

CORRECTION

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Correction: Over-expression of oncogenic pseudogene DUXAP10 promotes cell proliferation and invasion by regulating LATS1 and β -catenin in gastric cancer

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Following publication of the original article [1], an error was identified in Figs. 3b and 4d/e, and Fig. 7c.

The corrected figures are given below. The corrections do not affect the conclusions of the article.

The online version of the original article can be found at <https://doi.org/10.1186/s13046-018-0684-8>.

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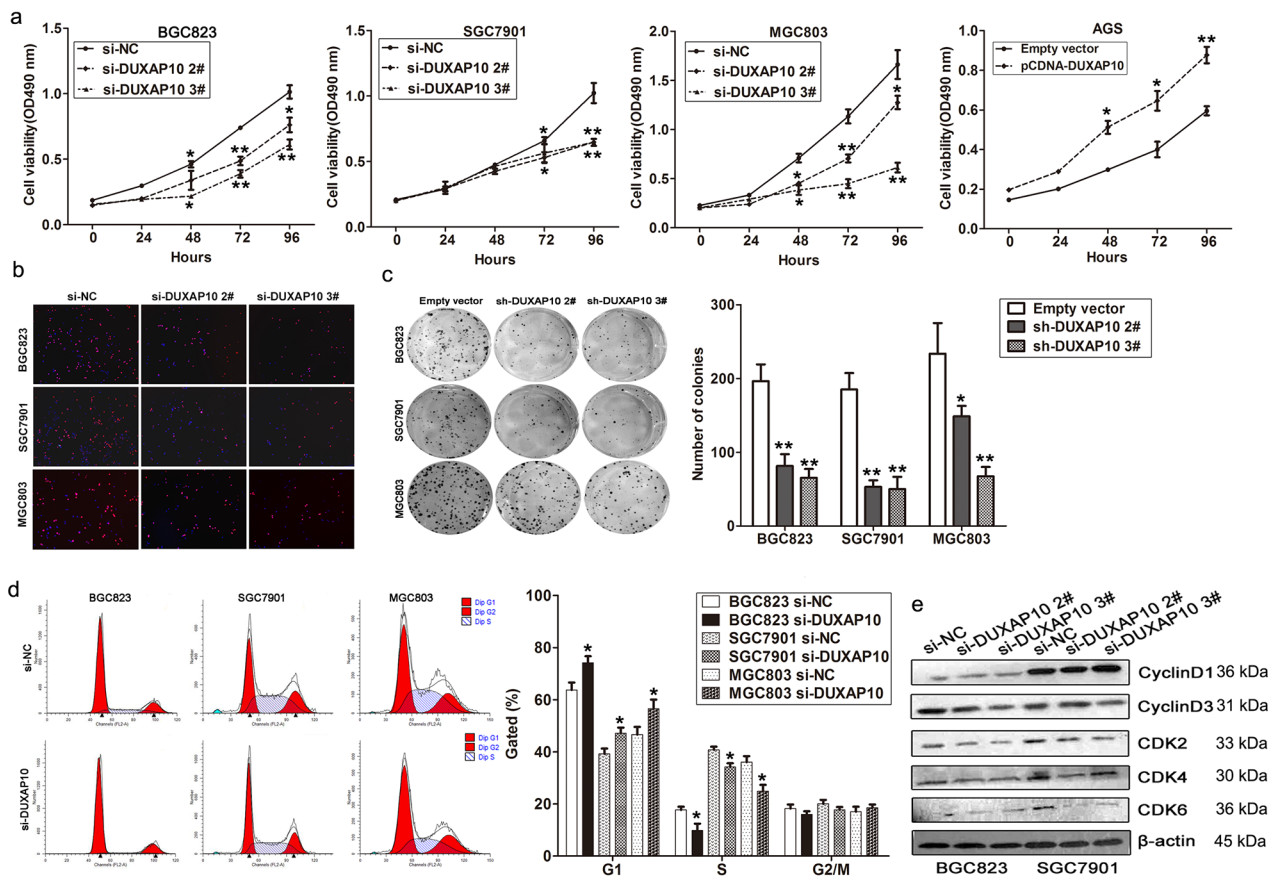


Fig. 3 DUXAP10 promotes GC cells growth and cell cycle progression. **a** MTT assays were used to determine the cell viability for si-DUXAP10 or si-NC transfected BGC823, SGC7901 and MGC803 cells, and DUXAP10 vector or empty vector transfected AGS cells. Values represented the mean ± s.d. from three independent experiments. **b** Edu staining analysis showing significant decrease of cell viability in si-DUXAP10 transfected BGC823, SGC7901 and MGC803 cells. **c** Colon formation assays showing significant decrease of cloning viability in si-DUXAP10 transfected GC cells. **d** FACS analysis shows significant increases or decreases of cells in G1 or S phase, respectively, in si-DUXAP10 transfected GC cells. **e** Cyclin D1, Cyclin D3, CDK2, CDK4, and CDK6 protein levels were detected by western blot analysis after DUXAP10 knockdown. *P < 0.05, **P < 0.01

