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ROLE OF MIR-26A IN FOLLICULAR THYROID CARCINOMA

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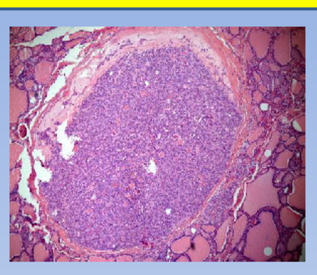
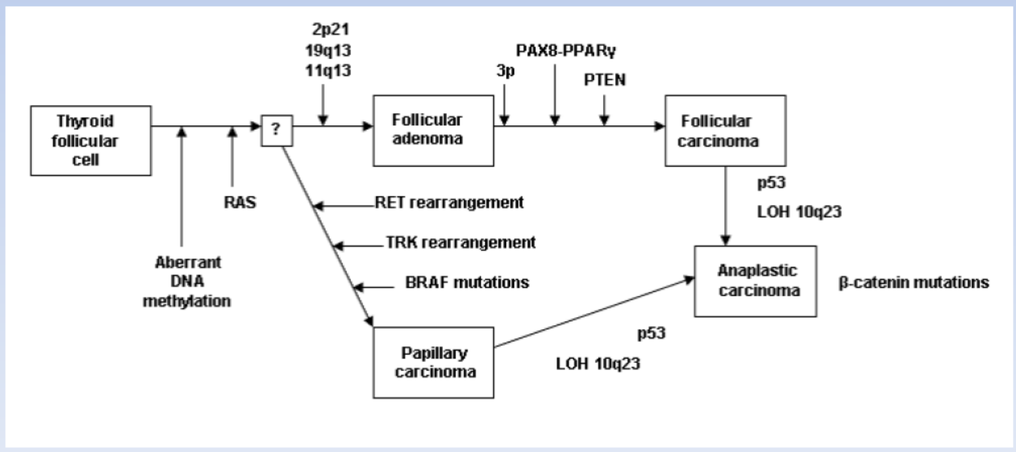
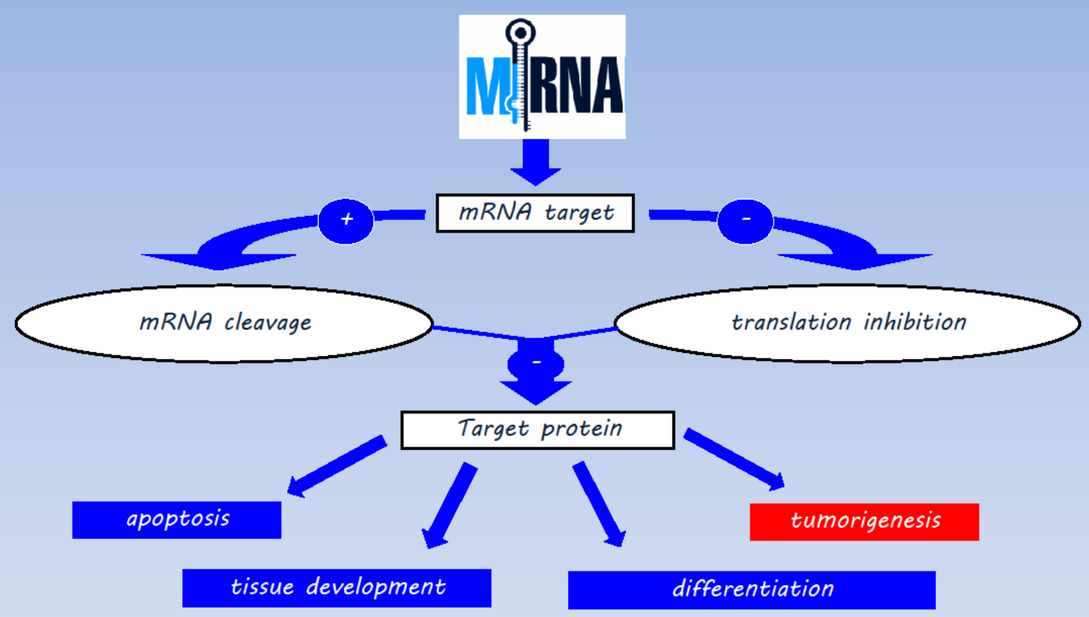
INTRODUCTION



Follicular thyroid carcinoma (FTC) is the second most common thyroid malignant epithelial tumor. It is clinically asymptomatic, usually represented by a single nodule, less often multiple, with origin from thyroid follicular epithelial cells. Despite its well-differentiated characteristics, FTC may develop distant metastases through hematogenous dissemination.
The molecular alterations involved in the pathogenesis of follicular neoplasia are not completely known.



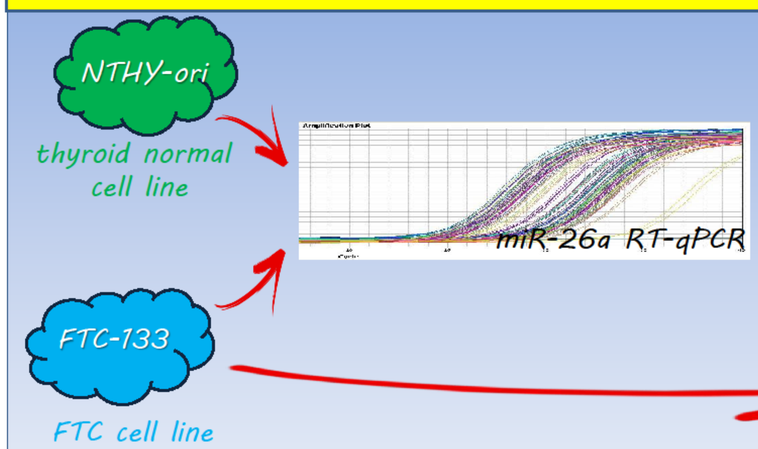


Recent studies have demonstrated the importance of miRNAs, small non-coding nucleic acids that regulate gene expression at post-transcriptional level. It is well known that the expression profile of miRNAs is altered in follicular thyroid carcinoma. To date, the aftermaths of deregulated expression of miRNAs in the FTC have not been clarified.

