

Functional effect of miR-1307-3p on breast cancer progression

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Functional effect of miR-1307-3p on breast cancer progression

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Background: MiRNAs are non-coding RNA molecules and its function is the regulation of gene expression. In cancer, the deregulation of miRNAs allows them to act as oncogenes or tumor suppressors. From an analysis of the expression of miRNAs in breast cancer (BC) in The Cancer Genome Atlas (TCGA), it was identified that miR-1307-3p is significantly overexpressed in the tumor tissue compared to healthy tissue from patients. So far, in BC, it has only been reported that this miRNA inhibits SMYD4 and that it is involved in resistance to cisplatin through its effect on Mdm4. In this project we propose to identify the role of miR-1307-3p in proliferation, migration, invasion, angiogenesis, and possible targets involved in these processes in BC cells.

Methods: RT-qPCR was used to evaluate basal levels of miR-1307-3p in the BC cell lines MDA-MB-231 and MCF-7, and the human epithelial breast MCF-10A cells. Later, we determined the effect of miR-1307-3p on proliferation, migration, and invasion in MDA-MB-231 and MCF-7, and angiogenesis in the HUVEC endothelial cells. All assays were carried out using the miR-1307-3p inhibitor. Then, nine miRNA-target prediction databases were analyzed to identify potential miR-1307-3p target genes, and their expression was analyzed by RT-qPCR in a designed 384-well plate. Finally, the targets that presented an alteration in their expression were evaluated by western blot.

Results: We found that miR-1307-3p is overexpressed in MDA-MB-231 and MCF-7, compared to MCF-10A cells. We also identified that transfection with the miR-1307-3p inhibitor causes a significant decrease in the processes of proliferation, migration, invasion, and angiogenesis, when compared with untreated or negative control transfected cells. For its part, prediction databases analysis allowed us to identify 19 potential targets of miR-1307-3p. We also found that 2 genes were overexpressed, CIC and PRM2. Finally, we found an overexpression of PRM2 protein.

Conclusions: MiR-1307-3p is overexpressed in BC cells. Furthermore, miR-1307-3p induces the processes of proliferation, migration and invasion in BC cells, and angiogenesis in HUVEC cells. These observations suggest that miR-1307-3p can acts as an onco-miRNA. In addition, a potential new target of miR-1307-3p was found, PRM2 which has not been previously reported in breast cancer. Further analysis to verify and validate the implication of this miR-1307-3p target are needed to understand its importance in BC.