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In-depth proteomic analysis to identify the cellular proteins and secretome of human tubular epithelial cells with fibrotic injury.

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Objectives: Fibrosis is the major pathophysiology in the development of chronic kidney disease. While there are several proteomic studies to reveal the mechanism of renal fibrosis, the in-depth analysis which elucidate the repertoire of proteins expressed on cell or released into extracellular space are lacking

Methods: To induce the fibrosis on kidney cells in stages, two different dose (1ng/ml and 2ng/ml) of TGFß were treated on primary cultured human renal proximal tubular epithelial cells (TECs). Liquid chromatography-tandem mass spectrometry based proteomic analysis were performed on isolated TECs and their media, respectively.

Results: When comparing the cellular proteins expressed in control and in cells with fibrotic damage, we identified 691 and 1344 differentially expressed proteins (DEP) in low and high dose treated cells, respectively. And the DEPs in secretomes from low and high dose treated cells were 168 and 283, respectively. Then we identified overlapping 74 DEPs which showed significant difference in both cell and secretome analysis. Eleven proteins including NAMPT and KRT18 were decreased in same manner in both cell and secretome analysis, suggesting overall decrease in expression of these proteins with fibrosis. Seventeen proteins including STRAP and EIF3B were significantly decreased in cells while increased in secretome, representing the possible extracellular release of proteins with the response to fibrosis injury. There were 25 proteins including SERPINE1 and CTGF significantly elevated in both cell and secretome identified. And the other proteins which increased in cells and decreased in secretome were identified as 15 including PIEZO1 and ABCD4, which are presumably translocated into the cells with damage.

Conclusions: We identified different protein expression changes in cells and secretomes following fibrotic injury. Further studies are needed to validate the pathophysiological role of these proteins on kidney tubulointerstitial fibrosis.