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Erratum

The following was originally published in Volume 26, Number 6, pages 901–911 (DOI: https://doi.org/10.3727/096504017X15061902533715). It was recently revealed there was an error in the process of apoptosis data, and so the flow cytometry analysis was repeated. Some of the results were found to be different from the experimental results, which affected the display in Figures 3 and 5. Corrected versions of the figures are shown here, and the figures have been replaced with the corrected versions in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2018/00000026/00000006/art00008).

Downregulation of MicroRNA-147 Inhibits Cell Proliferation and Increases the Chemosensitivity of Gastric Cancer Cells to 5-Fluorouracil by Directly Targeting PTEN

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Gastric cancer is the fourth most common malignancy and the third leading cause of cancer-related deaths worldwide. This study aimed to investigate the expression patterns, biological roles, and underlying mechanisms of microRNA-147 (miR-147) in gastric cancer. The present study demonstrated that miR-147 was significantly upregulated in gastric cancer tissues and cell lines. Downregulation of miR-147 decreased cell proliferation and enhanced the chemosensitivity of gastric cancer cells to 5-fluorouracil (5-FU) through the cell apoptosis pathway. In addition, phosphatase and tensin homolog (PTEN) was mechanically identified as the direct target of miR-147 in gastric cancer. PTEN knockdown reversed the effects of miR-147 downregulation on the proliferation, chemosensitivity, and 5-FU-induced apoptosis of gastric cancer cells. Moreover, miR-147 regulated the PI3K/AKT signaling pathway in gastric cancer by targeting PTEN. In conclusion, miR-147 suppressed the proliferation and enhanced the chemosensitivity of gastric cancer cells to 5-FU by promoting cell apoptosis through directly targeting PTEN and regulating the PI3K/AKT signaling pathway. This study provides important insight into the molecular mechanism that underlies the chemoresistance of gastric cancer cells. The results of this study could aid the development of a novel therapeutic strategy for gastric cancer.

Key words: Phosphatase and tensin homolog (PTEN); MicroRNA-147 (miR-147); Gastric cancer; Proliferation; Chemosensitivity; 5-Fluorouracil (5-FU)

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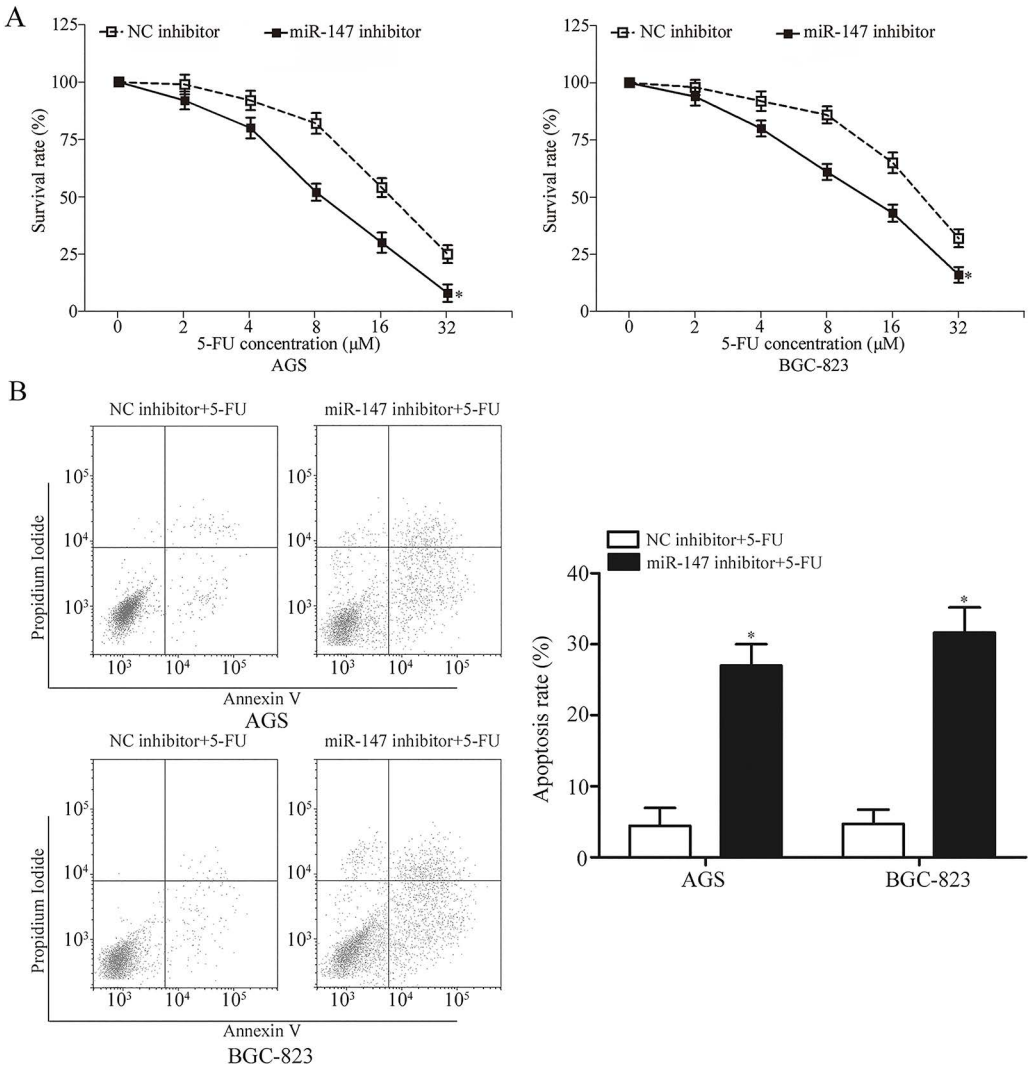

Figure 3. Downregulation of miR-147 enhances the chemosensitivity of gastric cancer cells to 5-fluorouracil (5-FU). (A) In vitro chemosensitivity assays demonstrated that miR-147 underexpression increases the chemosensitivity of AGS and BGC-823 cells to 5-FU relative to that of NC inhibitor-transfected cells. *$p < 0.05$ compared with NC inhibitor. (B) Downregulation of miR-147 promotes the rate of the 5-FU-induced apoptosis in AGS and BGC-823 cells. *$p < 0.05$ compared with NC inhibitor.

