

RANKL/RANK Pathway Abrogates Cetuximab Sensitivity in Gastric Cancer Cells via Activation of EGFR and c-Src [Corrigendum]

Zhang X, Song Y, Song N, et al. *Onco Targets Ther.* 2017;10:73–83.

In a detailed re-analyses of the data post-publication, the authors found the incorrect images were used for Figure 5A on page 80. Due to an error at the time of figure assembly the images from 5B was used as a placeholder and not replaced later. The correct Figure 5 is shown below.

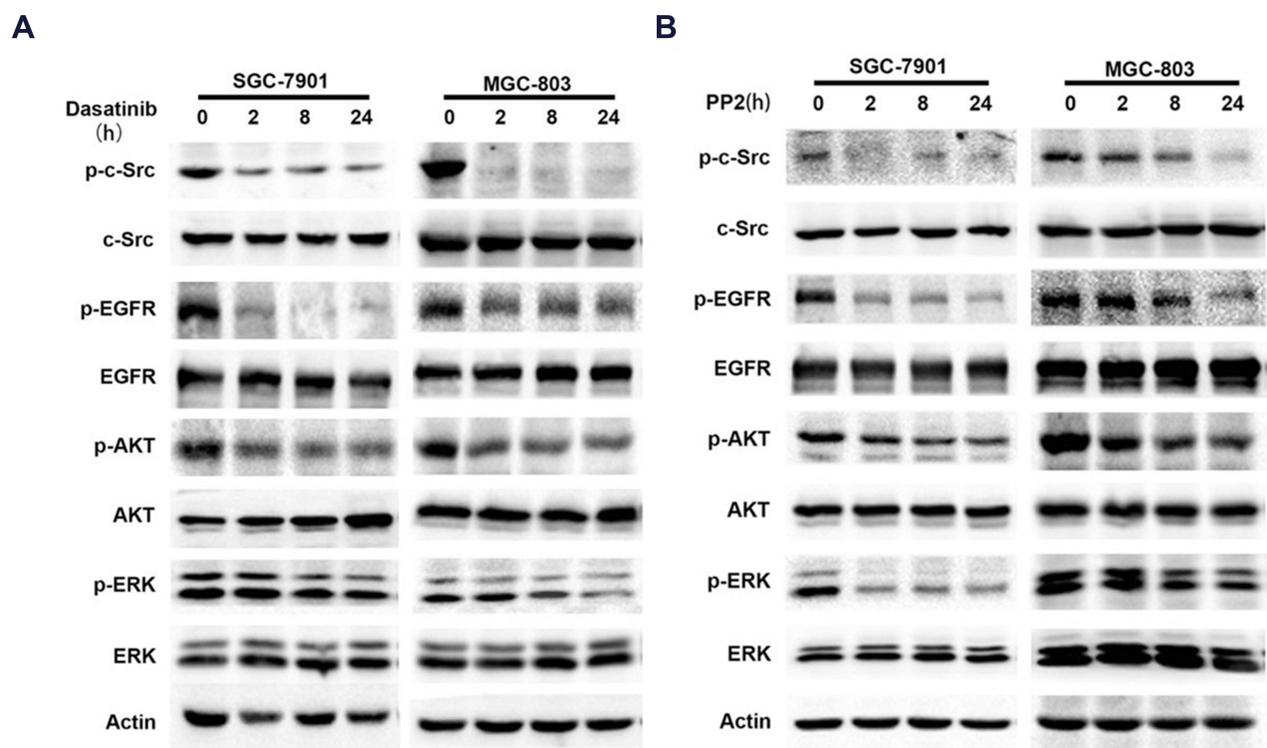


Figure 5 Continued.

