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Role of Non Coding RNAs In Biomarkers Study

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INTRODUCTION

Noncoding RNAs, including microRNAs, modulate gene expression at the posttranscriptional level in mammals, whereas long coding RNAs modulate gene expression at both the transcriptional and posttranscriptional levels. Evidence suggests that alterations in ncRNA expression are widespread in almost all types of liver disease. However, the role of ncRNAs in liver fibrosis is unknown. Liver fibrosis is the process by which extracellular matrix proteins accumulate in the liver, causing organ dysfunction and tumorigenesis. We summarise current knowledge on the role of nonviral ncRNAs in promoting or suppressing liver fibrosis, the potential use of circulating miRNAs as biomarkers of liver fibrosis, and therapeutic approaches to treat liver fibrosis by targeting dysregulated miRNAs.

DESCRIPTION

The most common pathological feature caused by chronic liver injury is liver fibrosis or scarring, which is widely regarded as one of the leading causes of morbidity and mortality. It is characterised primarily by hepatic stellate cell activation and an increase in extracellular matrix protein deposition. Overwhelming evidence suggests that dysregulation of several noncoding RNAs, primarily long non-coding RNAs, microRNAs, and circular RNAs, contributes to HSC activation and liver fibrosis progression. These ncRNAs not only bind to their target genes to promote the development and progression of liver fibrosis, but they also function as competing endogenous RNAs, sponging with miRNAs to form signalling cascades. Among these signalling cascades, lncRNA-miRNA-mRNA and circRNA-miRNA-mRNA are important modulators of liver fibrosis initiation, progression, and regression. Thus, targeting these interacting ncRNA cascades could be a novel and promising therapeutic target for inhibiting HSC activation as well as preventing and reversing liver fibrosis. Initially, noncoding RNAs were thought to be transcriptional byproducts. Recent advances in ncRNA research, on the other hand, have increased our understanding of the role of ncRNA in gene regulation and disease pathogenesis. Consistent with these developments, research into the relationship between ncRNAs and the pathology of liver fibrosis is rapidly expanding. The first priority was to investigate the function and regulation mechanisms of microRNAs. However, research into the mechanisms of long noncoding RNAs and lncRNA-mediated liver fibrosis has only recently begun. In this review, we focus on abnormal lncRNA expression in liver fibrosis. Furthermore, we discuss how the interaction of lncRNAs with miRNAs affects the expression of protein-coding genes in liver fibrosis. Recent advances in understanding dysregulated lncRNA expression and the lncRNA-miRNA interaction in liver fibrosis will aid in the development of new therapeutic targets and biomarkers. Long non-coding RNAs and genes were analysed in fibrotic rat liver tissues by RNA sequencing and verified by quantitative reverse transcription polymerase chain reaction to identify long non-coding RNAs and their potential roles in hepatic fibrosis in CCl4-induced rat liver issues.

CONCLUSION

Bioinformatics analysis of differentially expressed lncRNAs and genes was used to build a co-expression network. We discovered ten new DE-lncRNAs that were downregulated during the fibrosis process in the liver. XLOC118358, the cis target gene of DE-lncRNA, was Met, and the cis target gene of the other nine DE-lncRNAs, XLOC004600, XLOC004605, XLOC004610, XLOC004611, XLOC004568, XLOC004580, XLOC004598, XLOC004601, and XLOC004602 The findings of a pathway-DEG co-expression network show that lncRNA-Met and lncRNAs-Nox4 are involved in oxidation-reduction processes and the PI3K/Akt signalling pathway. Our findings linked ten DE-lncRNAs to hepatic fibrosis, and the potential roles of DE-lncRNAs and target genes in hepatic fibrosis may lead to new therapeutic strategies.

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