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Author Correction: Aptamer-mediated survivin RNAi enables 5-fluorouracil to eliminate colorectal cancer stem cells

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Correction to: *Scientific Reports* https://doi.org/10.1038/s41598-017-05859-z, published online 19 July 2017

This Article contains an error in the order of panels of Figure 1. Panels a, b, c, d and e should appear in the order d, e, a, b, c. The correct Figure appears below as Figure 1.

Additionally, the panels in Figures 3 and 4 are missing labels. The correct Figures appear below as Figures 2 and 3 respectively.

Finally, the Supplementary Information file contains a typographical error.

"The chimera structure features an asymmetric structure to facilitate recognition by an endogenous Dicer enzyme to cleave the chimera which results in the release of the expected 21-mer siRNA sequence [251] (Figure S1b)."

should read:

"The chimera structure features an asymmetric structure to facilitate recognition by an endogenous Dicer enzyme to cleave the chimera which results in the release of the expected 21-mer siRNA sequence (Figure S1b)."

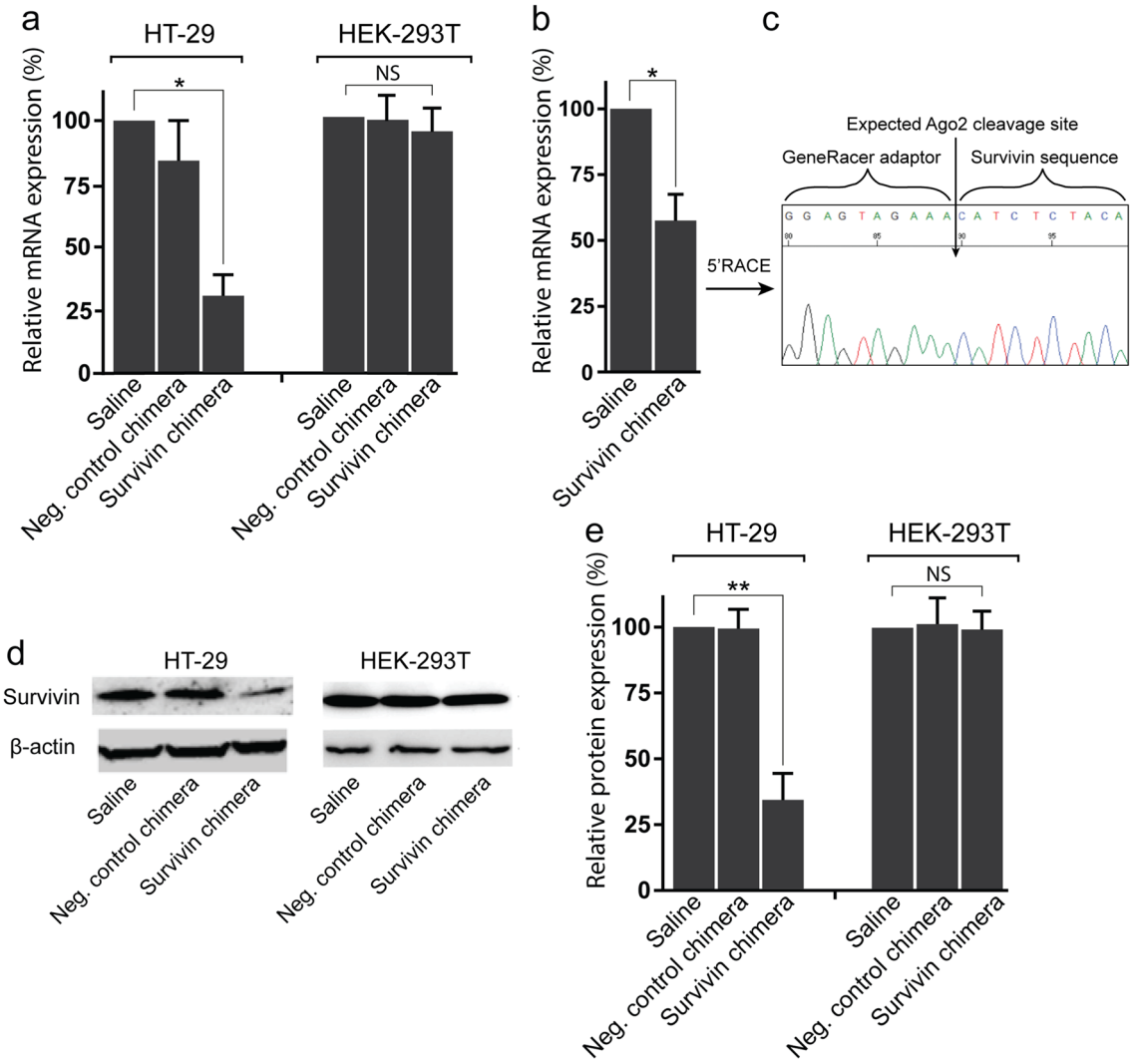

Figure 1. EpCAM aptamer-guided RNAi effectively silenced survivin. (**a**) Specificity and efficacy of EpCAMaptamer guided RNAi in knocking down survivin mRNA. Chimera or negative control chimera were incubated with HT-29 or HEK-2913T cells for 24 hours and the total RNA was extracted for qRT-PCR analysis of survivin mRNA levels. GAPDH was used as an internal control. (**b**, **c**) HT-29 Tumour-bearing mice were treated with 2 nmol/mouse of PEG-labelled chimera for 48 hours. The tumours were collected for RNA extraction followed by qRT-PCR analysis of survivin mRNA expression (**b**) and 5′RACE assay (**c**). (**d**) Effective downregulation of survivin protein via EpCAM aptamer-guided RNAi. Chimera or negative control chimera were incubated with HT-29 or HEK-2913T cells for 48 hours and the survivin protein levels were analyzed using Western blot analysis. β-actin was used as a loading control. (**e**) The bar graph shows the survivin protein levels in various treatment groups. Data shown are means ± SEM, n = 3. $^{*}p < 0.05$, $^{**}p < 0.005$. NS, no statistically significant difference..

