Results TGFβ1 and PDGF enhanced, whereas TNFα inhibited the expression of a1-PROC. Octreotide dose dependently inhibited the expression of a1-PROC in cells treated with TGFβ1, PDGF and increased the production of a1-PROC in TNF treated cells. Sodium orthovanadate significantly augmented the inhibition of a1-PROC production caused by octreotide only in TGFβ1 or PDGF treated cells. Okadaic acid uniformly inhibited the expression of a1-PROC. The expression of aSMA remained constant in all experiments. HSC proliferation increased by TGFβ1 and PDGF and was inhibited by TNFα Octreotide potentiated the effect of TGFβ1 and PDGF, and reversed TNFα inhibition. Orthovanadate and okadaic acid did not have any effect on the proliferation of cells. However, okadaic acid profoundly inhibited HSC proliferation when combined with octreotide, in TGFβ1 and PDGF treated cells.

Conclusion Somatostatin differentially influences HSC a1 procollagen production according to cytokine microenvironment and this effect is modulated by tyrosine and threonin phosphatases. Proliferation of HSCs is similarly influenced by Somatostatin by a phosphatase dependent mechanism.

Competing interests None.

Keywords hepatic stellate cells, liver fibrosis, somatostatin.

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DIFFERENTIAL EFFECT OF SOMATOSTATINERGIC SIGNALING ON COLLAGEN TYPE I PRODUCTION AND THE PROLIFERATION OF CYTOKINE PRIMED RAT HEPATIC STELLATE CELLS

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Introduction Somatostatin may influence hepatic fibrosis with mediators produced by Kupffer cells. The aim of this study is to investigate the role of somatostatinergic and cytokine signalling in hepatic stellate cells (HSC), the effector cells of hepatic fibrosis.

Methods The production of a1-procollagen (a1-PROC) by rat HSCs treated with TNFα (100 ng/ml), TGFβ1 (5 ng/ml) or PDGF (32 ng/ml) and their cellular proliferation with or without octreotide was investigated. a1-PROC and aSMA were analysed by Western blotting and cellular proliferation was assayed by MTT. The role of the phosphotyrosine (PTP) and phosphoserine-phosphothreonine (STP) phosphatases on somatostatin signalling, was investigated by using the PTP inhibitor sodium orthovanadate (0.1 μM) and the STP inhibitor okadaic acid (0.1 μM).