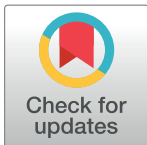


CORRECTION

Correction: *DeltaNp63alpha*-Mediated Induction of Epidermal Growth Factor Receptor Promotes Pancreatic Cancer Cell Growth and Chemoresistance

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There is an error in Fig 2. Fig 2D includes an incorrect image among the representative images showing cell migration. The image for SF (0h) for $\Delta Np63\alpha$ is a duplication of the image for EGF (0h) for Control.



OPEN ACCESS

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Please see the corrected Fig 2 here.

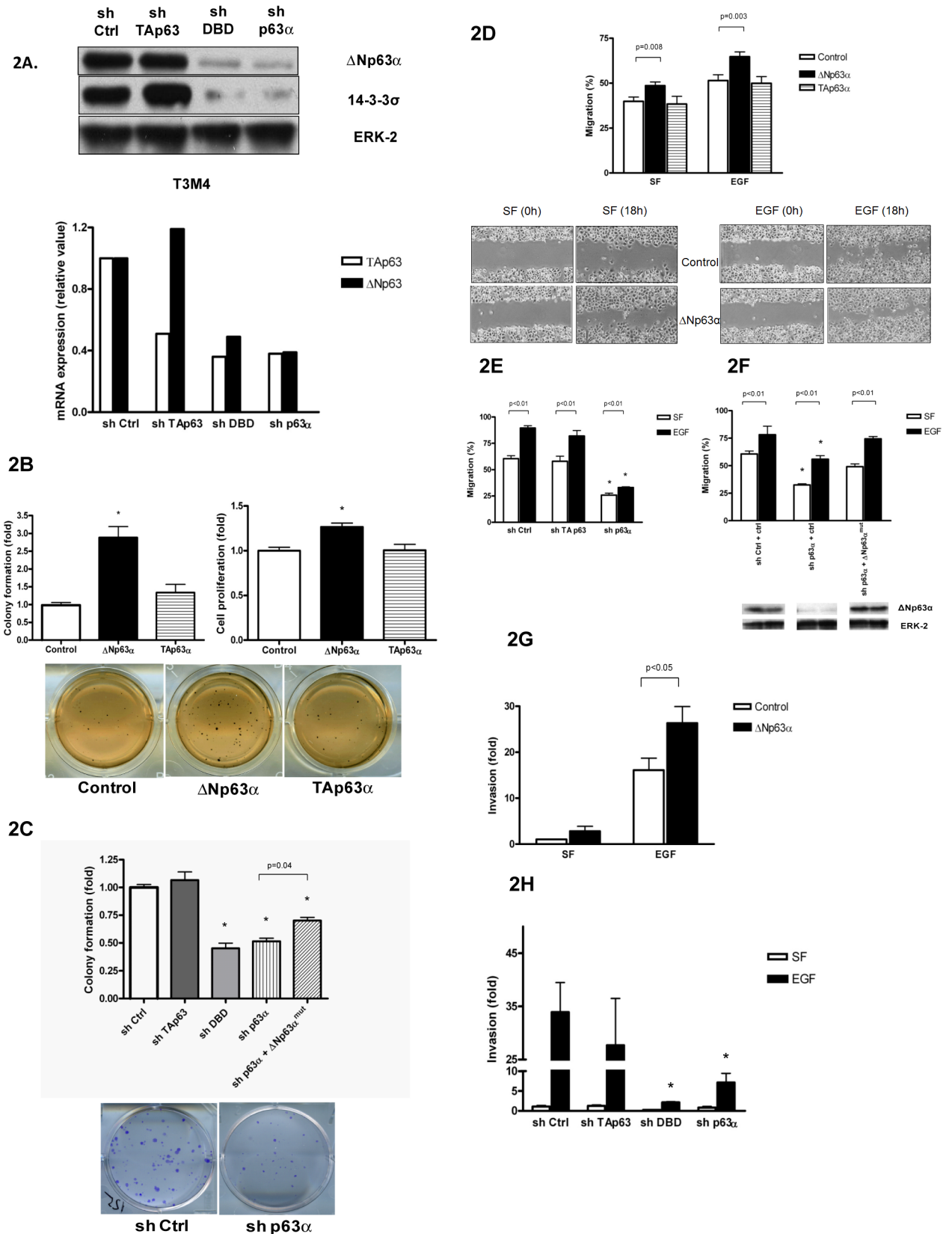


Fig 2. Effect of Δ Np63 α on anchorage-independent growth, motility, and invasion in pancreatic cancer cells. A, T3M4 cells were infected with GFP-expressing virus, or p63-specific shRNA complementary to DBD, TA and α -specific domains of p63. Whole-cell protein lysates were subjected to immunoblotting (top panel). Total RNA was isolated, reverse-transcribed and subjected to real-time PCR with probes specific for Δ N and TA isoforms of p63. Results were normalized to 18S levels (bottom panel). Knockdown of p63 isoforms was routinely monitored during the subsequent experiments. B, Δ Np63 α stimulates anchorage-independent growth of PANC-1 cells in soft agar assay (left and bottom panels) and cell proliferation in MTT assay (right panel). PANC-1 cells were transiently transfected with Δ Np63 α -expressing vector, TAp63 α or control vector. Cells were plated in soft agar at a density of 1500/well of a 12-well plate, four wells per sample. Colonies were counted after 14 days of incubation. For MTT assay cells were plated in 96-well plates, six wells per sample. MTT was added after incubation for 48 h. Data are the mean \pm SE of four independent experiments. *, $p < 0.001$ compared with control. C, Downregulation of Δ Np63 α slows proliferation of T3M4 cells in a clonogenic assay. Reconstitution of Δ Np63 α partially restores the proliferative ability. Cells were plated on 6 well plates at a density of 500 cells/well. 14 days later, plates were fixed in 3:1 methanol:glacial acetic acid and stained with 2% crystal violet. Data are the mean \pm SE of three independent experiments done in triplicates. *, $p < 0.01$ compared with control (sh ctrl). D–F, Effect of p63 on cell motility measured in wound-healing assays in PANC-1 (D) and T3M4 cells (E, F). Cells were incubated in serum-free (SF) conditions in the absence or presence of 1 nM EGF for 18 h after making a scratch. Quantitative analysis of the images was performed. Ectopic expression of mouse Δ Np63 α in sh p63 α T3M4 cells resulted in a partial restoration of cell motility (F); corresponding Δ Np63 α protein levels shown below. Data are the mean \pm SE of at least three independent experiments. Representative pictures shown (magnification $\times 40$). *, $p < 0.001$ compared with control (sh ctrl). G and H, Effect of Δ Np63 α on the invasion in Matrigel chambers. PANC-1 (G) or T3M4 (H) cells were plated in Matrigel chambers (5 \times 10⁴/ml) and incubated in absence (SF) or presence of 1 nM EGF for 18 hours. Effect was normalized to invasion of control in SF conditions. *, $p < 0.001$ compared with control (sh ctrl).

<https://doi.org/10.1371/journal.pone.0192927.g001>

Reference

1. Danilov AV, Neupane D, Nagaraja AS, Feofanova EV, Humphries LA, DiRenzo J, et al. (2011) *DeltaN-p63alpha*-Mediated Induction of Epidermal Growth Factor Receptor Promotes Pancreatic Cancer Cell Growth and Chemoresistance. PLoS ONE 6(10): e26815. <https://doi.org/10.1371/journal.pone.0026815> PMID: 22053213