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Junctional adhesion molecule-like protein promotes tumor progression via the Wnt/β-catenin signaling pathway in lung adenocarcinoma

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Abstract

Background: Lung adenocarcinoma (LUAD) is a heavy social burden worldwide. Because the mechanisms involved in LUAD remain unclear, the prognosis of LUAD remains poor. Consequently, it is urgent to investigate the potential mechanisms of LUAD. Junctional adhesion molecule-like protein (JAML), is recognized as a tumorigenesis molecule in gastric cancer. However, the role of JAML in LUAD is still unclear. Here we aimed to evaluate the role of JAML in LUAD.

Methods: qRT-PCR, Western blotting and immunohistochemistry were conducted to investigate the expression of JAML in LUAD tissues. JAML was knocked down and overexpressed in LUAD cells using transient transfection by siRNA and plasmids or stable transfection by lentivirus. Proliferation potential of LUAD cells were detected by Cell Counting Kit-8, EdU incorporation and Colony formation assay. Migration and invasion abilities of LUAD cells were determined by wound healing, transwell migration and invasion assays. Cell cycle and cell apoptosis were detected by flow cytometry. The effects of JAML in vivo were studied in xenograft tumor models. Western blotting was used to explore the molecular mechanisms of JAML function. In addition, rescue experiments were performed to verify the possible mechanisms.

Results: JAML expression was elevated in LUAD tissues compared with peritumor tissues, and this upregulation was positively related to pT and pTNM. Furthermore, both in vitro and in vivo, JAML silencing markedly repressed malignant behaviors of LUAD cells and vice versa. Knockdown of JAML also mediated cell cycle arrest at G_0/G_1 phase and promoted apoptosis in LUAD cells. Mechanistically, silencing JAML repressed the process of epithelial-mesenchymal transition by inactivating the Wnt/β-catenin pathway in LUAD cells. Effects of JAML can be rescued by Wnt/β-catenin pathway activator in A549 cells.

Conclusions: Our data reveal the oncogenic role of JAML in LUAD, indicating that JAML may be a predictive biomarker and novel therapeutic target for LUAD.

Keywords: Junctional adhesion molecule-like protein, Lung adenocarcinoma, Invasion, Migration, Tumor progression

Introduction

Lung cancer is an extremely lethal disease worldwide [1]. Lung adenocarcinoma (LUAD), accounting for approximately 40% of all lung cancers, is the most common cancer [2]. Although important progress has been made in drug therapy, especially targeted drugs and

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