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Notch Signaling Pathway in High Glucose Induced Mitochondrial Oxidative Damage and Apoptosis in Renal Tubular Epithelial Cells and Its Possible Mechanism

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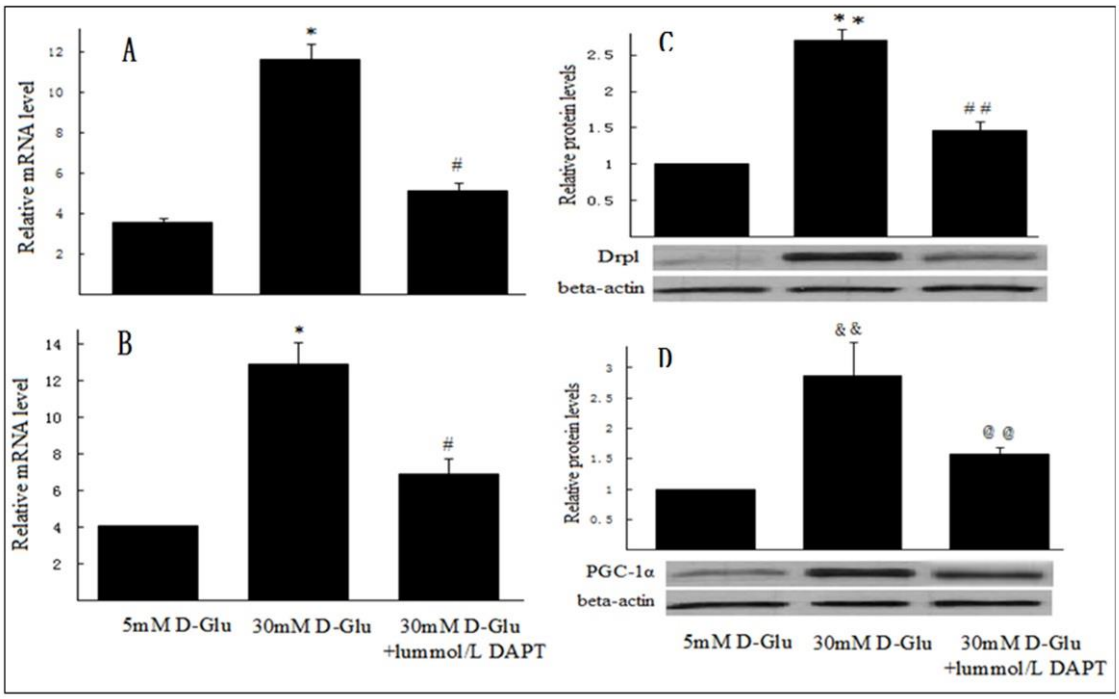
Objectives: Diabetic nephropathy (DN) is the most common and most serious chronic microvascular complication in diabetes. Recent researches have indicated that Notch pathway can mediate the impairment of glomerular and renal tubular function, and promote renal interstitial fibrosis and angiogenesis. Given this background, we explored the effects of Notch signaling pathway in high glucose induced mitochondrial oxidative damage and apoptosis in renal tubular epithelial cells and its possible mechanism.

Methods: Protein and mRNA expressions were detected by Western blot and Real-time PCR, respectively.

Results: High glucose up-regulated the protein expressions of Jaggedl, Notchl , pro-caspase-3, Drpl, and PGC-1α, and DAPT reversed such effects. Meanwhile, DAPT antagonized the mRNA expressions of Jaggedl, Notchl, MnSOD2, Drpl, and PGC-1α induced by high glucose

Conclusions: These data indicated that Notch signal pathway may regulate high glucose-induced oxidative damage and apoptosis in renal tubular epithelial cells by regulating mitochondrial dynein and biogenesis genes, thereby accelerating the interstitial fibrosis of DN. Notch signaling pathway might be a therapeutic target for DN mitochondrial oxidative damage and apoptosis

High glucose up-regulated the expressions of Drpl(A) and PGC-1α (B) mRNA and protein, DAPT antagonized the expression of high glucose induced Drpl and PGC-1α mRNA and protein expression


A
Relative mRNA level
12
10
8
6
4
2
*
#
B
Relative mRNA level
14
12
10
8
6
4
2
*
#
5mM D-Glu
30mM D-Glu
30mM D-Glu
+lummol/L DAPT
C
Relative protein levels
2.5
2
1.5
1
0.5
* *
# #
Drpl
beta-actin
D
Relative protein levels
3
2.5
2
1.5
1
0.5
& &
@ @
PGC-1α
beta-actin
5mM D-Glu
30mM D-Glu
30mM D-Glu
+lummol/L DAPT