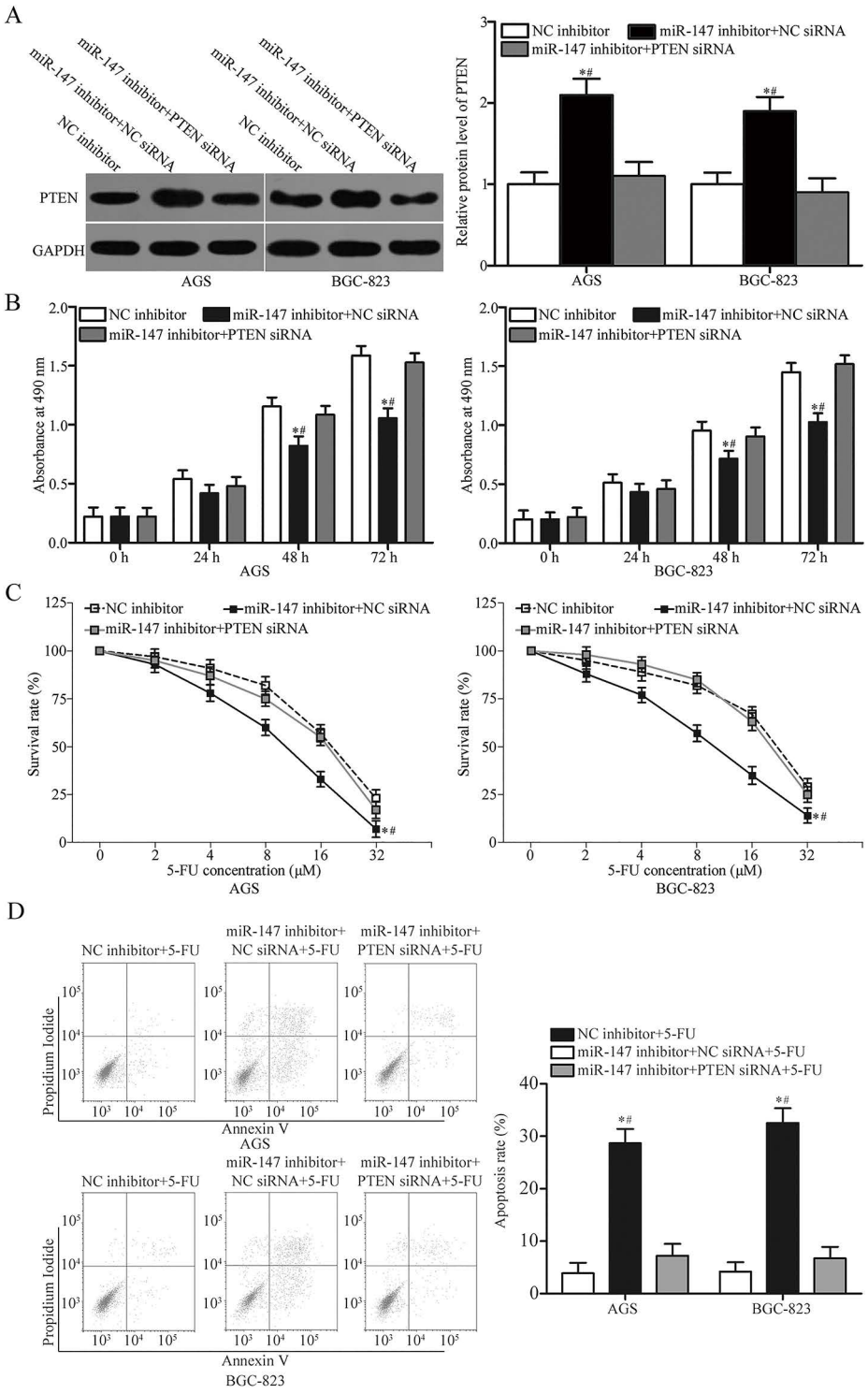

Figure 5. PTEN knockdown restores the effects of miR-147 underexpression on gastric cancer cells. AGS and BGC-823 cells were transfected with NC inhibitor, miR-147 inhibitor + NC small interfering RNA (siRNA), or miR-147 inhibitor + PTEN siRNA. (A) Western blotting analysis was performed to quantify PTEN expression at 72 h posttransfection. *$p < 0.05$ compared with NC inhibitor. #$p < 0.05$ compared with miR-147 inhibitor + PTEN siRNA. (B) MTT assay was carried out to examine cell proliferation in indicated cells. *$p < 0.05$ compared with NC inhibitor. #$p < 0.05$ compared with miR-147 inhibitor + PTEN siRNA. (C) Cell chemosensitivity was assessed in the indicated cells using an in vitro chemosensitivity assay. (D) The rate of 5-FU-induced apoptosis in the indicated cells was evaluated using flow cytometry analysis. *$p < 0.05$ compared with NC inhibitor. #$p < 0.05$ compared with miR-147 inhibitor + PTEN siRNA.