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Erratum

The following was originally published in Volume 25, Number 7, pages 1169–1176 (doi: https://doi.org/10.3727/096504017X14847395834985). In the original article there were some errors in images of flow cytometry analysis and Western blots in Figures 2, 3, 5 and 6. We have revised the figures to correct these images. 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/2017/00000025/00000007/art00014). The corrections do not change any results or conclusion of the article. The authors apologize for any inconvenience caused.

MicroRNA-133a Inhibits Proliferation of Gastric Cancer Cells by Downregulating ERBB2 Expression

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Gastric cancer is the fourth most common type of cancer and the second highest leading cause of cancer-related deaths worldwide. It has already been established that miR-133a is involved in gastric cancer. In this study, we investigated the molecular mechanisms by which miR-133a inhibits the proliferation of gastric cancer cells. We analyzed the proliferative capacity of human gastric cancer cells SNU-1 using an MTT assay. Cell apoptosis was determined using flow cytometry. The expression levels of ERBB2, p-ERK1/2, and p-AKT in SNU-1 cells were determined using Western blot analysis. To confirm that ERBB2 is a direct target of miR-133a, a luciferase reporter assay was performed. Results showed that miR-133a overexpression inhibited SNU-1 cell proliferation and increased apoptosis. ERBB2 was a direct target of miR-133a, and it was negatively regulated by miR-133a. Interestingly, ERBB2 silencing has a similar impact to miR-133a overexpression, in that it significantly induced apoptosis and inhibited ERK and AKT activation. Our study showed that miR-133a inhibits the proliferation of gastric cancer cells by downregulating the expression of ERBB2 and its downstream signaling molecules p-ERK1/2 and p-AKT. Therefore, miR-133a might be used as a therapeutic target for treating gastric cancer.

Key words: MicroRNA-133a (miR-133a); Gastric cancer; ERBB2; p-ERK1/2; p-AKT

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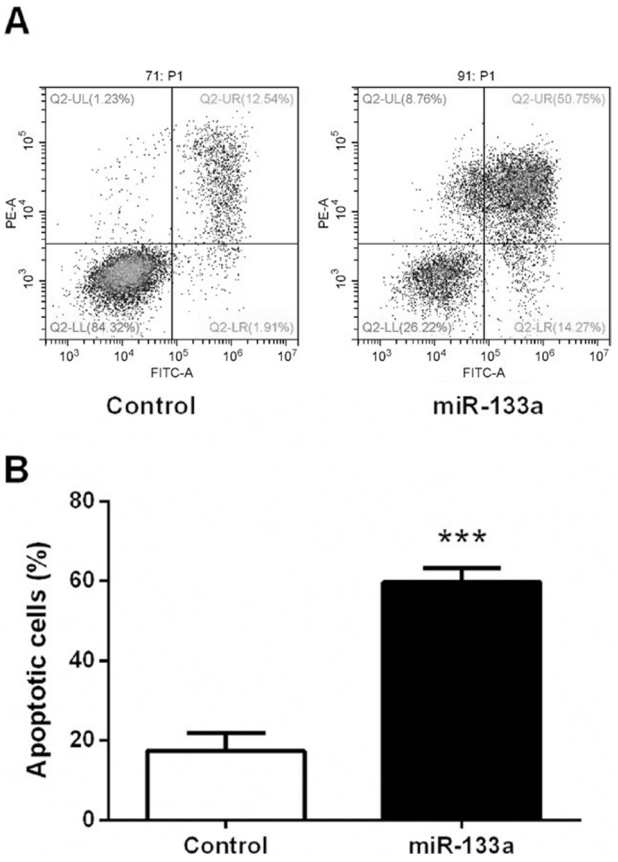

Figure 2. miR-133a overexpression promotes apoptosis of gastric cancer cells. A cell apoptosis assay was performed to examine the rate of apoptosis of SNU-1 cells transfected with miR-133a or control. (A) Flow cytometry analysis. (B) Data from the three samples were quantitated and expressed as mean ± standard error. Control: Pre-miR™ miRNA precursor molecule. ***$p < 0.001$.

