CORRECTION Open Access



Correction: *Helicobacter pylori*-induced NAT10 stabilizes MDM2 mRNA via RNA acetylation to facilitate gastric cancer progression

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Following publication of the original article [1], an error was identified in Fig. 4, specifically:

• Figure 4D - AGS/CTR and AGS/KO+MDM2 have been uploaded repeatedly

Correct figure is presented below:

[†]Min Deng, Long Zhang and Wenying Zheng contributed equally to this work.

The original article can be found online at https://doi.org/10.1186/s13046-022-02586-w.

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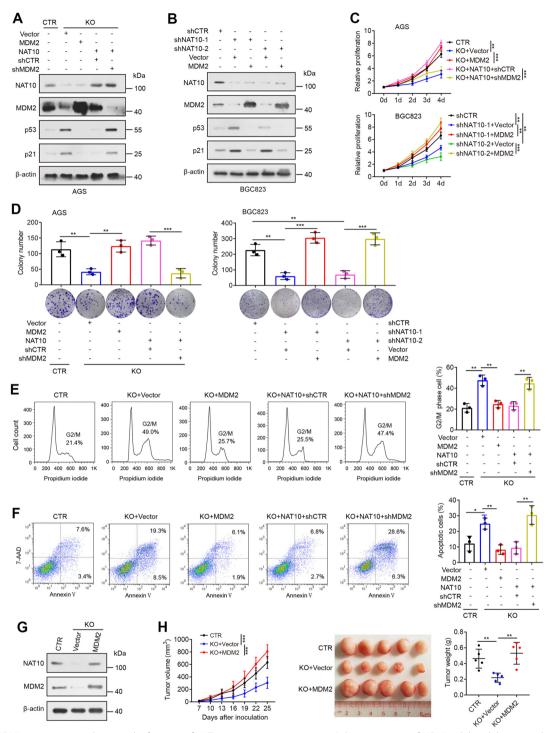


Fig. 4 MDM2 is a major contributor to the function of NAT10 in gastric carcinogenesis. **A** Overexpression of MDM2 inhibited the upregulation of p53 and p21 proteins in NAT10-knockout AGS cells, while knockdown of MDM2 efectively reversed the inhibitory efect of NAT10 overexpression on p53 and p21. **B** MDM2 overexpression reversed the upregulation of p53 and p21 proteins by NAT10 knockdown in BGC823 cells. **C** and **D** The efects of NAT10 depletion on cell proliferation (**C**) and colonic growth (**D**) were rescued by transfection with MDM2, whereas cell proliferation and colonic growth of NAT10-overexpressing cells were prevented by knockdown of MDM2. **E** and **F** Cell cycle (**E**) and apoptosis (**F**) were measured in the indicated cells by fow cytometry. **G** MDM2 and NAT10 proteins were evaluated in NAT10-knockout AGS cells stably expressing MDM2 or vector control. **H** MDM2 overexpression rescued the impaired capacity of tumor growth triggered by NAT10 knockout (*n*=5 mice/group). Error bars, SD. **P*<0.05, ***P*<0.05, ***P*<0.001, ****P*<0.001 using a two-tailed t-test