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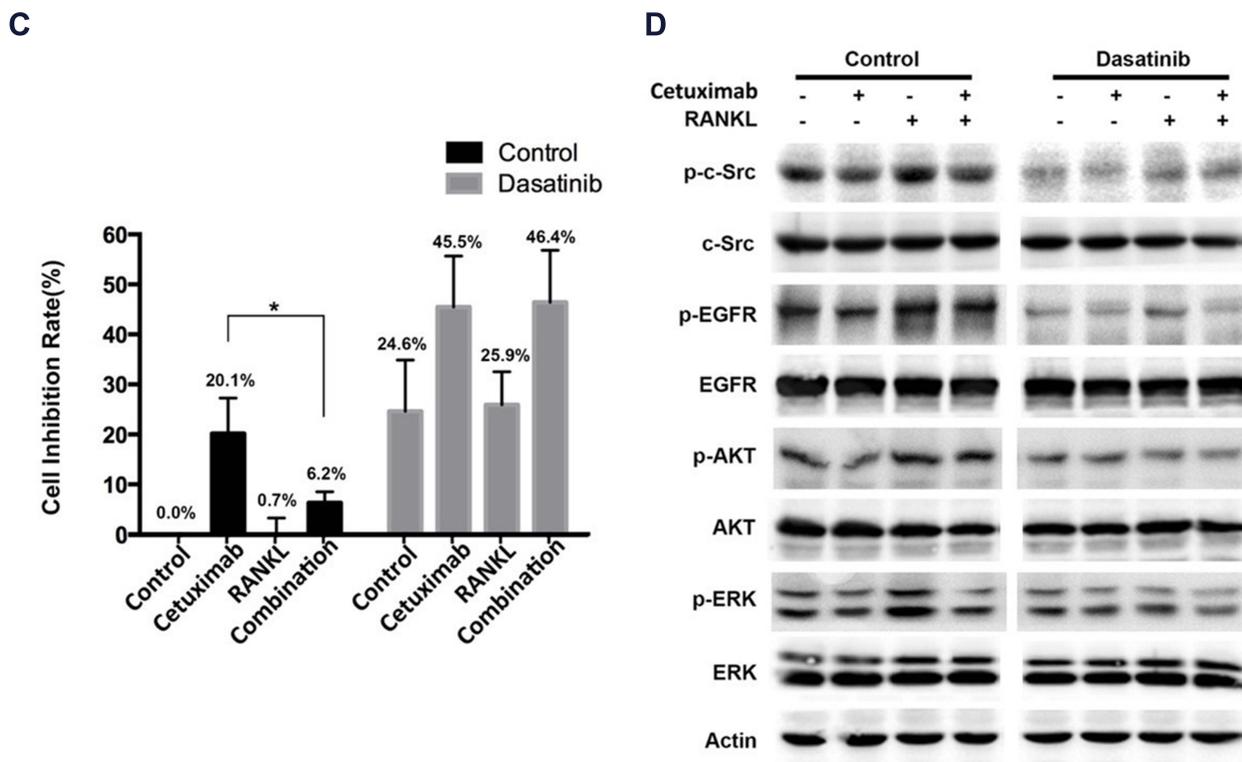


Figure 5 The effect of dasatinib on RANKL-induced cetuximab resistance in SGC-7901 cells.

Notes: (A and B) SGC-7901 and MGC-803 cells were treated with 100 nM dasatinib or 10 μ M PP2 for the indicated times. Expression of c-Src, EGFR, AKT, and ERK and phosphorylation levels were analyzed by Western blot. (C) SGC-7901 cells were treated with 100 nM dasatinib for 24 hours, followed by 1 μ g/mL RANKL for 1 hour and then 10 μ g/mL cetuximab for 48 hours. The cell viability was assessed by MTT assay. *Comparisons between the cetuximab-treated cells and the combined-treated cells in the control arm, $P < 0.05$. (D) SGC-7901 cells were treated with or without 100 nM dasatinib for 2 hours and then preincubated with 1 μ g/mL RANKL for 1 hour, followed by 10 μ g/mL cetuximab for 48 hours. The expression of c-Src, EGFR, AKT, and ERK and phosphorylation levels were analyzed by Western blot.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way.

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