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Title: Specific knockdown of versican 1 ameliorated the impairment of elastin deposit in pulmonary fibroblasts co-cultured with cigarette smoke

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Body: Background Loss of elastin is a feature of emphysema. After secretion from cells, tropoelastin assembles into functional insoluble elastin. In our previous study, despite the increased expression of tropoelastin by the COPD fibroblasts this did not translate into increased elastin deposit, which was accompanied by an increased production of versican 1 (V1). We hypothesized that down-regulation of V1 could improve elastin repair. Methods Pulmonary fibroblasts CCL-210 were cultured and treated with or without cigarette smoke (CS). Expression of mRNA for tropoelastin and V1 were measured by qPCR. Supernatant protein levels for V1 and soluble elastin were measured by ELISA. Insoluble elastin in lysates was measured by Fastin Elastin assay. Specific siRNA was used to knockdown V1 expression. Results CS inhibited elastin mRNA expression at 3 days and 14 days after treatment ($p<0.01$); levels of tropoelastin and insoluble elastin were both decreased as compared to untreated cells at 14 days ($p<0.01$). mRNA and protein levels of V1 were increased after CS insult ($p<0.01$), which was diminished by V1 siRNA. V1 siRNA did not alter the decreased tropoelastin after CS treatment, but attenuated the decreased elastin deposit.

Conclusions V1 played a role in the impaired elastin deposit in pulmonary fibroblasts after CS exposure. Specific knockdown of V1 can alleviate the elastin loss.