

CORRECTION

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Correction: Long non-coding RNA SNHG17 is an unfavourable prognostic factor and promotes cell proliferation by epigenetically silencing P57 in colorectal cancer

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 Correction for 'Long non-coding RNA SNHG17 is an unfavourable prognostic factor and promotes cell proliferation by epigenetically silencing P57 in colorectal cancer' by Zhonghua Ma *et al.*, *Mol. Biosyst.*, 2017, **13**, 2350–2361.

The authors regret that incorrect flow cytometry images were inadvertently used for Fig. 3 in the original article. The corrected Fig. 3 is included herein.

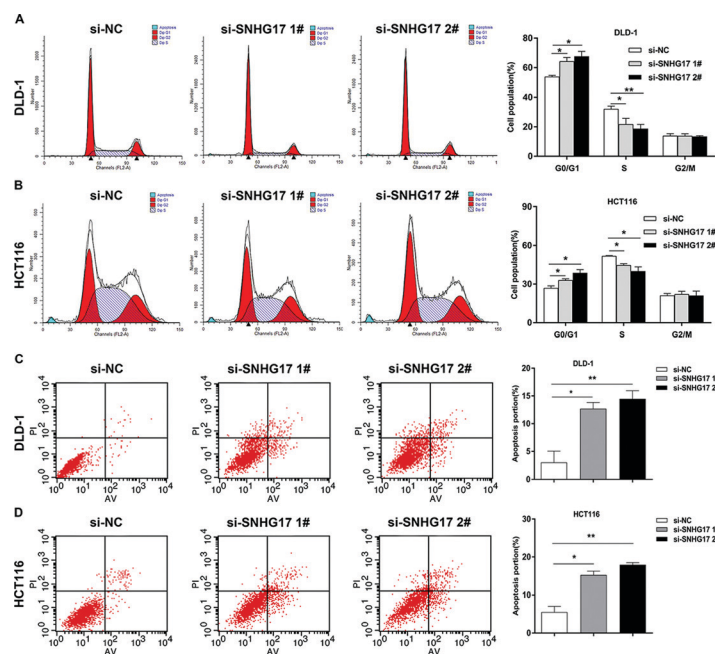


Fig. 3 SNHG17 downregulation promotes cell cycle arrest and apoptosis in CRC cells *in vitro*. (A and B) The bar chart represents the percentage of cells in G0/G1, S, or G2/M phase, as indicated. (C and D) The percentage of apoptotic cells was determined by flow-cytometric analysis. The data represent the mean \pm SD from three independent experiments. * $P < 0.05$ and ** $P < 0.01$.