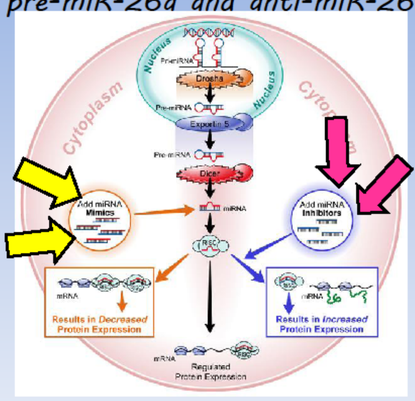
AIM

In order to investigate the molecular mechanisms involved in thyroid tumorigenesis, we evaluated the effects of miR-26a modulation in a human FTC cell line

MATERIAL AND METHODS

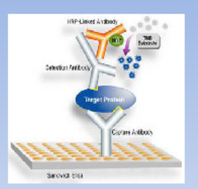


Transfection with pre-miR-26a and anti-miR-26a

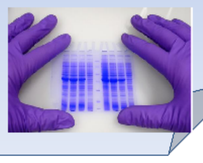


M	Mock
P	premiR-26a
A	antimiR-26a
cP	premiR control
cA	antimiR control

VEGF secretion

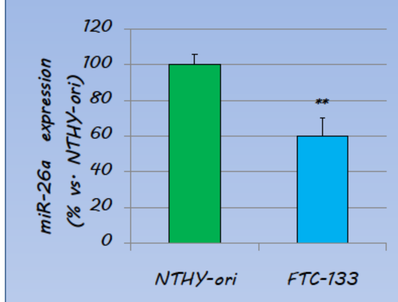


Western blot



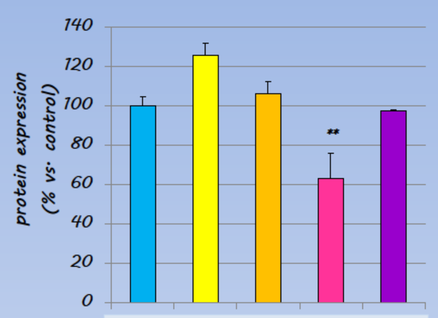
RESULTS

miR-26a expression



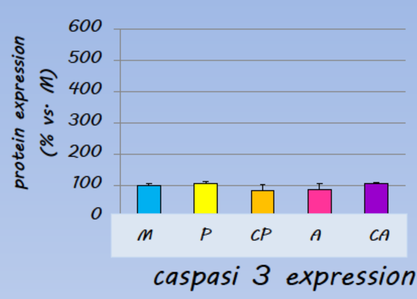
miR-26a is significantly down-regulated in FTC-133 cells as compared to the thyroid normal cell line, NTHY-ori

PRKCD expression

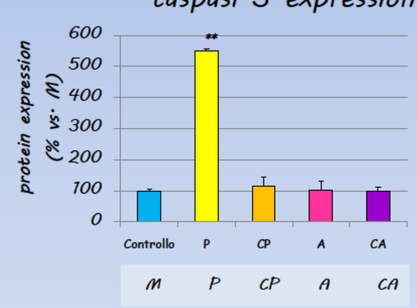


miR-26a up-regulation increases protein kinase C delta (PRKCD) levels. PRKCD is a regulator of caspase-mediated apoptosis, VEGF-mediated cell proliferation and iodide uptake via NIS.

procaspase 3 expression

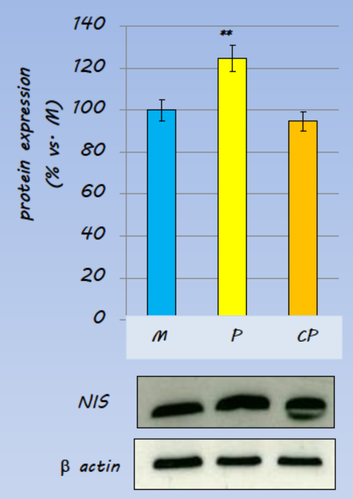


caspasi 3 expression



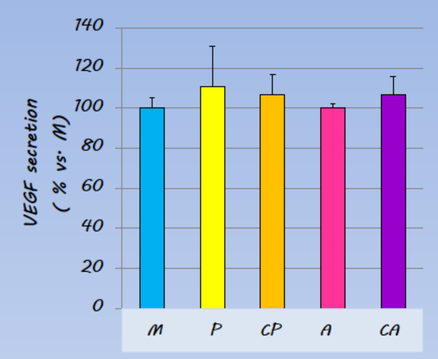
miR-26a up-regulation increases caspase 3 protein levels

NIS expression



miR-26a up-regulation increases sodium-iodide symporter protein levels

VEGF secretion



miR-26a modulation does not influence vascular endothelial growth factor secretion

** = p<0,01 vs. M

CONCLUSIONS

These results support the hypothesis that miR-26a may influence thyroid differentiation processes and may represent a therapeutic target for future innovative therapy in advanced radio-refractory disease.

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