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Downregulation of miR-24 Could Restore the Normal Splicing of the Oncogenes in Breast Cancer

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Abstract

Introduction Global incidence of breast cancer according to the National Cancer Institute highlights the importance of focusing as much as possible on developing treatment strategies. microRNAs (miR) are emerging as biomarkers and potential therapeutic targets in tumor management. Human Cleavage Factor Im (CFIm) is an essential component of the pre-mRNA processing complex that functions by the regulation of poly (A) site selection through the recognition of UGUA sequences upstream of the poly (A) site. Recently, CFIm25 has been identified as a broad repressor of proximal poly (A) site usage that, when depleted, increases cell proliferation. In this area, we have previously suggested the possible role of some oncogenic microRNAs including miR-24 in regulation of tumor suppressor gene CFIm 25. So, the current study aimed to investigate the influence of miR-24 downregulation on CFIm 25 expression level in human breast cancer cell line and its proliferation.
Materials and Methods: Human breast cancer cell line of MDA was infected with lentiviruses containing either anti-miR-24 precursor sequences. The RNA expression level of miR-24 and CFIm25 were estimated in MDA infected cells by QRT-PCR. Protein level of CFLm25 genes in human MDA were also analyzed by western blotting.
Results: Real-time PCR showed that miR-24 knockdown enhanced CFIm25 expression level in MDA treated cells. Western blotting results also confirmed that downregulation of miR-24 in MDA cell lines dramatically enhanced CFIm25 expression.
Conclusions: Overall, this is the first study which evidently indicates miRNAs correlation with CFIm25 expression in breast cancer cells. We indicated that miR-24 overexpression might be one of the factors contributing to the cell proliferation in breast cancer through CFIm25 down-regulation. So, our data suggests that oncogenic role for selected miRNA and presents a rationale for the down regulation of this miRNA as a novel strategy to improve treatment response in breast cancer.