ROLE OF MIR-26A IN FOLLICULAR THYROID CARCINOMA

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.

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

RESULTS

miR-26a expression

PRKGD expression

protease 3 expression

NIS expression

VEGF secretion

miR-26a is significantly downregulated in FTC-133 cells as compared to the thyroid normal cell line, Nthy-ori.

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

miR-26a up-regulation increases protease 3, a protein catabolic enzyme.

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

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

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.