

Erratum to: Celestrol suppresses invasion of colon and pancreatic cancer cells through the downregulation of expression of CXCR4 chemokine receptor

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Published online: 11 January 2013
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Erratum to: J Mol Med. 2010 Dec;88(12):1243-53
DOI 10.1007/s00109-010-0669-3

The authors claim that Figs. 5a and b, and 6a and d, reporting data from in vitro invasion assays were published incorrectly. Specifically, two images of invaded cells were by mistake duplicated. The authors performed 2 additional experiments to confirm their data. The results from these experiments are reported in the corrected versions of the figures and legends, as well as the corresponding results section that are shown below. The authors claim that this correction does not influence the conclusion of the study and would like to apologize for this oversight.

Results

CXCR4 is essential for CXCL12-induced invasion

Disruption of CXCR4 and CXCL12 interaction by selective antagonists or anti-CXCR4 antibody blocks cancer metastasis, suggesting an essential role for CXCR4. Therefore, when HCT116 cells were transfected with siRNA specific for CXCR4, it efficiently inhibited CXCL12-mediated invasion to about 31 % compared to the control condition. (Fig. 5a, b). Indeed, the CXCR4-specific siRNA reduced CXCR4 protein expression (Fig. 5c).

The online version of the original article can be found at <http://dx.doi.org/10.1007/s00109-010-0669-3>.

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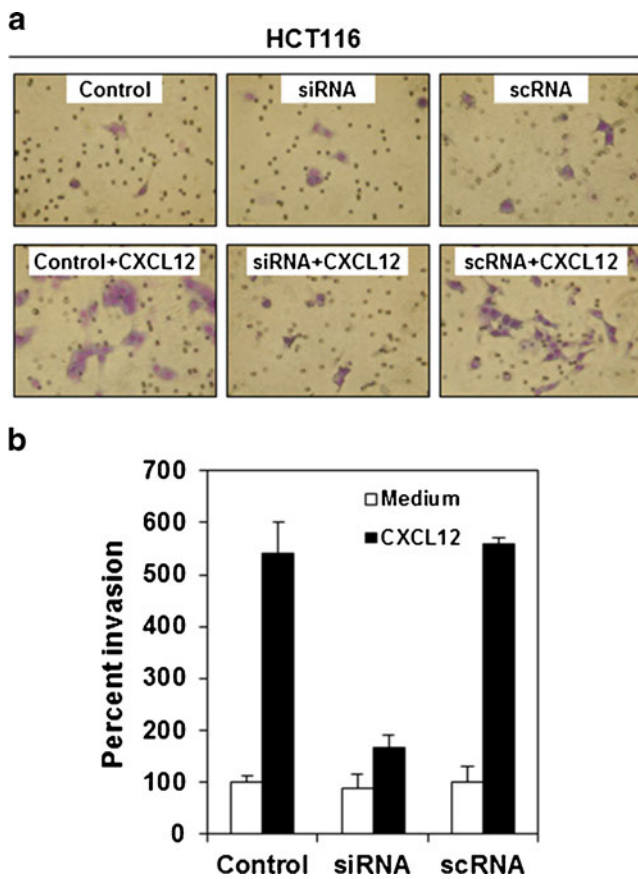


Fig. 5 Celastrol suppresses invasion in colon cancer cells. **a** HCT116 cells (0.25×10^6 cells per well) were transfected with siRNAs and the transfected cells were collected after 48 h. After transfection, cells were seeded in the top chamber of Matrigel. Transwell chambers were then placed into 24-well plates in which either the basal medium was added or 100 ng/mL CXCL12 in the basal medium. After the incubation, invasion assay was done as described in the “Materials and methods” section. The results shown are representative of two independent experiments. **b** Histogram of data obtained from invasion assay in Fig. 5a. ..., bars SE. * $P < 0.05$

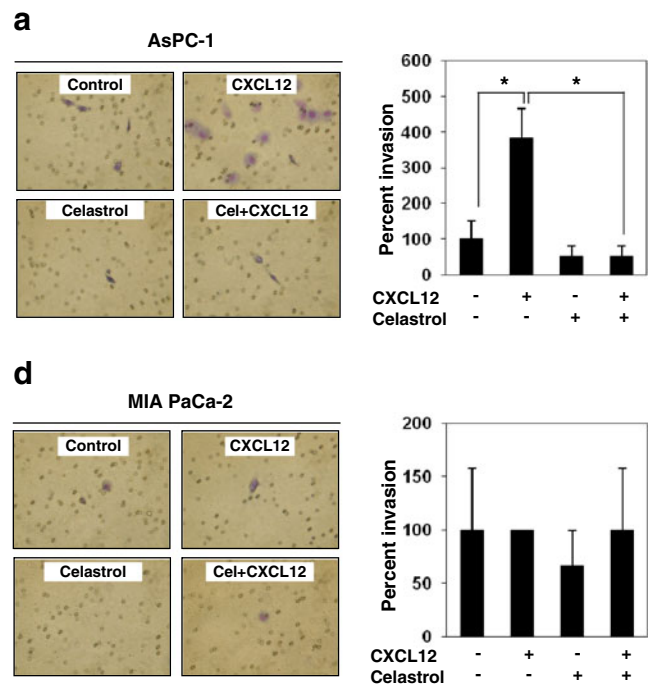


Fig. 6 Celastrol suppresses CXCR4 and invasion in pancreatic cancer cells. **a** *Left panel* AsPC-1 cells (2×10^5 ; 2 % FBS–DMEM) were seeded in the top chamber of Matrigel. After preincubation with or without celastrol (3 $\mu\text{mol/L}$) for 6 h, Transwell chambers were then placed into 24-well plates in which either the basal medium was added or 100 ng/mL CXCL12 in basal medium. After incubation, invasion assay was done as described in the “Materials and methods” section. The results shown are representative of two independent experiments. *Right panel* histogram of data obtained from invasion assay in Fig. 6a, left panel, SE. * $P < 0.05$**d** *Left panel* MIA PaCa-2 cells (2×10^5 ; 2 % FBS–DMEM) were seeded in the top chamber of Matrigel and invasion assay was done as describe above. The results shown are representative of two independent experiments. *Right panel* histogram of data obtained from invasion assay in Fig. 6d, left panel