

## Erratum

# Circular RNA Hsa\_circRNA\_101996 promotes the development of Gastric Cancer via Upregulating Matrix Metalloproteinases-2/Matrix Metalloproteinases-9 through MicroRNA-143/Ten-eleven translocation-2 Pathway: Erratum

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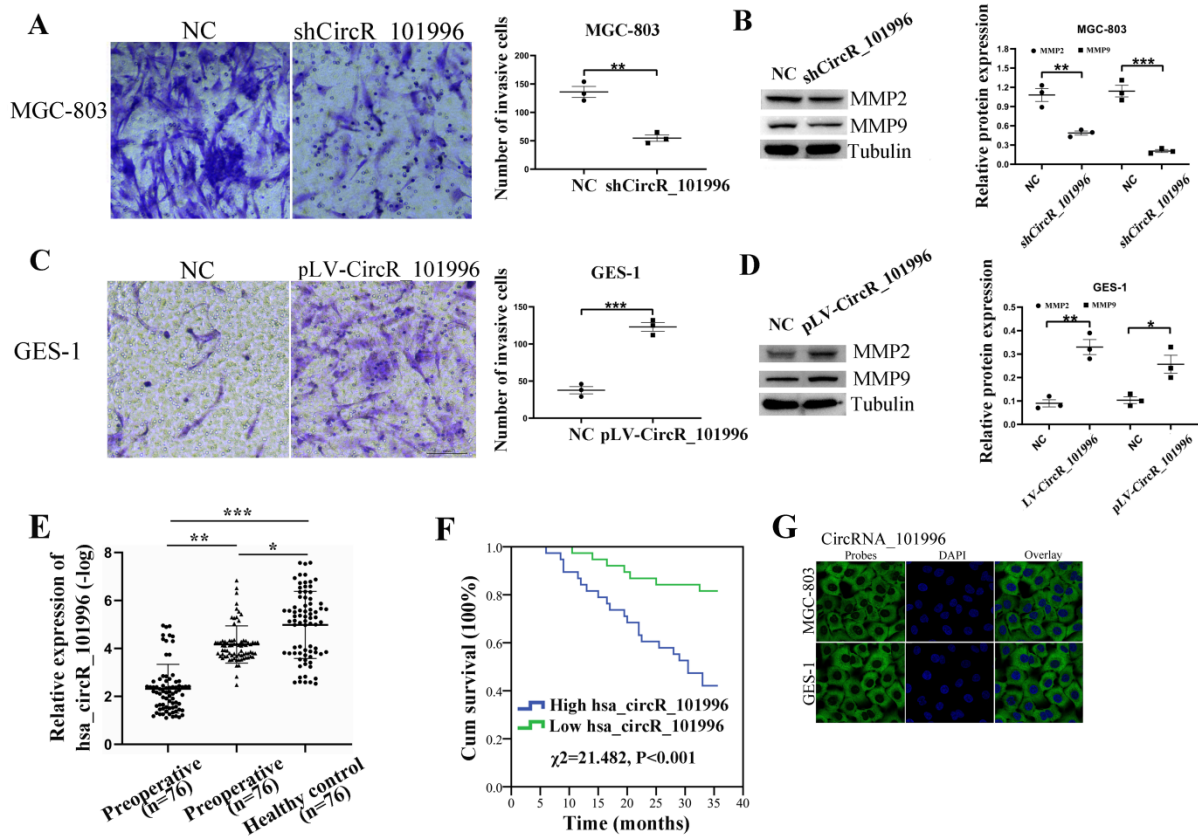
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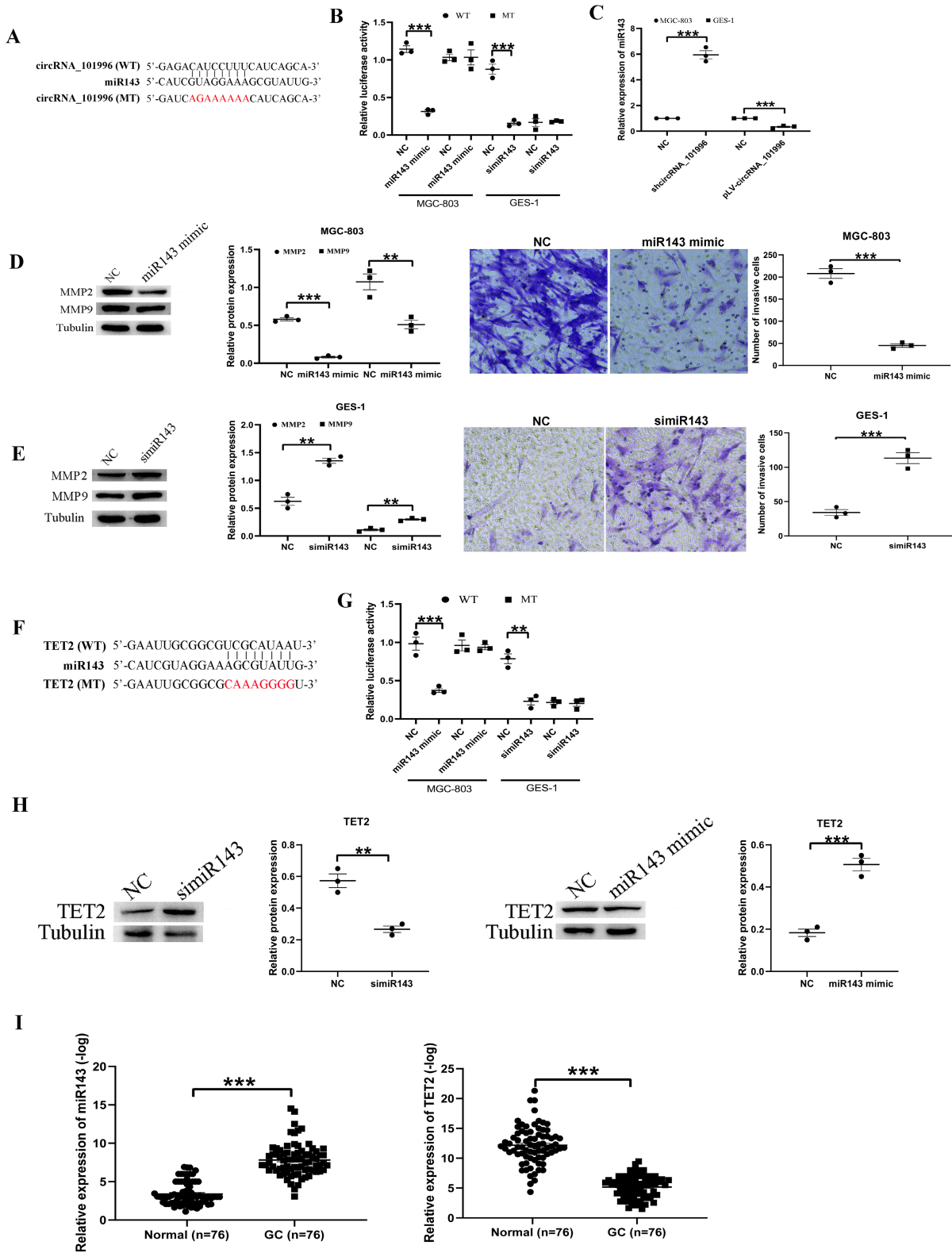
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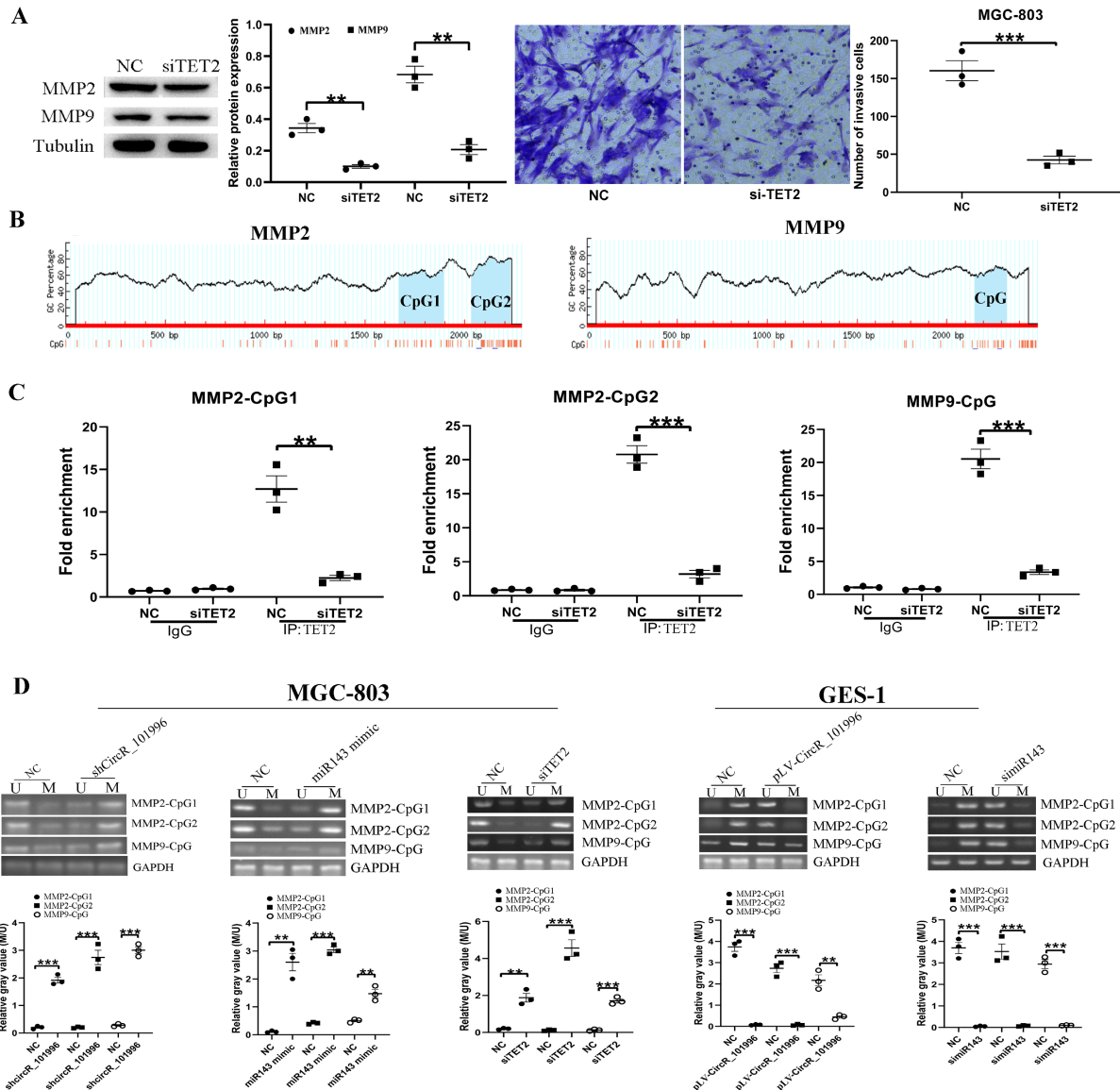
We regret that the original version of our paper unfortunately contained incorrect representative images. The wrong images were placed in Figures 2A-D, 2G, 3D, 3E, 3H, 4A, 5B and 5C when choosing representative images from very large amount of data. The correct version of the Figures 2A-D, 2G, 3D, 3E, 3H, 4A, 5B and 5C appears below. The authors confirm that the corrections made in this erratum do not affect the original conclusions. All the authors of the paper have agreed to this correction. The authors apologize for any inconvenience that the errors may have caused.



