Erratum

The following was originally published in Volume 25, No. 6, pages 967–974, 2017 (doi: https://doi.org/10.3727/09 6504016X14803476672380). Because it was recently revealed that the testing reagent supplier used had some inferior products, we wanted to repeat the testing and check the reliability of the data. As a result, some of the expression levels were revealed to be different from the experimental results, which affected some of the display in the figures. Therefore, corrected versions of Figures 1, 2, 3, and 4 are provided here. The figures have also been replaced with the corrected versions in the original published version in the online site (https://www.ingentaconnect.com/contentone/cog/ or/2017/00000025/00000006/art00013). These corrections do not affect the conclusion of the original article.

miR-107 Promotes Proliferation and Inhibits Apoptosis of Colon Cancer Cells by Targeting Prostate Apoptosis Response-4 (Par4)

Fen Liu,*† Shaojun Liu,* Feiyan Ai,*† Decai Zhang,*† Zhiming Xiao,* Xinmin Nie,‡ and Yunfeng Fu§

*Department of Gastroenterology, The Third Xiangya Hospital of Central South University, Changsha, Hunan, P.R. China †Hunan Key Laboratory of Nonresolving Inflammation and Cancer, Changsha, Hunan, P.R. China
‡ Clinical Laboratory of The Third Xiangya Hospital of Central South University, Changsha, Hunan, P.R. China §The Third Xiangya Hospital of Central South University, Changsha, Hunan, P.R. China

Colorectal cancer (CRC) is one of the most common malignancies in the world, with a high incidence and a high mortality. However, the pathogenesis of CRC carcinogenesis is still unexplored. In this study, we investigated the role of miR-107 in the regulation of CRC cell proliferation and apoptosis. First, the expression of miR-107 was observed to be aberrantly increased in human CRC tumor tissues and cell lines when compared to the colonic control tissues and colon epithelial cells. Further study showed that the proliferative and apoptotic capacities of human CRC SW480 and LoVo cells were aberrantly regulated by miR-107. The proliferation of SW480 and LoVo cells was remarkably enhanced by the miR-107 mimic but suppressed by the miR-107 inhibitor when compared to the negative control. On the contrary, the apoptotic rate of both SW480 and LoVo cells was significantly inhibited by miR-107 overexpression but increased by miR-107 inhibition. In addition, we identified prostate apoptosis response-4 (Par4) as a direct target of miR-107 with a potential binding site on the 3 -UTR of mRNA, as evaluated by bioinformatics prediction and luciferase reporter assay. Par4 expression levels were significantly inhibited by the miR-107 mimic but upregulated by the miR-107 inhibitor in both SW480 and LoVo cells. Compared to the control, the increase in Par4 expression significantly inhibited the induction role of miR-107 in the proliferation of SW480 and LoVo cells, and the apoptotic rate of cells repressed by the miR-107 mimic was also reversed by Par4 overexpression. In summary, our results demonstrated that miR-107 exerts a positive role in the survival of CRC cells by directly targeting Par4. This might reveal a novel understanding about human CRC pathogenesis.

Key words: miR-107; Prostate apoptosis response-4 (Par4); Colorectal cancer (CRC); Proliferation; Apoptosis

Address correspondence to Xinmin Nie, The Third Xiangya Hospital of Central South University, No. 138 Tongzipo Road, Yuelu District, Changsha 410013, Hunan Province, P.R. China. Tel: +86-0731-88638888; E-mail: niexinmin7440@sina.com *or* Yunfeng Fu, The Third Xiangya Hospital of Central South University, No. 138 Tongzipo Road, Yuelu District, Changsha 410013, Hunan Province, P.R. China. Tel: +86-0731-88638888; E-mail: fuyfeng427@163.com





Figure 1. miR-107 expression is increased in CRC tumor tissues and cells. (A) The miR-107 expression was examined in human CRC tumor tissues as well as in adjacent colonic tissues by RT-PCR (p < 0.001 vs. adjacent colonic tissues). (B) miR-107 expression was measured in human CRC cell lines (SW480, HCT-116, and LoVo) as well as in the normal colon epithelial cell line NCM460 (**p < 0.01 vs. NCM460).





Article(s) and/or figure(s) cannot be used for resale. Please use proper citation format when citing this article including the DOI publisher reference, volume number and page location



Figure 3. Par4 is the target of miR-107. (A) The potential binding sites of miR-107 in the 3 -UTR of Par4 mRNA. (B) Luciferase activities were measured in SW480 and LoVo cells after transfection by wild Par4-3 -UTR (Par4-wt) or mutant Par4-3 -UTR (Par4-mut). The expression of Par4 in SW480 and LoVo cells was examined by RT-PCR assay (*p < 0.05, **p < 0.01 vs. control).



Figure 4. Par4 is involved in the miR-107-mediated regulation of CRC cell survival. SW480 and LoVo cells were transfected with Par4 plasmid or plasmid control. (A) The expression levels of Par4 were tested by RT-PCR assay in both CRC cells. Cell proliferation (B) and apoptosis (C) were then measured in SW480 and LoVo cells transfected with the Par4 plasmid in the presence or absence of the miR-107 mimic (*p < 0.05 vs. control).

Delivered by Ingenta IP: 128.14.159.178 On: Tue, 21 Jun 2022 03:42:35 Article(s) and/or figure(s) cannot be used for resale. Please use proper citation format when citing this article including the DOI, publisher reference, volume number and page location.