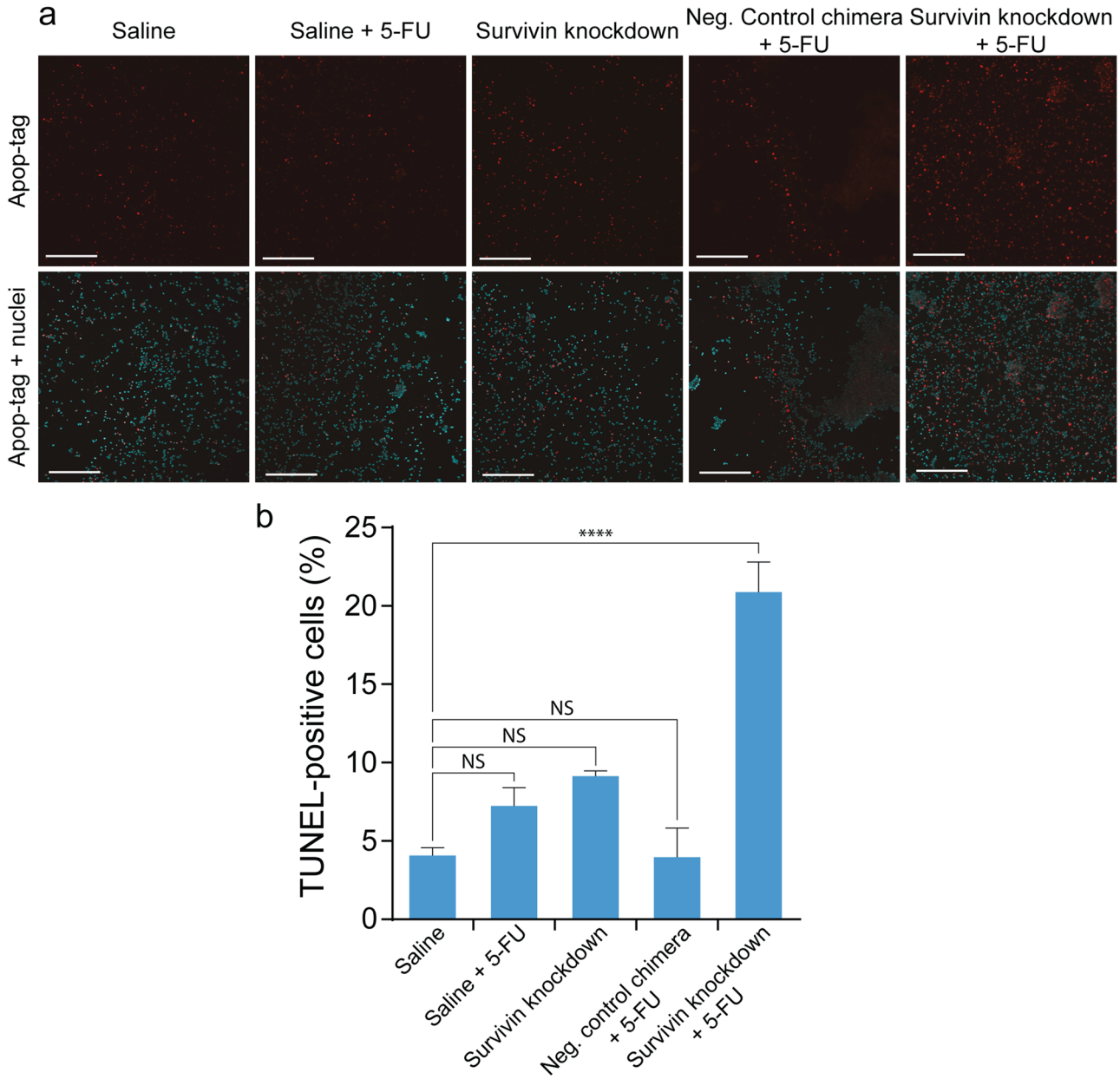

Figure 2. Survivin knockdown *in vivo* enhances 5-FU-induced apoptosis in HT-29 tumour cells. (**a**) Representative images of TUNEL apoptosis assay on dissociated HT-29 xenograft tumours after *in vivo* treatment with chimera and 5-FU. NOD/SCID mice bearing HT-29 tumours (60 mm^3) were treated intravenously with 3 injections of 2 nmol/mouse of chimera with or without 3 additional treatment of 30 mg/kg of 5-FU. Two days after the final treatment, tumours were dissociated by collagenase digestion and