scientific reports

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# Author Correction: MiR-99b-5p and miR-203a-3p Function as Tumor Suppressors by Targeting IGF-1R in Gastric Cancer

Published online: 17 January 2023

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Correction to: *Scientific Reports* https://doi.org/10.1038/s41598-018-27583-y, published online 04 July 2018

This Article contains an error in Figure 4(E), where β-actin should be the same as in Figure 2(F).

The correct Figure 4 and accompanying legend appear below.

This change does not affect the conclusions of the Article.

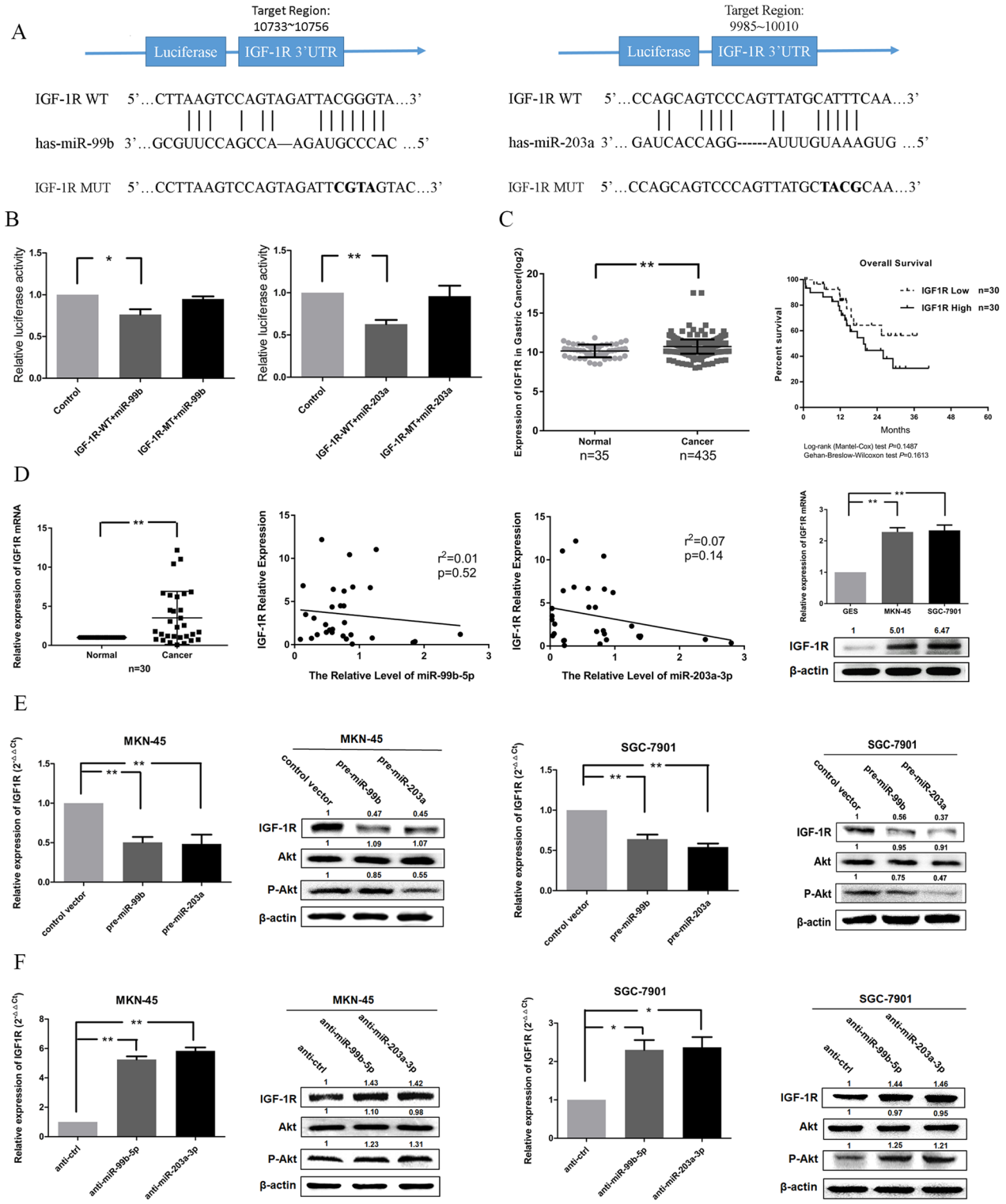


**Figure 4.** IGF-1R is experimentally validated as a co-target of miR-99b-5p and miR-203a-3p in GC cells. (**A**) Putative miR-99b-5p/203a-3p-binding sites in the IGR-1R 3′UTRs, mutations were generated in the IGF-1R 3′UTR sequences by mutating 4 nt for the seed region of miR-99b-5p/203a-3p, as indicated. (**B**) Dual luciferase assays were performed in HEK293 cells after co-transfection with the wild-type or mutant IGR-1R 3′-UTR plasmids and pre-miR-99b/203a. (**C**) The TCGA data of IGF1R mRNA expression in GC tissues (n = 35) and normal tissues (n = 435). Overall survival analysis showed that there was no statistically significant between IGF1R high expression and low expression tumors. (**D**) IGF-1R was determined by qRT-PCR in GC tissues (left). The correlation between miR-99b-5p/203a-3p and IGR-1R was analyzed. IGF-1R was determined by qRT-PCR and western blot in GC cell lines (right). β-actin was employed as a housekeeping control. (**E**,**F**) IGF-1R expression level was measured by qRT-PCR and western blot after transfection with pre-miR-99b/203a and anti-miR-99b-5p/203a-3p in MKN-45/SGC-7901 cells (*$P < 0.05$, **$P < 0.01$, Student's t test or Mann-Whitney test).