Tenascin-C (TN-C) is an extracellular matrix glycoprotein markedly upregulated during liver fibrosis. The study is performed to explore the role of TN-C during the growth and activation of hepatic stellate cells (HSCs). We found that TN-C was accumulated accompanying with the HSC activation. Our data on cell migration assay revealed that the rTN-C treatment enhanced HSC migration in a dose- and time-dependent manner, but did not influence their proliferation. HSCs transfected with pTARGET-TN-C overexpression vector displayed increased the type I collagen (Col I) production. TN-C overexpression enhanced the process of HSC activation through TGF-β1 signaling. Moreover, the anti-α9β1 integrin antibody treatment blocked the TN-C-driven Col I increase in rat HSCs. Collectively, TN-C had a positive role in activation of HSCs mediated by TGF-β1 and α9β1 integrin, manifesting elevation of Col I production and promotion of cell migration. Our results provide a potential insight for the therapy of hepatic fibrosis.

TN-C induced TGF-β1 and α9β1 expression to promote the cell migration and type I Collagen production in the activated HSCs.