

## CORRIGENDUM

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**Ski prevents TGF- $\beta$ -induced EMT and cell invasion by repressing SMAD-dependent signaling in non-small cell lung cancer**

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Following the publication of the above paper, an interested reader drew to our attention apparent anomalies associated with Figs. 2, 3 and 4; essentially, these three figures contained panels exhibiting overlapping data, such that data purportedly relating to different experiments were apparently drawn from the same original sources. [Specifically, the Ski, +TGF- $\beta$ 1 data panel in Fig. 2B, the Mock, +TGF- $\beta$ 1 data panel in Fig. 3A, and the +TGF- $\beta$ 1, +SIS3 data panel in Fig. 4B in the original figures were chosen incorrectly.]

Upon investigating this matter with the authors, the authors have realized that they made errors in the compilation of the affected figures. The errors were made inadvertently, and the authors have been able to identify the correct data for each of the figures concerned. The corrected versions of these figures are shown opposite and on the next page. Note that these errors did not affect the overall conclusions reported in the study. The authors are grateful to the Editor of *Oncology Reports* for allowing them the opportunity to publish this Corrigendum; furthermore, the authors apologize for any inconvenience caused to the readership of the Journal.



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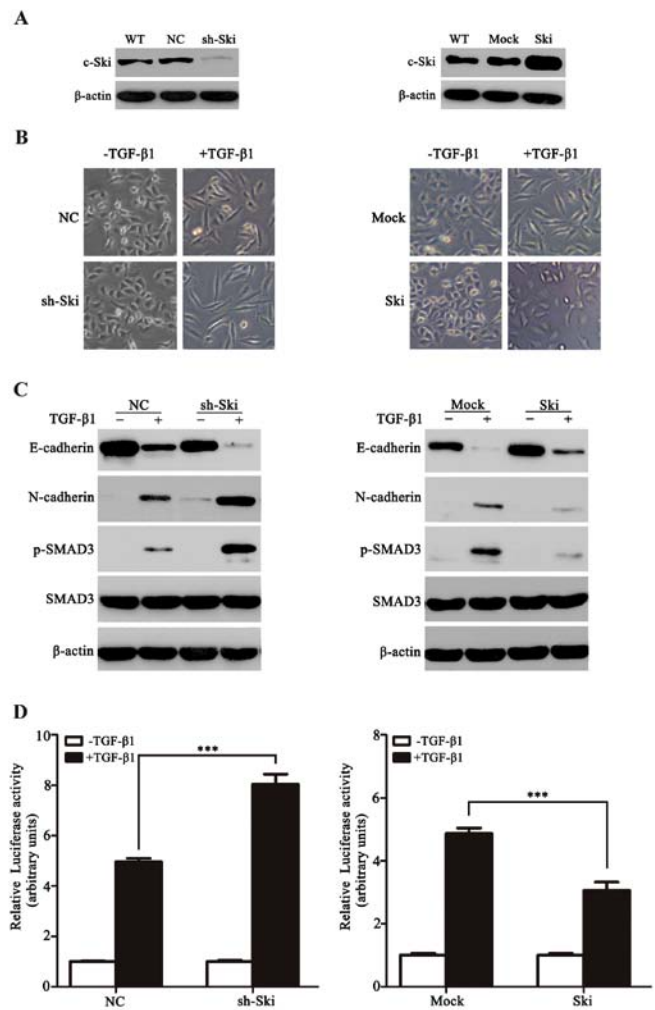


Figure 2. Ski significantly prevents TGF- $\beta$ 1-induced EMT and TGF- $\beta$ 1-activated transcriptional response. (A) Expression of Ski protein in stable Ski-silenced A549 cells (A549-sh-Ski) and Ski-overexpressed A549 cells (A549-Ski). (B) After being serum-starved for 24 h, A549-sh-Ski and A549-Ski cells were treated with or without 5 ng/ml TGF- $\beta$ 1 for 24 h, respectively. Cell morphology was examined and photographed using a phase-contrast microscope. (C) A549-sh-Ski and A549-Ski cells were treated as above. The protein levels of E-cadherin, N-cadherin, SMAD3 and p-SMAD3 were determined by western blotting. (D) A549-sh-Ski and A549-Ski cells transfected with PAI-1 promoter luciferase constructs were incubated for 24 h in the absence or presence of 5 ng/ml TGF- $\beta$ 1, and then subjected to luciferase assays as described in Materials and methods. Relative reporter activity is normalized to that of pGL3-basic reporter. \*\*\*P<0.001. TGF- $\beta$ 1, transforming growth factor- $\beta$ 1; EMT, epithelial-mesenchymal transition; p-SMAD3, phosphorylated-SMAD3; PAI-1, plasminogen activator inhibitor-1.

