#158 - THE ROLE OF KCNQ1OT1 IN GASTRIC CANCER PATHOGENESIS: CERNA NETWORKS AND DNA HYPERMETHYLATION

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Introduction. Gastric cancer (GC) is a multifactorial disease in which the environment interacts with genetic factors to promote disease development. GC progression is characterized by the Correa cascade, a set of well-defined histological lesions. In the stomach, cellular senescence is induced by H. *pylori*, which promotes a pro-tumoral microenvironment. Our previous work indicated that KCNQ1OT1 is overexpressed in GC and is associated with poorer survival. To date, the role of KCNQ1OT1 in GC remains unclear.

Aims. To characterize the role of KCNQ1OT1 in GC pathogenesis through competing endogenous RNA (ceRNA) networks and DNA-hypermethylation of the Retinoblastoma transcriptional corepressor 1 (RB1) gene.

Methods. Gene expression datasets were downloaded from The Cancer Genome Atlas (TCGA) repositories. To construct ceRNA networks, Diana-Tools software was used. *In vitro,* studies were performed using tumor cell lines (KATOIII, NCI-N87, and HS746t) and normal gastric cells GES1. Transcript expression was measured by RT-PCR.

Results.*In silico* analyses predicted ceRNA networks to exist between KCNQ1OT1 and 10 miRNA and higher DNA-methylation levels of the RB1 gene were observed in tumor samples. *In vitro*, studies show that KCNQ1OT1 expression can be induced by *H. pylori* in GES1 cells. The enhanced expression of KCNQ1OT1 was also observed in cancer cell lines and correlated with the upregulation of RB1 transcripts.

Conclusions. Our results identify KCNQ1OT1 as a novel component of ceRNAs networks that promotes cell proliferation and may thereby contribute to GC progression. Future efforts are required to validate this possibility.