

Anti-fibrotic impact of Carvedilol in a CCl-4 model of liver fibrosis via serum microRNA-200a/SMAD7 enhancement to bridle TGF-β1/EMTtrack

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Circulating microRNAs (miRNAs) play a role in modulating the prevalence of fibrosis and have been a target of the cardiac anti-fibrotic effect of carvedilol. However, the impact of miRNAs on the hepatoprotective effect of this non-selective β-blocker has not been yet elucidated. Hence, the current goal is to evaluate the potential role of circulating miR-200a in the hepatic anti-fibrotic pathway of carvedilol. Male Wistar rats were randomized into normal, CCl_4 (2 ml/kg, i.p, twice weekly for 8 weeks), and CCl_4+Carvedilol (10mg/kg, p.o, daily). Carvedilol over-expressed the circulating miR-200a to modulate epithelial mesenchymal transition (EMT) markers (vimentin, E-Cadherin). In turn, carvedilol increased SMAD7 gene expression and protein content to attenuate the pro-fibrogenic marker transforming growth factor β1 (TGF-β1) and the inflammatory markers (p-38 MAPK and *p*-S536-NF-κB p65). The anti-fibrotic potential was reflected on the decreased expression of the mesenchymal product and EMT marker α-SMA, besides the improved histopathological examination, and the fibrosis scores/collagen quantification to enhance liver functions (AST, ALT, ALP, and AST/ platelet ratio index; APRI). In conclusion, circulating miR-200a/SMAD7/ TGF-β1/EMT/ MAPK axis is crucial in the hepatic anti-fibrotic mechanism of carvedilol.

Biography

I got my PhD in Pharmaceutical Sciences (Pharmacology and Toxicology speciality) from faculty of Pharmacy Cairo University in May 2019. Now, i'm an assistant professor at Pharmacy Practice and Clinical Pharmacy Department, at Misr International University (MIU). This abstract, is from my PhD thesis, that was published in Scientific Reports journal, Nature publisher.

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