PalStudent Journal
A Palestinian Scientific Journal for the Youth

ABSTRACTS: VOLUME 7, SPECIAL ISSUE {7th Undergraduate Conference}

ABSTRACT

Investigating The Regulation of MIR503HG and POT1-AS1 Expression in Breast Cancer Cells by Microenvironmental Cues: Hypoxia, Oxidative Stress, Serum Starvation, and HGF

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Long non-coding RNAs (lncRNAs) are increasingly recognized as key regulators within the tumor microenvironment, particularly in how cancer cells adapt to stressful conditions such as hypoxia, starvation, and oxidative stress through transcriptional reprogramming. However, the expression patterns of many lncRNAs, including MIR503HG isoform 2 and POT1-AS1, remain poorly understood under these conditions. This study aimed to investigate the expression of these two lncRNAs in MDA-MB-231 triple-negative breast cancer (TNBC) cells exposed to serum starvation, H2O2-induced oxidative stress, hepatocyte growth factor (HGF) stimulation, and hypoxia. A semi-quantitative PCR approach was used, followed by gel electrophoresis and densitometric analysis using ImageJ software to quantify band intensities.

Expression of MIR503HG isoform 2 was normalized to β-actin and further compared to the corresponding untreated controls. The results revealed that MIR503HG isoform 2 was notably upregulated under hypoxia, HGF stimulation, and serum starvation, suggesting its possible involvement in TNBC stress adaptation, potentially supporting tumor survival or progression. In contrast, amplification of POT1-AS1 was unsuccessful due to technical issues linked to the primer sequences obtained from previous literature, highlighting the need for further optimization.

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Overall, the findings identify MIR503HG isoform 2 as a stress-responsive lncRNA that may play a significant role in tumor adaptation mechanisms, warranting further investigation, while also underscoring the importance of accurate primer validation in lncRNA expression studies.

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