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REVISTA DO HOSPITAL DE CLÍNICAS DE PORTO ALEGRE E  
FACULDADE DE MEDICINA DA UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL

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REVISTA HCPA 2007;27 (Supl 1) :1-292

# 27<sup>a</sup> Semana Científica do Hospital de Clínicas de Porto Alegre

14º Congresso de Pesquisa e Desenvolvimento em Saúde do Mercosul  
10 a 14 de setembro de 2007

# Anais

REVERSION OF ACTIVATED PHENOTYPE IN GRX CELLS USING RNAI FOR TGF-BETA1: PRELIMINARY RESULTS  
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TGF-Beta is a cytokine involved in cellular proliferation and differentiation. Its pro-fibrogenic role in liver is well established in the priming and maintenance of the activated phenotype hepatic stellate cells. In this study the cell line GRX was used as model for activated hepatic stellate cells. To inhibit TGF-Beta1 expression a plasmid containing the short hairpin interfering RNA (shRNAi) for TGF-Beta1 (pSUPER-TGFb) was transferred to GRX cells using liposomes. Control plasmid (pSUPER without shRNAi) was also transfected. Cells were selected with Puromycin (0.8 ug/mL) for 21 days. Changes in cell morphology were observed macroscopically. During the initial 7 days of selection, the vast majority of cells died in both groups, probably due to the low efficiency of liposome transfection. After 14 days, differences in cell proliferation were observed between the groups, with control cells reaching confluence after 20 days. At this time point, pSUPER-TGFb1 transfected cells started to show changes in morphology, displaying a more polygonal phenotype and lipid droplets. This cell morphology is characteristic of inactive GRX. Studies evaluating lipid content, actin rearrangement and levels of TGFbeta1 mRNA are undergoing. Our preliminary results indicate the ability of pSUPER-TGFb for reversing the phenotype of activated hepatic stellate cells. Future studies will be performed in the animal model of liver fibrosis induced by Carbon Tetrachloride.