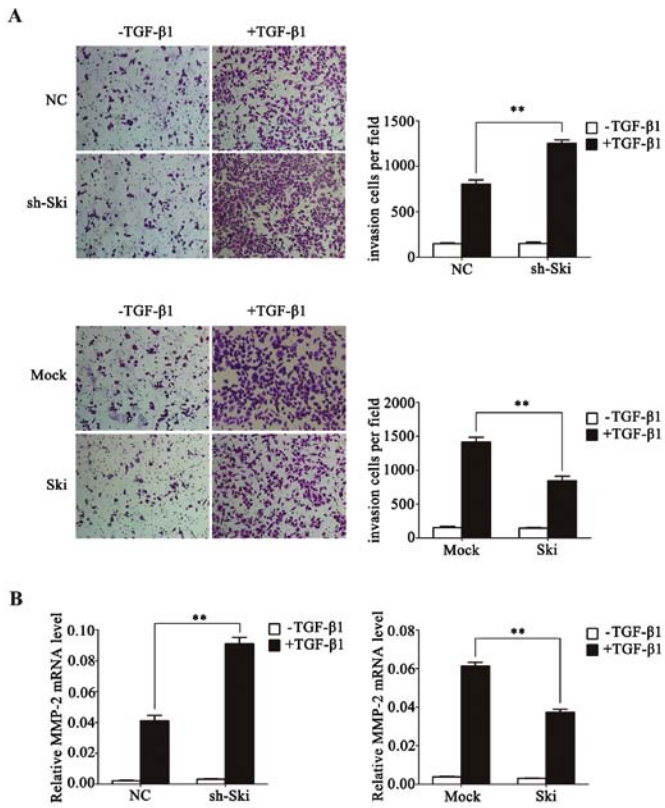


Figure 3. Ski inhibits TGF-β1-mediated cell invasion in A549 cells. (A) A Transwell assay was performed for the effect of Ski on TGF-β1-mediated cell invasion. A549-sh-Ski and A549-Ski stable cells were plated onto Matrigel pre-coated Transwell chambers in the presence or absence of 5 ng/ml TGF-β1 for 24 h, and allowed to invade through an 8-μM pore in Transwells. Invasive cells through the pores were stained and counted in at least three microscopic fields (magnification, ×100). (B) After being serum-starved for 24 h, A549-sh-Ski and A549-Ski cells were incubated with 5 ng/ml TGF-β1 for 24 h, and MMP-2 mRNA levels were determined by RT-qPCR. \*\*P<0.01. TGF-β1, transforming growth factor-β1.

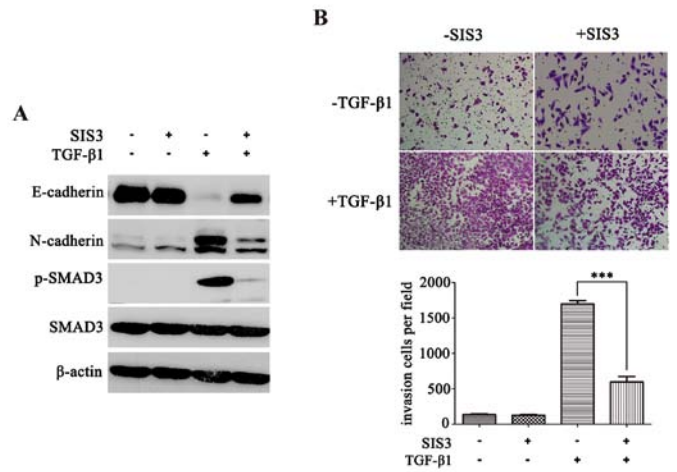


Figure 4. p-SMAD3 plays a vital role in TGF-β1-induced EMT and cell invasion. (A) Repression of p-SMAD3 significantly attenuated TGF-β1-induced EMT. After being pre-treated with 3 μM SIS3 for 6 h, A549 cells were exposed to 5 ng/ml TGF-β1 for 24 h. The levels of E-cadherin, N-cadherin, p-SMAD3 and SMAD3 were determined by western blotting. (B) The effect of p-SMAD3 inactivation on TGF-β1-induced cell invasion. A549 cells were plated onto Matrigel pre-coated Transwell chambers. After being treated with 3 μM SIS3 for 6 h, A549 cells were incubated with 5 ng/ml TGF-β1 for 24 h. Invasive cells were stained and counted in at least three microscopic fields (magnification, ×100). \*\*\*P<0.001. p-SMAD3, phosphorylated-SMAD3; TGF-β1, transforming growth factor-β1; EMT, epithelial-mesenchymal transition.