High glucose and TGF-β1 reduce expression of endoplasmic reticulum-resident selenoprotein S and selenoprotein N in human mesangial cells.

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Abstract
There are seven endoplasmic reticulum (ER)-resident selenoproteins in human body and they can regulate the inflammation, oxidative stress, and ER stress. We established transforming growth factor-β1 (TGF-β1) or high glucose (HG) induced human mesangial cells (HMCs) fibronectin expression model in vitro. Next, the expression changes of seven ER-resident selenoproteins were detected under HG conditions and we found selenoprotein S (SELENOS), selenoprotein N (SELENON) were significantly down-regulated but selenoprotein M was significantly up-regulated in transcription level. Furthermore, we found that TGF-β1 and HG down-regulated the expression of SELENOS and SELENON in a time- and dose-dependent manner, respectively. Finally, SELENOS was knocked down by siRNA and we found that knocking down SELENOS decreased TGF-β1 induced fibronectin expression. Our research indicates the potential value of ER-resident selenoproteins on renal fibrosis.

KEYWORDS: Endoplasmic reticulum; HMCs; TGF-β1; glucose; selenoprotein N; selenoprotein S