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Development of Esophageal Carcinoma by Targeting Specific Genes

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Abstract

As one of the common malignant tumors in the world, the esophageal carcinoma can be grouped in two categories based on its pathological features, i.e. esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma.

Keywords: Malignant • Carcinoma

Introduction

The morbidity and mortality of ESCC in China ranks first in the world while such category accounts for more than 90% of all pathological patterns, featuring the easy metastasis and invasion, as well as recurrence. The major therapies used for the ESCC currently are surgery in combination with neoadjuvant chemoradiation. Thus it may be of great significance if the effective therapeutic targets might be identified based on understanding the mechanism of ESCC at the molecular level. MicroRNAs (miRNAs) are a group of small non-coding RNAs which may regulate the gene expression at post-transcriptional level. As the important regulator in cell pathway, miRNA may act as the oncogene, antioncogene or transfer regulator. In the prior experiment, we had determined that miR-483-5p would be up-regulated in ESCC which might facilitate the occurrence of such cancer. The recent studies had shown that miR193b-3p as the cancer suppressive factor would be down-regulated in the various tumors. Pekow et al. reported that expression of miR-193a-3p decreased in the process in which the ulcerative colitis progressed to tumor, which would cause the up-regulation of target gene IL17RD to function in a carcinogenic manner [8]. The ERBB4 and S6K2 targeted by miR-193a-3p might suppress the ERBB signaling pathway to play a role in inhibiting the lung cancer. The change in its expression had been demonstrated to be related to the tumors such as gastric cancer, colon cancer and endometrial cancer [10-12]. Nevertheless, the potential role and related target gene of which miR-193b-3p functioned in the development and progress of ESCC were unclear yet. The miR-193b-3p may play a part in the inhibiton of tumor in many malignant tumors and interact with many target genes, so that it may become the new target of diagnosis and treatment of malignant tumor. The abnormal expression of CCND1 would cause the change of cell cycle and then the development of tumor. The recent studies had shown that miR-193b-3p promotes the development of acute myeloid leukemia by targeting CCND1 and KIT-RAS-RAF-MEK-ERK (MAPK). The family of insulin-like growth factors (IGFs) was highly correlated with tumor among which IGF1R was the focus of many studies for it was closely related to the growth and differentiation of cells. The high expression of IGF1R would be seen in liver, breast, prostate and bowel cancer and was associated with the development, progress and metastasis of tumor.

The recent studies had shown that miR-193b inhibits the growth and metastasis of renal cell carcinoma by targeting IGF1R. Unlike the prior studies, we tested the expression of upstream and downstream gene in signaling pathway cdk4/Rb/CCND1/p16 and PI3K/AKT in which CCND1 and IGF1R located. We predicted that miR-193b-3p might affect the relevant pathways via the target gene, and both pathways, which were associated with the cell survival rate in the ESCC would further influence the development and progress of tumor. The DNA modification of methylation is the important way in which the expression and regulation of epigenetic gene presents. Therefore, this study analyzed further if the abnormal expression of miR-193b-3p in ESCC was associated with the methylation of promoter region. Such efforts will provide the new idea for the miR-193b-3p acting as the new biomarker in the clinical diagnosis of ESCC and further the target therapy.

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