3-O-D2-4 Oral Sessions**Functional expression of TREK-1 channels in human hepatic stellate cells**

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Activated phenotype of hepatic stellate cells is associated with the development of liver fibrosis following enhanced Ca^{2+} signaling. Although cytosolic Ca^{2+} concentration is regulated by Ca^{2+} and K^{+} channels, their pathophysiological roles remain unclear. In the present study, we focused on the two-pore domain K^{+} ($\text{K}_{2\text{P}}$) channels, which regulate the resting membrane potentials, in hepatic stellate cells. In the presence of blockers for voltage-dependent K^{+} channels (10 mM tetraethylammonium and 5 mM 4-aminopyridine) and small-conductance Ca^{2+} -activated K^{+} channels (100 nM apamin), outward K^{+} currents were detected in human hepatic stellate LX-2 cells. Expression analyses revealed that, among the $\text{K}_{2\text{P}}$ family, TREK-1 (KCNK2) channels were abundantly expressed in LX-2 cells. TREK-1 siRNA (20 nM, for 48~72 h) specifically knocked-down the expression of TREK-1 channels. TREK-1 siRNA did not affect the expression of α -SMA (a marker for cell motility). On the other hand, TREK-1 siRNA reduced the expression of type I collagen (an extracellular matrix). TREK-1 siRNA also downregulated the expression of platelet-derived growth factor-BB (a cytokine associated with cell proliferation). These results suggest that TREK-1 channels contribute to the extracellular matrix production and cell proliferation in hepatic stellate cells.