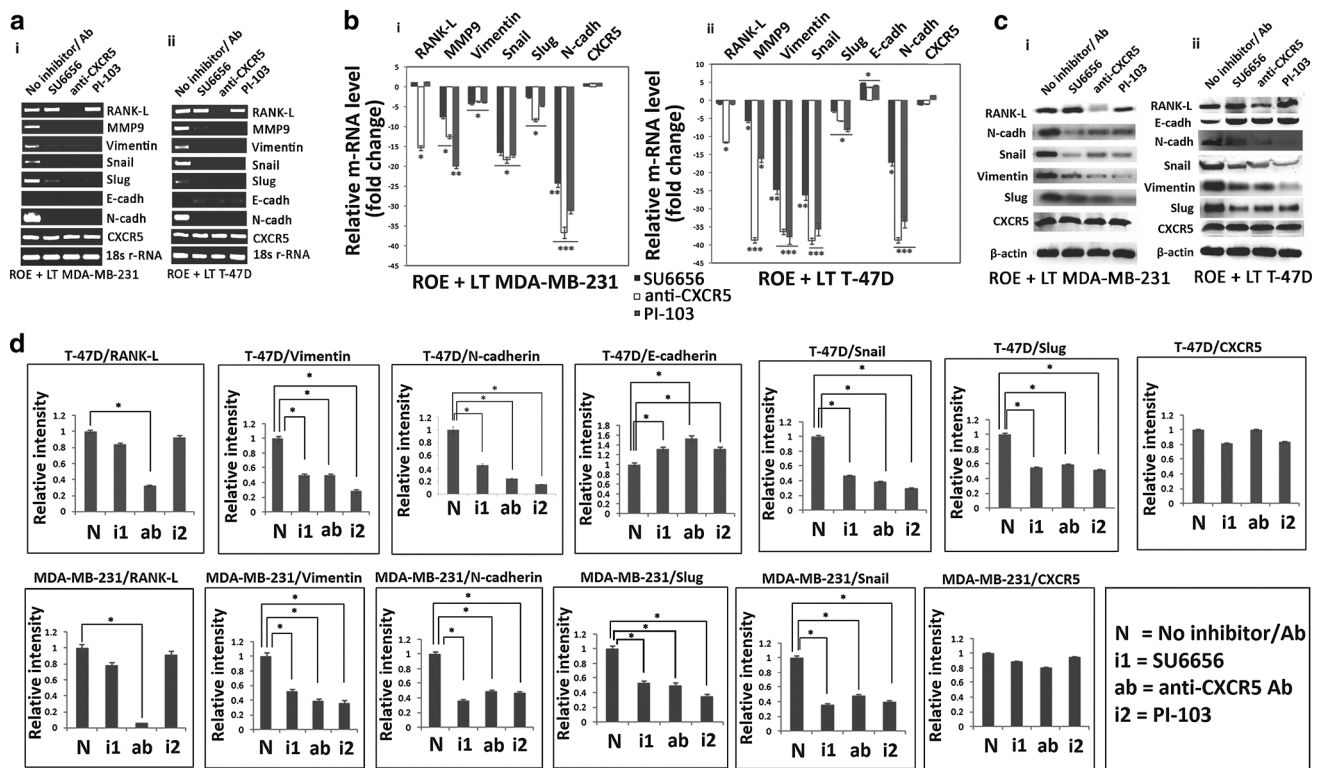


Fig. 6 Involvement of RANKL, Src, PI3Kp110 α in CXCL13-induced EMT and MMP9 expression. Prior to CXCL13 treatment, ROE + LT cells were treated with inhibitors of Src and PI3Kp110 α , i.e., SU6656 and PI-103, respectively, or with anti-CXCR5 monoclonal antibody. **a** mRNA levels of Vimentin, Slug, Snail, E-cadh, N-cadh, CXCR5, RANKL, and MMP9 were assessed by conventional RT-PCR, followed by agarose gel electrophoresis. 18s r-RNA was used as internal control. **b** Quantitative real-time RT-PCR was performed for these mRNAs. Fold changes are represented as relative values ($2^{-\Delta\Delta C_t}$) for SU6656, anti-CXCR5-antibody, PI-103-treated ROE + LT cells normalized with internal control and quantified. Fold changes in MDA-MB-231 were as follows: Vimentin decreased 4.4, 3.7, 4.0-fold, respectively; N-cadh decreased 22.5, 31.8, 36.3-fold, respectively; Snail decreased 16.1, 17.2, 16.7-fold, respectively; Slug decreased 2.2, 8.33, 3.9-fold, respectively; and MMP9 decreased 7.7, 12.5, 20.0-fold, respectively. For T-47D cells, fold changes were as follows: Vimentin decreased 24.9, 37.3, 38.5-fold, respectively; N-cadh decreased 17.3, 38.6, 34.2-fold, respectively; Snail decreased

26.0, 38.5, 35.1-fold, respectively; Slug decreased 3.1, 5.7, 8.2-fold, respectively; and MMP9 was decreased 5.9, 37.3, 16.5-fold, respectively. E-cadh expression increased significantly in all the treatment conditions for T-47D cells. RANKL expression, however, decreased significantly only upon anti-CXCR5 treatment. No treatment had any effect on CXCR5 mRNA level. **c** Expressions of Vimentin, Slug, Snail, N-cadh, E-cadh, RANKL, and CXCR5 were evaluated by immunoblotting. β -actin was used as loading control. Vimentin, Slug, Snail, and N-cadh expressions were decreased in SU6656, PI-103, anti-CXCR5-antibody treated ROE + LT cells. Inhibitor/anti-CXCR5-antibody treated ROE + LT T-47D cells have decreased E-cadh expression. RANKL expression was only found to be down-regulated in anti-CXCR5-antibody treated ROE + LT cells. **d** WB band densitometries shown in bar graphs. Results are representative of three independent experiments performed in triplicate and are represented as mean \pm SD. One-way ANOVA (Bonferroni correction) was performed, where significance level stands for $*p \leq 0.05$, $**p \leq 0.01$, $***p \leq 0.001$