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LncRNA FER1L4 suppresses cancer cell proliferation and cycle by regulating PTEN expression in Endometrial Carcinoma

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Running title: FER1L4 inhibits EC development via PTEN/Akt pathway

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Abstract

Background: Dysregulated long non-coding RNAs (lncRNAs) might exert key roles in pathways associated with endometrial carcinoma (EC) development. This study aims to investigate the new role of lncRNA FER1L4 in EC pathogenesis due to its correlation with phosphatase and tensin homolog (PTEN), one important indicator of EC progression.

Methods: Real time PCR was performed to detect the expression of FER1L4 in thirty paired EC samples and two EC cell lines. Plasmid containing FER1L4 was transfected into HEC-50 cells with a relative lower level of FER1L4 expression, followed which PTEN expression and Akt phosphorylation were measured by western blotting. Cell proliferation was analyzed through MTT and colony-formation assays, while cell cycle and apoptosis were determined by flow cytometry.

Results: FER1L4 showed significantly downregulation in EC tissues compared to control, which was positively correlated with decreased PTEN expression. Moreover, FER1L4 could promote PTEN expression and inhibit Akt phosphorylation. Additionally, a significant decrease of cell proliferation was observed in FER1L4 overexpressing cells, along with cell cycle arrest at G0/G1 phase and increased proportion of apoptotic cells.

Conclusion: FER1L4 not only showed downregulation in EC tissues and cells, but also regulated PTEN expression and Akt signaling, which might contribute to its inhibition on cell proliferation. This study might provide a new potential therapeutic target for EC treatment.

Keywords: Endometrial carcinoma; Cell proliferation; Cell cycle; FER1L4; PTEN

1. Introduction

Endometrial carcinoma (EC) is one of the most common malignancies occurring in female genital system, accounting for 3.6% of all new cancer cases in the United States [1]. Classically, EC can be classified into two main types based on pathological and demographic characteristics: Type I

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