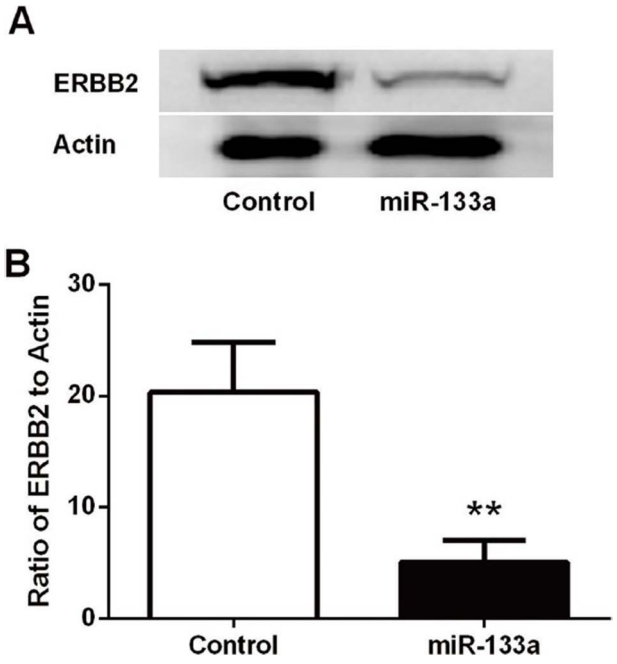

Figure 3. miR-133a overexpression downregulates the expression of ERBB2 in gastric cancer cells. Western blotting was performed to examine the expression level of ERBB2 and -actin in SNU-1 cells transfected with miR-133a or control. -Actin served as control for ERBB2. (A) Western blot images. (B) Data from the three samples were quantitated and expressed as mean ± standard error. Control: Pre-miR™ miRNA precursor molecule. **$p < 0.01$.

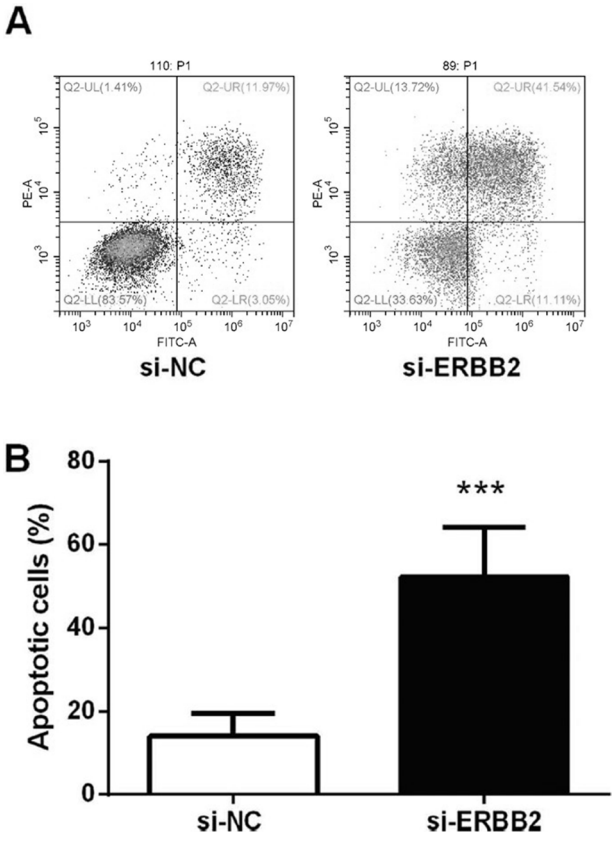

Figure 5. ERBB2 silencing promotes apoptosis of gastric cancer cells. A cell apoptosis assay was performed to examine the rate of apoptosis of SNU-1 cells transfected with ERBB2 siRNA or negative control. (A) Flow cytometry analysis. (B) Data from the three samples were quantitated and expressed as mean ± standard error. ***$p < 0.001$.

