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ABSTRACT

Upregulation of MIR4500HG003 LncRNA Expression in Response to Hypoxia, HGF, and Serum Starvation in the Triple-Negative Breast Cancer Cell Line MDA-MB-231

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Background: Cancer remains a leading cause of global mortality, with triple-negative breast cancer (TNBC) representing one of its most aggressive and difficult-to-treat subtypes. TNBC lacks expression of estrogen receptors (ER), progesterone receptors (PR), and HER2, eliminating the possibility of targeted hormonal therapies. As a result, it is associated with a high rate of early metastasis and poor clinical outcomes. Emerging research has identified long non-coding RNAs (lncRNAs) as key regulators of tumor progression, metastasis, and therapy resistance. One such lncRNA, MIR4500HG003, was recently reported to promote TNBC metastasis via the miR-483-3p/MMP9 axis, yet this is the only study that has investigated it to date. Beyond this single publication, nothing is known about how MIR4500HG003 is regulated in response to tumor microenvironmental stressors, which are critical for metastatic adaptation.

Problem Statement: The molecular mechanisms regulating MIR4500HG003 expression remain unknown. Without understanding how it responds to environmental cues such as hypoxia, nutrient deprivation, oxidative stress, or growth factor stimulation, we lack critical insight into its potential role in TNBC pathophysiology and metastasis.

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Purpose: This study investigates the expression of MIR4500HG003 in MDA-MB-231 TNBC cells under microenvironmental conditions: hypoxia, serum starvation, HGF stimulation, and oxidative
stress (H2O2).

Methods: MDA-MB-231 cells were cultured under different stress conditions. Semi-quantitative RT-PCR was performed to amplify MIR4500HG003, with β-actin as a housekeeping control. Gel electrophoresis and ImageJ software were used to analyze and quantify band intensity.

Results: MIR4500HG003 expression was significantly upregulated under hypoxia, HGF stimulation, and serum starvation, but showed no change under oxidative stress.

Conclusion: This study represents only the second investigation of MIR4500HG003 and provides novel insights into its regulation by key tumor microenvironmental stressors. Our findings suggest that MIR4500HG003 may act as a stress-responsive lncRNA that plays a role in TNBC progression and metastasis, highlighting it as a promising candidate for future functional and therapeutic research.

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