**Figure 2.** hsa\_circRNA\_101996 enhances cell invasion and is related to the metastasis and poor prognosis of gastric cancer. (A) Comparing to the NC group, shCircR\_101996 significantly inhibited the invasion ability of MGC-803. (B) Comparing to the NC group, shCircR\_101996 significantly down-regulated the expression of MMP2/MMP9. (C) Comparing to the NC group, overexpression of hsa\_circRNA\_101996 significantly enhanced cell invasion of GES-1. (D) Comparing to the NC group, overexpression of hsa\_circRNA\_101996 significantly enhanced the expressions of MMP2/MMP9. (E) The postoperative plasma hsa\_circRNA\_101996 level of gastric cancer patients was significantly lower than that of preoperative patients, but it was still significantly higher than that of healthy controls. (F) Compared to the low hsa\_circRNA\_101996 expression group, the three-year survival rate of patients in the high hsa\_circRNA\_101996 expression group was significantly lower ( $\chi^2=21.482, P<0.001$ ). \*,  $P<0.05$ ; \*\*,  $P<0.01$ ; \*\*\*,  $P<0.001$ .

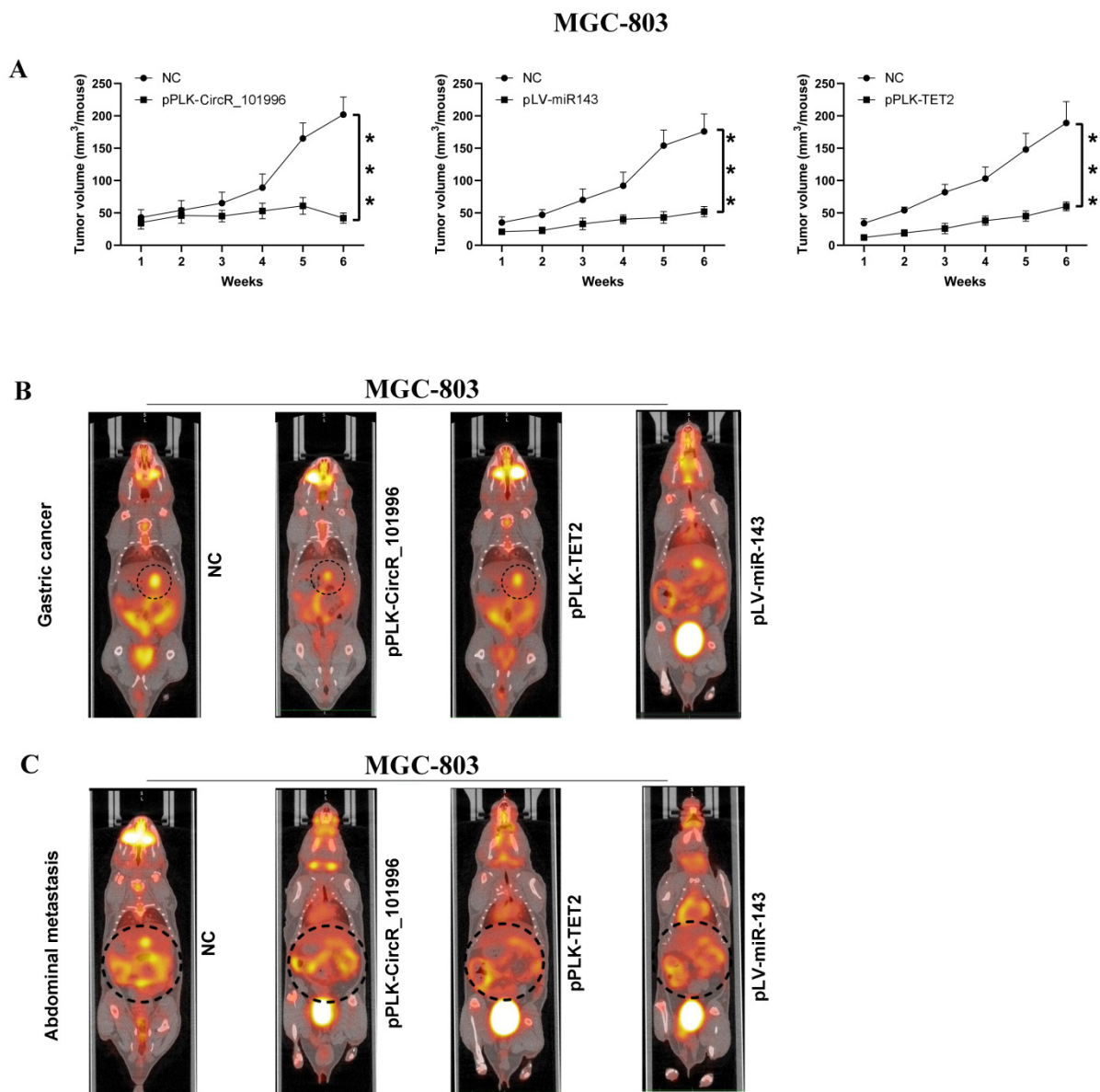


**Figure 3.** hsa\_circRNA\_101996 sponges miR-143 to regulate TET2 expression. (A) hsa\_circRNA\_101996 could bind to miR-143. (B) Overexpression of miR-143 significantly weakened the luciferase activity of wide-type hsa\_circRNA\_101996, while mutation of the binding site blocked the inhibitory effect. (C) Knockdown of hsa\_circRNA\_101996 in MGC-803 increased miR-143 level, while its overexpression in GES-1 led to the decrease of miR-143. (D-E) Overexpression of miR-143 in MGC-803 significantly