72P Up-regulation of miR-1266-5p suppressed hTERT expression and telomerase activity in cancer cell lines

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Background: Telomerase is in charge of telomere extending and is triggered in around 90% of cancers. hTERT is the controlling subunit of telomerase and plays a critical role in the activation of telomerase. The mechanism through which hTERT regulate the invasion and metastasis of cancer is unclear. miRNAs can regulate the expression of hTERT. It was previously reported that miR-1266 can target hTERT in gastric cancer. In this study, we have made the first report of miR-1266-5p role on the hTERT expression, telomerase activity, and biological functions, including cell proliferation and cell cycle in AGS, MCF7, A375, and HepG2 cell lines.

Methods: AGS, MCF7, A375, and HepG2 cell lines were transfected with miR-1266-5p mimic and inhibitor reagents. The Expression levels of miR-1266-5p, hTERT, and transfection efficiency were analyzed by Taqman qRT-PCR. The cell proliferation and cell cycle changes were detected by MTT Calorimetric Assay and flow cytometry, respectively. Also, Quantitative TRAP Assay was used to detect telomerase activity.

Results: The expression of miR-1266-5p significantly was increased after transfection by mimic compared to control cells. While its expression was decreased by the inhibitor. Upregulated miR-1266-5p significantly decreased cell growth, although inhibitor promoted cell proliferation. This finding was confirmed by cell cycle analysis, as upregulation of miR-1266-5p induced cells cycle arrest at the transition of G1 to S phase and led to G0/G1 entry, while the downregulation of miR-1266-5p promoted cell growth and led to G2/M entry. Concordantly, the overexpression of miR-1266-5p resulted in down-regulated hTERT expression and also suppressed telomerase activity.

Conclusions: Taken together, the findings showed that miR-1266-5p acts as hTERT and telomerase activity suppressor. miR-1266-5p could also decrease cell proliferation and induce cell cycle arrest, while its inhibitor eliminates miR-1266-5p effects. Thus, upregulation of miR-1266-5p may be considered as a novel therapeutic target in cancer.

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