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MicroRNA-181a-5p Could be One of Potential Therapeutic Strategies for ALF

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Abstract

The study of miRNAs has flourished during the decade, and several specific miRNA expression profiles have been identified in liver disease. This short commentary on the research of microRNA-181a-5p, evaluated the strengths and limitations of the article MicroRNA-181a-5p alleviates acute liver failure in mice by inhibiting High Mobility Group Box 1 (*HMGB1*), aimed to explore the therapeutic prospects of microRNA-181a-5p in Acute Liver Failure (ALF).

Keywords: MicroRNA-181a-5p; ALF; MicroRNAs; Apoptosis

Description

The research aimed to explore the role of microRNA-181a-5p in liver failure through the mouse ALF model. In the study, ALF models were induced by injecting D-Galactosamine (D-Gal) or Lipopolysaccharides (LPS) at different times, and relevant genes and proteins were detected by using scientific molecular biology techniques. Differently expressed genes in the ALF model were screened by operating miRNA microarray and Quantitative Reverse Transcription Polymerase Chain Reaction (qRT-PCR) technology. To estimate the role of microRNA-181a-5p in cell apoptosis, cells were transfected with microRNA-181a-5p and subsequently verified apoptosis by detecting the levels of caspase-3. A decrease in cell apoptosis investigated in the *HMGB1* knockout model suggested that microRNA-181a-5p could alleviate cell apoptosis in ALF by down-regulating *HMGB1*. Nevertheless, the relationship between microRNA-181a-5p and *HMGB1* has not been elucidated and the mechanism of microRNA-181a-5p targeted regulation has not been investigated in the study.

As we know, microRNAs, post-transcriptional regulators of gene expression involved in almost all cellular processes, are considered promising biomarkers for diagnosis and prognosis of disease [1,2]. In the past ten years, a large number of studies related to miR-181a-5p mainly focused on the alleviation of inflammation as well as the inhibiton of tumor cell proliferation *via* regulating signaling pathways. Previous studies showed that microRNA-181a-5p could play a significant role in acute lung injury or kidney injury. Targeting microRNA-181a-5p could alleviate liver damage and fatty liver disease [3-5]. A report indicated that compared with single miRNA strand, miR-181a-5p and miR-181a-3p cooperatively receded endothelium inflammation [6]. Therefore, the combined effect of microRNA-181a-5p with other RNA and even cell cycle regulatory factors deserves more attention as well. On the other hand, regulation of miRNA expression might be a key point in liver diseases, further research should subsequently be carried out to validate the effect of microRNA-181a-5p in hepatocyte apoptosis. Moreover, success in exploring "microRNA-181a-5p targets in the liver" would rely on pairing between miRNAs and targets and guaranteeing authenticity and scientificity. Multiple technological platforms have been used and generous target prediction tools are developed for microRNA by scientists from various fields of biology and medicine *in vitro* and *in vivo* for the past few years [7]. Further study on the relationship between microRNA-181a-5p and *HMGB1* could develop a novel strategy to ameliorate ALF in the future.

In short, the study proposed the role of microRNA-181a-5p in ALF for the first time, which conceived a novel idea and insight for the treatment of ALF. The breakthrough in the field of ALF would result from the true correlation between alterations in miRNAs and corresponding targets in liver cells. More effective utilization of scientific platforms and molecular biology technologies, better identification of miRNA targets, and clear cognition of the mechanisms of miR-181a-5p involved in cell apoptosis, would offer new diagnostic and therapeutic strategies for ALF.

Competing interests

The authors report no conflict of interest.

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Not applicable.

Author contributions

Yuqiao Zeng wrote the commentary. Likun Wang provided advice on the manuscript. All authors contributed to the commentary and approved the submitted version.

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