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miR-25 promotes gastric cancer cells growth and motility by targeting RECK

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Abstract Gastric cancer (GC) is the second leading cause of cancer-related death worldwide. Recently, accumulating evidence suggests that microRNAs (miRNAs) play prominent roles in tumorigenesis and metastasis. Here, we confirmed that miR-25 was significantly increased in human GC tissues and cell lines. Forced expression of miR-25 remarkably enhanced cell proliferation, migration, and invasion in GC cells, whereas inhibition of miR-25 by inhibitor caused significant suppression of proliferation and significant increase of apoptosis. Moreover, inhibition of miR-25 significantly decreased migration and invasion of GC cells. Finally, reversion-inducing-cysteine-rich protein with kazal motifs (RECK) was found to be a target of miR-25. Overexpression of RECK could significantly reverse the oncogenic effect of miR-25. Taken together, miR-25 might promote GC cells growth and motility partially by targeting RECK.

Keywords miR-25 · RECK · Gastric cancer · Proliferation · Migration · Invasion

Introduction

Gastric cancer (GC), the second most common cause of cancer-related death worldwide, accounts for approximately 10 % of all cancer-related death and claims approximately 700,000 lives annually [1]. GC is a complex genetic disorder, and previous studies have identified that several genes, known as oncogenes or tumor suppressors, were related to the progression of GC, but the detailed molecular mechanisms of GC remain poorly understood [2].

MicroRNAs (miRNAs) are a new class of short, non-coding RNAs that suppress target gene expression by binding to the 3′-untranslated region (3′-UTR) to induce translational repression or mRNA decay [3]. Aberrant miRNA expression has also been frequently reported in numerous tumors such as breast cancer, colorectal cancer, GC, and lung cancer [4–8]. In the recent years, emerging evidence suggests that miRNAs play essential roles in tumor cell biological processes, including cell proliferation, differentiation, migration, and invasion [9–11].

miR-25 has been reported to be aberrantly overexpressed in several tumors. For example, Xu et al. [12] reported that expression of miR-25 was remarkably elevated in esophageal squamous cell carcinoma (ESCC) and contributed to the malignant phenotype by blocking expression of E-cadherin. Zhang et al. [13] found that miR-25 was highly expressed in ovarian cancer and found that miR-25 inhibited apoptosis of ovarian cancer cells by targeting Bim. miR-25 was also reported to be overexpressed in cholangiocarcinoma, which promoted apoptosis resistance in cholangiocarcinoma by targeting TNF-related apoptosis inducing ligand (TRAIL) death receptor-4 [14]. miR-25 was also found to be significantly elevated in GC [15, 16]. However, the role and relevant pathway of miR-25 in GC carcinogenesis is largely unknown.

In the present study, the effects of miR-25 on proliferation, migration, and invasion abilities were investigated. Moreover, reversion-inducing-cysteine-rich protein with kazal motifs (RECK), a tumor suppressor gene, was

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