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The effects of syndecan-2 fragments on TGF- β induced genes in breast cancer cells

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Introduction & Aim: Syndecan-2 (Sdc-2) is a transmembrane heparin sulfate proteoglycan that is up-regulated in breast tumors. Preliminary data indicates that over expression of Sdc-2 peptides in Breast Cancer Cells (BCC's) increase their migratory and immune-suppressive properties. Sdc-2 fragments were designed and cloned into a vector to mimic a component of endogenous Sdc-2. Overexpression of TGF- β results in pro-tumorigenic modifications to cells in the tumor microenvironment. Therefore, inhibition of the TGF- β pathway would be a rational approach in breast cancer therapies. Our objective was to determine the role of Sdc-2 on the TGF- β pathway in MDA-MB-231 BCC's.

Method: Cultured MDA-MB-231 breast cancer cells were transfected with Fc empty vector, Sdc-F1 or Sdc-F2. A serum starvation and a TGF- β 3 time course were carried out. RNA was harvested from the cells at 0, 1, 2, 4, 6 after TGF- β 3 treatment. The RNA was purified and quantified, followed by cDNA synthesis via reverse transcription. qPCR was carried out to determine the effect of Sdc-2 fragments on TGF- β induced genes such as SMAd7, Serpine1 and CTGF.

Result: Promising data was collected from all three experiments, however due to sensitivity of qPCR the figures were different preventing statistical significance. Throughout all three experiments consistent trends were observed such as SMAD7 and Serpine1 down-regulation by Sdc-2-Fc-peptides indicating TGF- β suppression especially at the 6-hours' time point.

Conclusion: Further investigation of Sdc-2-Fc-peptides is imperative since data collected revealed Sdc-2 interaction with TGF- β induced genes.

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