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Rho kinase signal pathway participates in the tubular mitochondrial oxidative injury and apoptosis via regulating mitochondrial dyneins/biogenic genes in uric acid nephropathys

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Objectives: Oxidative stress is a pathologic feature of hyperuricemia which is highly prevalent and contributes to kidney tubular interstitial fibrosis. Rho-kinase is closely related to oxidative stress induced by mitochondria. Here, we designed to explore the expression and role of Rho-kinase in hyperuricemia nephropathy. Secondary objective were to investigate whether the Rho-kinase signal pathway regulated the hyperuricemic tubular oxidative injury and apoptosis via mitochondrial pathway and the mechanisms involved.

Methods: HK-2 were divided into five groups for study as follows: normal; UA; UA+Fasudil; UA+ROCK1 si-RNA; UA+sc-siRNA. To measure Rho-kinase activity, mitochondrial oxidative injury and apoptosis-related protein of each group. T test was adopted to analysis the difference between groups.

Results: Overexpression of MYPT1 were shown in HK-2 cells induced by UA. High uric acid treatment also up-regulated the expression of Rho-kinase activity, mitochondria and apoptosis-related protein, while treatment with Fasudil and ROCK1 si-RNA significantly attenuated these response.

Conclusions: Rho-kinase signal pathway participates in the tubular mitochondrial oxidative injury and apoptosis via regulating mitochondrial dyneins/biogenic genes in uric acid nephropathy, which suggests that mitochondria pathway may represent a potential therapeutic target for hyperuricemia nephropathy.

Figure 1. Effects of Rho kinase pathway on mitochondria induced oxidative damage induced by high uric acid in HK-2 cells.

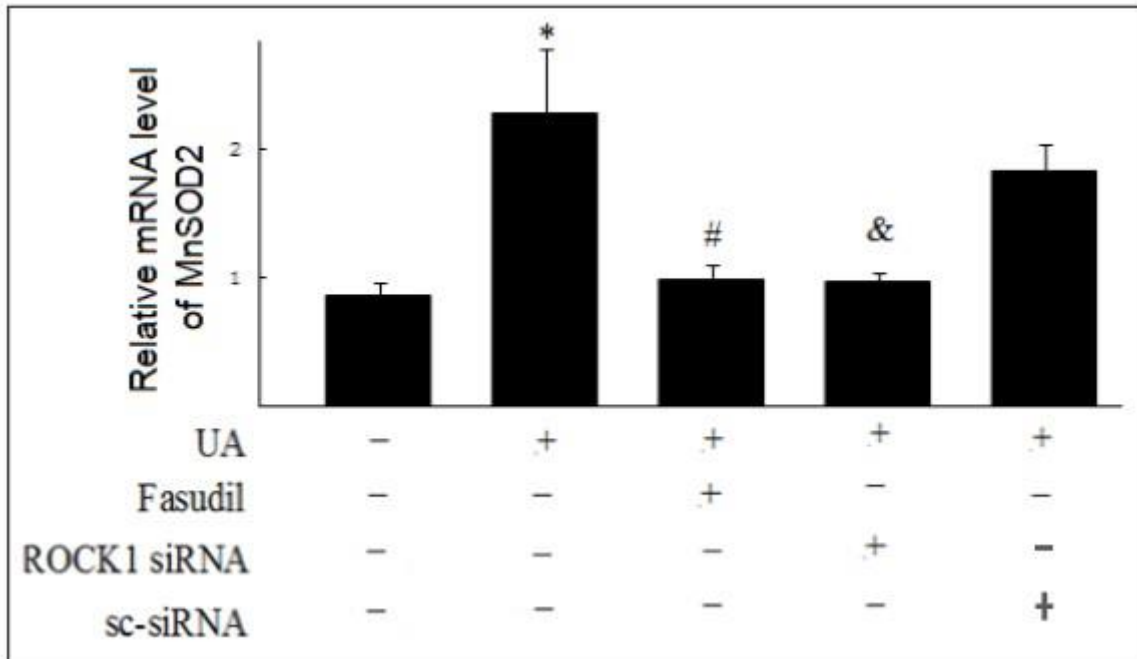


Figure 2. Mitochondria related proteins levels in high uric acid-induced HK-2 cells.

