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## Rho kinase signal pathway is likely responsible for mitochondrial oxidative damage and apoptosis induced by high glucose in renal tubular epithelial cells

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**Objectives:** Growing evidences showed that tubules play a vital role in the pathogenesis of diabetic nephropathy in recent years, in which oxidative stress is induced under diabetic conditions. The purpose of this study was to investigate the role of oxidative stress in human renal tubular epithelial cells (HK-2) induced by high glucose and the underlying signal pathway in vitro.

**Methods:** MYPT1, pro-caspase-3, PGC-1 $\alpha$ , and Drp1 protein expressions were measured by Western blot. MnSOD2, Drp1 and PGC-1 $\alpha$  mRNA expressions were detected by real time PCR.

**Results:** Our study revealed that high glucose significantly up-regulated the protein expressions of MYPT1, pro-caspase-3 and the mRNA expression of MnSOD2 in HK-2 cells. While, Rho kinase inhibitor Fasudil and ROCK1 siRNA inhibited protein expressions of pro-caspase-3 and the mRNA expression of MnSOD2 in HK-2 cells induced by high glucose. Importantly, Fasudil and ROCK1 siRNA markedly inhibited the expressions of mitochondrial motor proteins Drp1 and mitochondrial gene PGC-1 $\alpha$  in HK-2 cells induced by high glucose.

**Conclusions:** In conclusion, our findings suggested that Rho kinase signal pathway was involved in mitochondrial oxidative damage and apoptosis in high glucose-induced renal tubular epithelial cells by regulating mitochondrial motor proteins Drp1 and mitochondrial gene PGC-1 $\alpha$ . Targeting Rho kinase signal pathway might be a potential strategy for the treatment of diabetic nephropathy.

Figure 1. Upregulation of MYPT1 protein in HK-2 cells induced by high glucose. The Rho kinase signal pathway was involved in high glucose-induced mitochondrial-mediated oxidative damage and apoptosis in HK-2 cells.

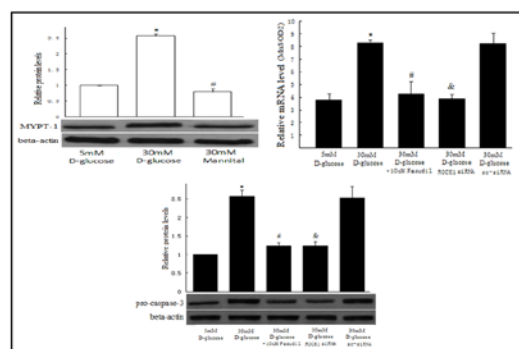


Figure 2. The Rho kinase signal pathway regulating mitochondria related proteins in high glucose-induced HK-2 cells.