inhibited the expressions of MMP2/MMP9 and the invasion ability of cells, while knockdown of miR-143 in GES-1 led to the enhanced cell invasion and the increased MMP2/MMP9 expression. (F) miR-143 could bind to the 3'-UTR regions of TET2 mRNA. (G) Overexpression of miR-143 significantly weakened the luciferase activity of wide-type TET2, while mutation of the binding site blocked the inhibitory effect. (H) Overexpression of miR-143 in MGC-803 significantly inhibited the expressions of TET2, while knockdown of miR-143 in GES-1 led to the increased TET2 expression. (I) miR-143 level decreased markedly in gastric cancer tissue compared to that in the adjacent tissue, while the change of TET2 expression was contrary to miR-143. \*\*,  $P < 0.01$ ; \*\*\*,  $P < 0.001$ .



**Figure 4.** TET2 regulates MMP2/MMP9 expression through the epigenetic pathway. (A) MGC-803 showed lower MMP2/MMP9 expression and decreased invasive ability after TET2 knockdown. (B-C) ChIP assay showed that TET2 could bind to the CpG islands in the promoter and Exon 1 of the MMP2/MMP9 gene directly. (D) Overexpression of hsa\_circRNA\_101996 and knockdown of miR-143 could both lead to the hypermethylation of CpG islands. \*\*,  $P < 0.01$ ; \*\*\*,  $P < 0.001$ .





**Figure 5.** Suppressing hsa\_circRNA\_101996/TET2 or overexpressing miR-143 inhibits tumor growth. (A-B) pPLK-CircR\_101996, pPLK-TET2, and pLV-miR-143 all markedly reduced the tumor size of gastric cancer. The scale bar is 5 mm. (C) The pPLK-CircR\_101996 and pPLK-TET2 could both inhibit abdominal metastasis. **\*\***,  $P < 0.001$ .