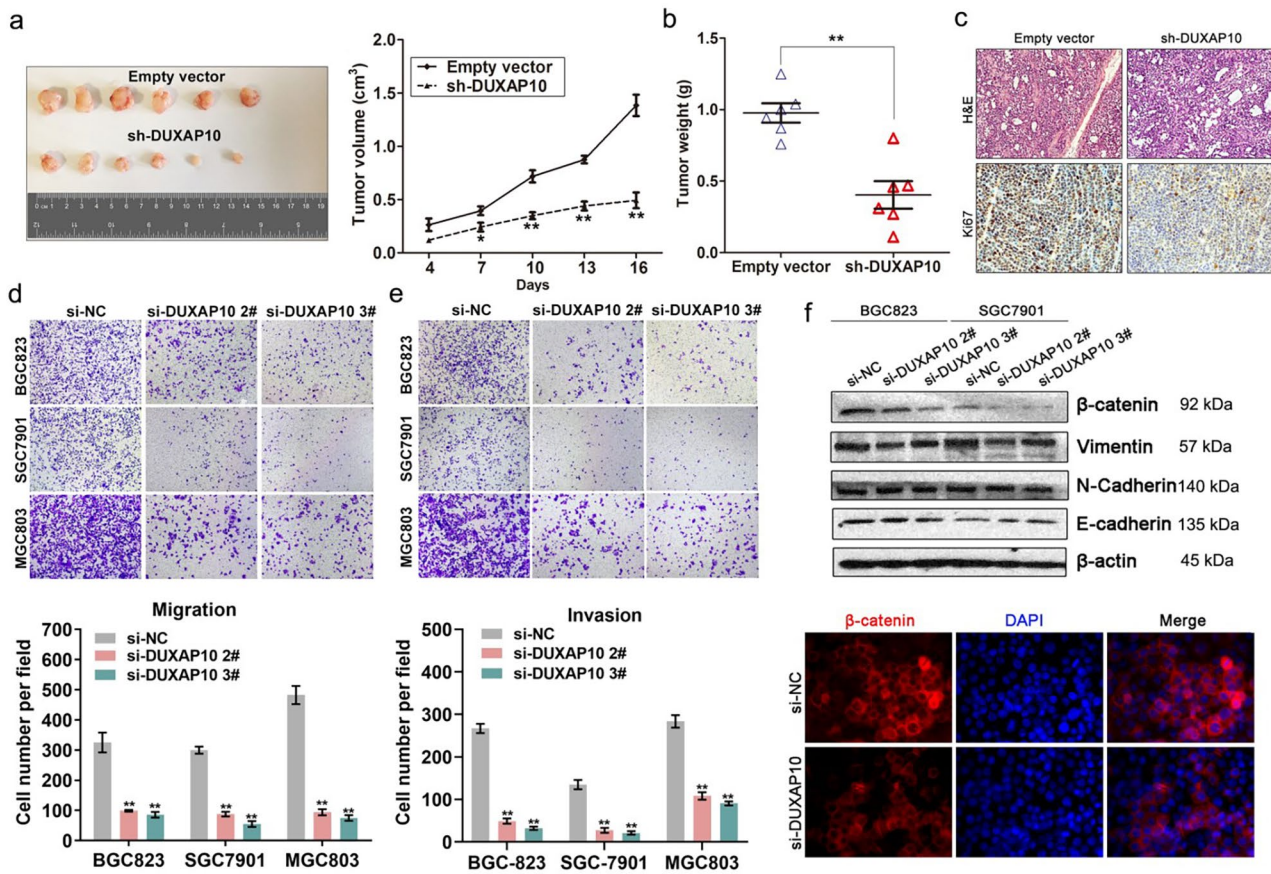


Fig. 4 DUXAP10 down-regulation inhibits GC cells tumor growth in vivo, and invasion in vitro. **a** Representative images of tumors formed in nude mice injected subcutaneously with DUXAP10 knockdown BGC823 cells, and the tumor growth curves of DUXAP10 down-regulation and control groups. **b** Tumors induced by DUXAP10 knockdown in BGC823 cells showed markedly lower weight than control cells. **c** Tumors developed from sh-DUXAP10 transfected BGC823 cells showed lower ki67 protein levels than tumors developed by control cells. Up: H & E staining; Down: immunostaining. **d,e** Transwell assays were used to investigate the changes in migratory and invasive abilities of DUXAP10 knockdown cells. **f** E-cadherin, N-cadherin, Vimentin and β -catenin protein levels were detected by western blot and Immunofluorescence analysis after DUXAP10 knockdown in BGC823 cells. * $P < 0.05$, ** $P < 0.01$