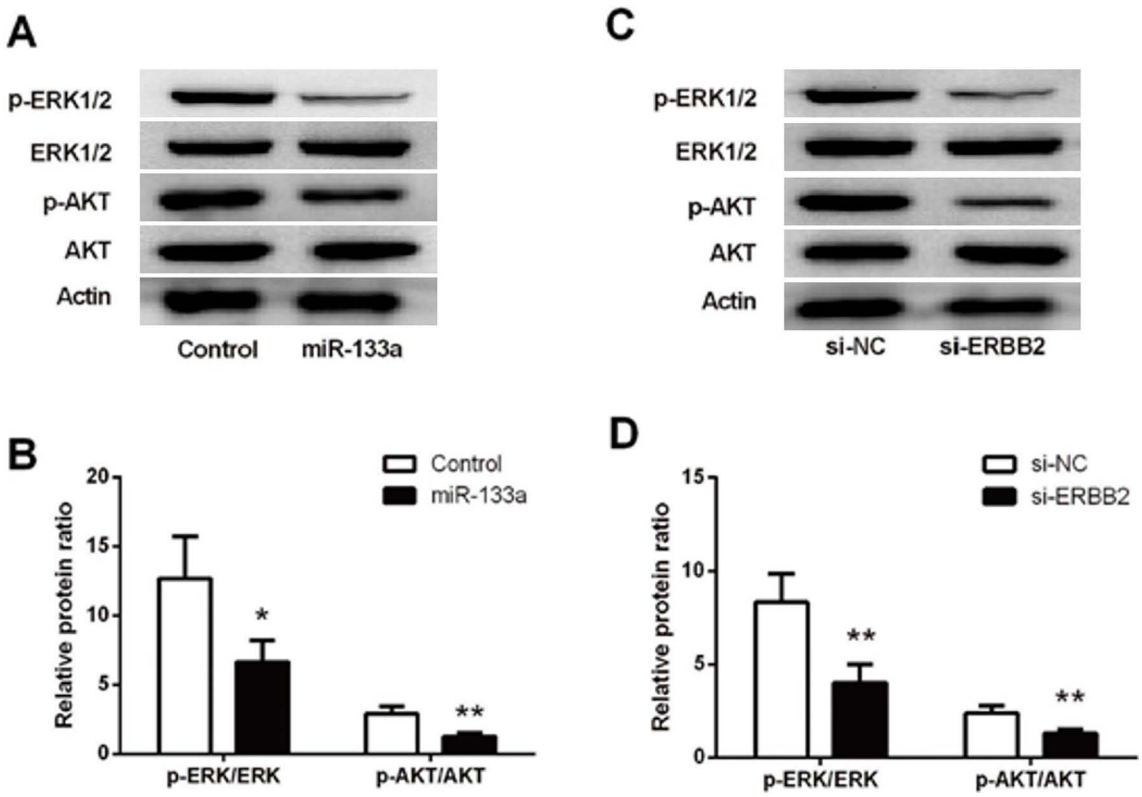

Figure 6. miR-133a overexpression reduces the expression of ERBB2 downstream signaling molecules phosphorylated extracellular signal-regulated kinase (p-ERK) and p-AKT. (A) Western blotting was performed to examine the expression level of p-ERK1/2, ERK1/2, p-AKT, AKT, and -actin in SNU-1 cells transfected with miR-133a or control (Pre-miR™ miRNA precursor molecule). (B) The data from three samples were quantitated for p-ERK1/2 and p-AKT and expressed as mean ± standard error. (C) The expression of these proteins in SNU-1 cells transfected with ERBB2 siRNA or negative control was also detected. (D) Quantitative results from three samples were expressed as mean ± standard error. -Actin served as control for other proteins. *$p < 0.05$; **$p < 0.01$.