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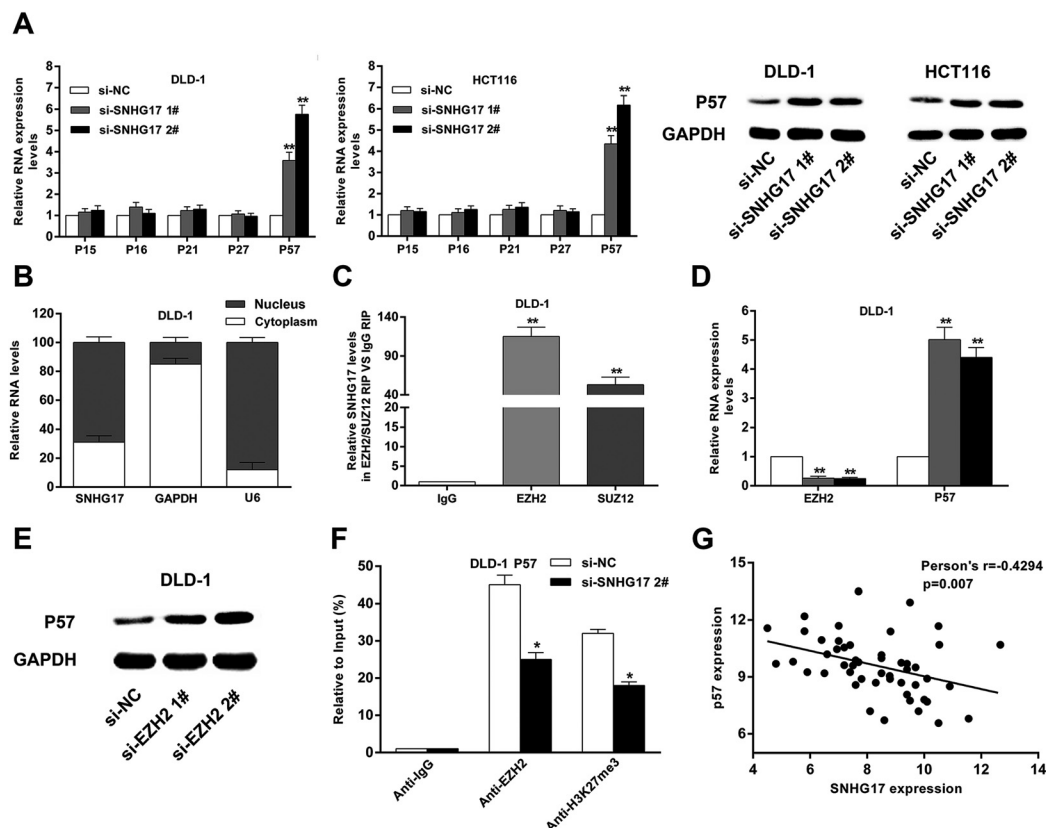



Fig. 5 SNHG17 epigenetically silences P57 transcription by binding to EZH2. (A) The expression of P15, P16, P21, P27 and P57 was determined after knockdown of SNHG17 by qRT-PCR and western blot assays after transfection. (B) The expression levels of SNHG17 in cell nuclear or cytoplasm in DLD1 cells were investigated by qRT-PCR. U6 was used as a nucleus marker, and GAPDH was used as a cytosol marker. (C) RIP assays were performed in DLD-1 cells and the coprecipitated RNA was subjected to qRT-PCR for SNHG17. (D and E) qRT-PCR assays were performed to determine the expression of EZH2 in DLD-1 cells after EZH2 knockdown. P57 expression was investigated in DLD-1 cells after knockdown of EZH2 through qRT-PCR and western blot assays. (F) ChIP-qRT-PCR of EZH2 occupancy and H3K27me3 binding in the P57 promoter in DLD1 cells treated with si-SNHG17 2# (48 h) or si-NC; IgG as a negative control. Error bars indicate mean \pm standard errors of the mean. * $P < 0.05$, ** $P < 0.01$. (G) qRT-PCR experiments were used to detect the expression levels of P57 in 50 pair CRC tissues and the results elucidated a significantly negative correlation between SNHG17 and P57.

The authors regret that the siRNA labels in the two graphs in Fig. 5A of the original article are incorrect. The labels si-SNHG17 2# and si-SNHG17 3# in the original article should read si-SNHG17 1# and si-SNHG17 2#, respectively. The corrected Fig. 5 is included herein.

The authors would also like to add a clearer citation to the CRC gene expression data from the Gene Expression Omnibus (GEO) dataset GSE21510 used in the study (ref. 1). These data were cited in the original article using the link <https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE21510>.

Finally, the authors would like to draw attention to the fact that the analysis presented in Fig. 1A in the original manuscript is the same as that presented in Fig. 1a of ref. 2. In each case the data were used to select the candidate gene to study.

The Royal Society of Chemistry apologises for these errors and any consequent inconvenience to authors and readers.

References

- 1 S. Tsukamoto, T. Ishikawa, S. Iida, M. Ishiguro, K. Mogushi, H. Mizushima, H. Uetake, H. Tanaka and K. Sugihara, *Clin. Cancer Res.*, 2011, **17**, 2444–2450.
- 2 J. Ding, J. Li, H. Wang, Y. Tian, M. Xie, X. He, H. Ji, Z. Ma, B. Hui, K. Wang and G. Ji, *Cell Death Dis.*, 2017, **8**, e2997.