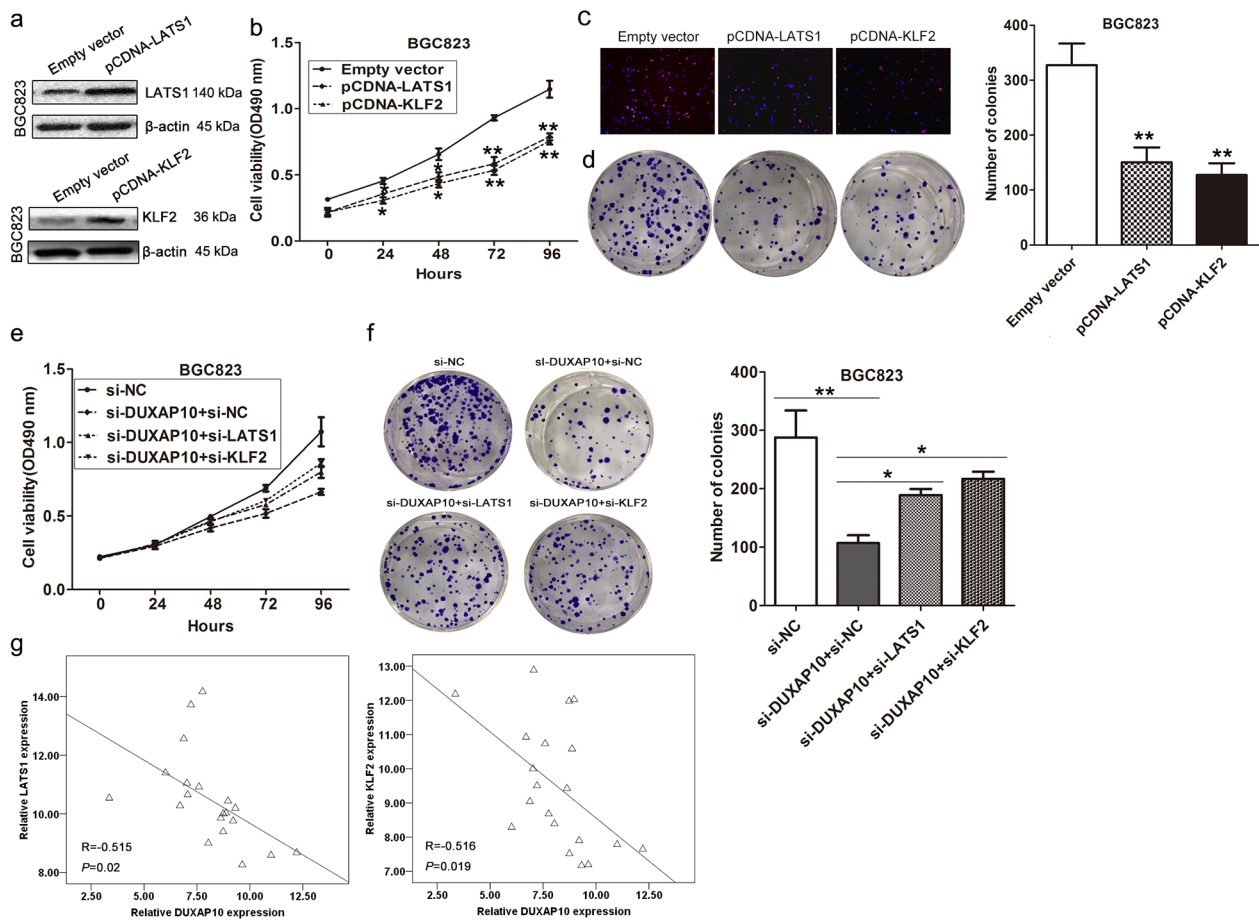


Fig. 7 DUXAP10 promotes GC cell proliferation partly via regulating LATS1 and KLF2. **a** KLF2 and LATS1 protein levels were detected by western blot in BGC823 cells transfected with KLF2 or LATS1 vector. **b** MTT assays were used to determine the cell viability for LATS1 and KLF2 vector or empty vector transfected BGC823 and SGC7901 cells. **c,d** Edu staining and colony formation assays were used to determine the cell viability for LATS1, KLF2 vector or empty vector transfected cells. **e,f** MTT and colony formation assays showed that cell proliferation was partly rescued by KLF2 and LATS1 knockdown in DUXAP10 siRNA transfected cells. **g** The correlation between DUXAP10 and KLF2, or LATS1 expression was detected in 20 pairs of GC and corresponding noncancerous tissues by qRT-PCR

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References

1. Xu Y, Yu X, Wei C, et al. Over-expression of oncogenic pseudogene DUXAP10 promotes cell proliferation and invasion by regulating LATS1 and β -catenin

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