subjected to TUNEL apoptosis assay. (**b**) Percentage of apoptotic cells in treated tumours. Data shown are means ± SEM, n = 3. ****$p < 0.001$. NS, no statistically significant difference.

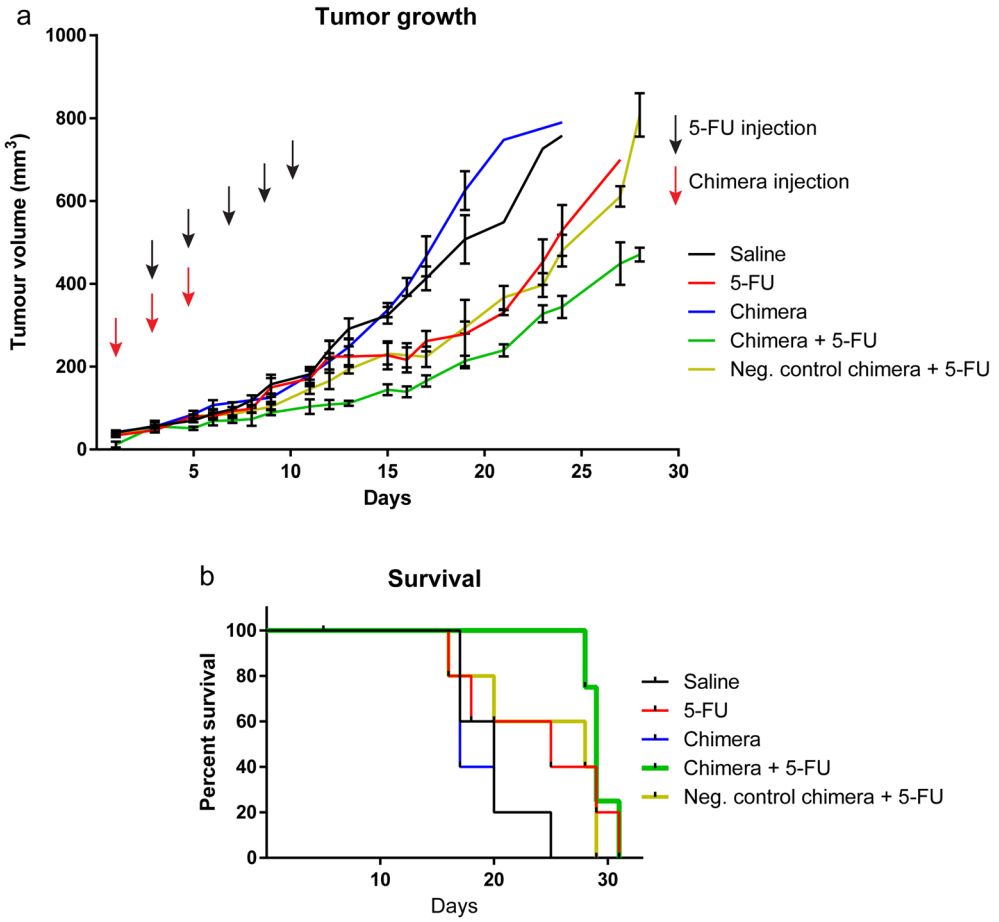

Figure 3. Combined chimera and 5-FU treatments improves therapeutic outcome in HT-29 tumour-bearing mice. (**a**) The graph represents the tumour volume in mice in response to various treatments as indicated. (**b**) Survival rate of mice observed over the course of various treatments. Data shown are mean ± SEM, n = 4–5.