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Research Article

LncRNA PVT1 Promotes Cell Proliferation, Invasion, and Migration and Inhibits Cell Apoptosis by Phosphorylating YAP

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Gastric cancer (GC) as a serious global health problem is a threat to human longevity. Plasmacytoma variant translocation 1 (PVT1) participates in the formation and progression of various cancers, including GC. The aim of this study is to investigate the mechanism underlying the functions of PVT1 and explore a novel target for the diagnosis and treatment of GC. Analysis of the TCGA dataset using the R software identified that the lncRNA PVT1 was greatly upregulated in GC tissues. Twenty pairs of GC and adjacent normal tissues were acquired from patients with GC, and the expression of PVT1 was evaluated using RT-qPCR. Furthermore, PVT1 expression was knocked down in GC cells using siRNA, and the GC cells were divided into control, negative control (NC), and siRNA groups. Cell proliferation ability was analyzed using Cell Counting Kit-8 (CCK8) and colony formation assays, whereas cell migration and invasion ability were investigated through wound healing and Transwell assays. Moreover, Western blotting was used to analyze the expression of Yes-associated protein (YAP) and epithelial-to-mesenchymal transition (EMT) proteins. We also found that PVT1 and YAP expressions were upregulated in the GC tissues compared with those in the adjacent nontumor tissues. Knockdown of PVT1 was found to inhibit the proliferation, invasion, and migration and promote apoptosis of GC cells. Furthermore, knockdown of PVT1 downregulated YAP and promoted phosphorylation of YAP, suggesting that PVT1 exerts actions on GC cells by targeting YAP and inhibits cell apoptosis in vitro. The EMT process was also inhibited by the knockdown of PVT1. In summary, lncRNA PVT1 facilitated cell proliferation, invasion, and migration and suppressed cell apoptosis by targeting YAP. This study suggests that the expressions of PVT1 and YAP could be used for the early detection of GC and the occurrence and development of GC could be inhibited by interfering the interaction of PVT1 and YAP, which will provide new insights for the diagnosis, treatment, and prognosis of GC.

1. Introduction

Gastric cancer (GC) as a severe global health problem is one of the major threats to global health and human longevity [1]. As the fifth most prevalent type of malignant tumor, GC has become the third leading cause of cancer-related death all over the world [2]. Owing to the advancement of endoscopic diagnosis and treatment technology, many early-stage gastric cancers can be detected and resected using endoscopic submucosal dissection. However, early diagnosis may not always be possible because of the insidious symptoms in the early stage of the disease [3]. Hence, most patients are diagnosed with advanced gastric cancer with metastasis when detected [4]. Despite the advances in treatment strategies, the overall prognosis of GC is poor due to tumor recurrence and metastasis [5]. Hence, it is crucial to find out the molecular biological mechanism underlying the progression of the disease and explore therapeutic targets for early diagnosis and treatment of GC.

Long noncoding RNAs (lncRNAs) are transcripts over 200 nucleotides in length with no or low protein-coding potential [6–8]. Abnormalities in lncRNAs may lead to the formation of tumors by promoting cell proliferation, invasion, and metastasis and inhibiting apoptotic mechanisms of cells [9]. Thus, detecting changes in lncRNAs in the early stages of tumor formation and development is essential for