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Down-regulation of miR-185 in liver fibrosis correlates with RHEB and RICTOR overexpression and HSCs activation

Jun Cheng^{1,2,3}, Li Zhou^{1,2,3}, Ming Han^{1,2,3} and Shunai Liu^{1,2}

¹Beijing Ditan Hospital - Capital Medical University, China

²Beijing Key Laboratory of Emerging Infectious Diseases, China

³Peking University Teaching Hospital, China

Activation of hepatic stellate cells (HSCs) into collagen producing myofibroblasts is critical for pathogenesis of liver fibrosis. Recent studies have shown effect of microRNAs (miRNAs) on regulating HSC activation during liver fibrosis. Here, we aimed to explore the roles of miR-185 in human liver fibrosis. Expression of plasma miR-185 was detected in HBV-related liver fibrosis patients (S2/3, n=10) and the liver cirrhosis patients (n=8) by quantitative real-time polymerase chain reaction (qRT-PCR), and healthy volunteers was selected (n=8) as control group. We found that the expression of plasma miR-185 in the HBV-related liver fibrosis patients was significantly downregulated. Carbon tetrachloride (CCl₄)-induced mice fibrotic liver tissues and TGF- β 1-induced activated HSCs also present down-regulation of miR-185 concomitant with increased expression of RHEB and RICTOR. To analyze the correlation of RHEB and RICTOR expression with miR-185, LX2 cells were transiently transfected with miR-185 mimics, and the expression of α -SMA, collagen I and collagen III was decreased as well as the expression of RHEB and RICTOR. Furthermore, dual-luciferase reporter assays indicated that miR-185 inhibited the expression of RHEB and RICTOR through direct targeting their 3'UTR regions. Moreover, knockdown of RHEB and RICTOR suppressed α -SMA and collagen I and collagen III in HSCs. In conclusion, miR-185 prevents liver fibrogenesis by inhibiting HSC activation through inhibiting RHEB and RICTOR. These results provide novel mechanistic insights for the anti-fibrotic effect of miR-185.

Biography

Jun Cheng is currently working as a Professor at Peking University Teaching Hospital, China. He has published numerous research papers and articles in reputed journals and has various other achievements in the related studies. He has extended his valuable service towards the scientific community with his extensive research work.

chengj0817@sina.cn

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