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Research Article

miR-559 Inhibits Proliferation, Autophagy, and Angiogenesis of Hepatocellular Carcinoma Cells by Targeting PARD3

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Hepatocellular carcinoma (HCC) is one of the most common cancers in the world and has a high mortality rate. Although prevention and treatment of HCC has improved, it still faces poor prognosis and high mortality. miRNAs play a critical role in the tumorigenesis of HCC, but the underlying mechanism has not been well investigated. Here, the functions and interaction between miR-559 and PARD3 were investigated in HCC cells. Increased PARD3 and decreased miR-559 expression were observed in HCC cells compared with those in normal liver cells, especially in Huh-7 cells. Studies further demonstrated that PARD3 silencing or miR-559 overexpression impaired the proliferation, autophagy, and angiogenesis in Huh-7 cells. Mechanistically, PARD3 represents a target of miR-559. Furthermore, investigations revealed that miR-559 inhibition induced the expression of PARD3, thereby enhancing cell proliferation, autophagy, and angiogenesis in Huh-7 cells. These results reveal the interaction between miR-559 and PARD3 in HCC cells and provide new insights into their potential targets as therapeutic treatment against HCC.

1. Introduction

Hepatocellular carcinoma (HCC) is one of the most common malignancies in the world [1]. Although increasing studies are focusing on HCC, the underlying mechanisms about its tumorigenesis and development are still unclear. Since the proliferation and metastasis of tumor cells depend upon the formation of angiogenesis, the progress of neovascularization is actually important for cell proliferation, invasion, and metastasis in solid tumors [2]. Currently, a large number of drugs targeting angiogenesis have been approved for the first- and second-line treatment of HCC [3].

Autophagy is a highly regulated catabolic process in cells. Under normal or stress conditions, autophagy participates in the removal of damaged organelles and the transformation of intracellular substances to maintain homeostasis [4, 5]. Dysregulation of autophagy has serious consequences and is related to the development and progression of various diseases, such as infectious neurodegenerative and metabolic diseases, as well as cancer [6]. Moreover, studies have shown that autophagy may be involved in maintaining the occurrence of HCC [7]. In the early stage in the development of HCC, autophagy is against tumor formation by inhibiting inflammation and maintaining genomic stability. Once cancer develops, autophagy may act as a prosurvival mechanism to protect HCC cells from death induced by different types of stimulation, including oxidative stress, and thus maintain cancer progression [8].

miRNAs are short noncoding RNAs of 19-25 nucleotides in length that inhibit target gene expression by specifically binding in a sequence-specific manner to the 3′-UTR regions of the target gene [9]. Abnormal expression of miRNAs is associated with the development of most tumors, such as HCC, and involved in the regulation of tumor cell growth, autophagy, and angiogenesis [10]. miR-559 is one of these miRNAs, which has been demonstrated to be involved in the inhibition of cell proliferation and invasion in HCC cells [11]. PARD3 (zonal defect 3