Results: HFHS diet led to vacuolization and thyroidisation of the renal tubules. Glucose caused mitochondrial fragmentation and cellular apoptosis in HK2 cells. Autophagy was activated in glucose-treated cells as evidenced by the enhanced LC3B-II expression and autophagosome formation. When autophagy was inhibited, either by the inhibitor, 3-MA, or by siATG5, tubular cells were more susceptible to glucose-induced mitochondrial fragmentation and cellular apoptosis. When MFN1 or MFN2 was knockdown, mitochondria became more fragmented and autophagy was activated. When FIS1 was silenced, mitochondria were not fragmented and the expressions of LC3B-II, p62 and BECN1 remained unchanged.
Conclusion: Our results characterized the pathology of diabetic tubulopathy and suggested that glucose leads to mitochondrial fragmentation and cellular apoptosis in renal tubules. We also offered evidence that autophagy protects mitochondrial from fragmentation in renal tubules.

http://dx.doi.org/10.1016/j.hkjn.2015.08.020

0090
Protective Effect of Tangshen Formula on Renal Inflammation in Type 2 Diabetes

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Objective: Tangshen Formula (TSF), a traditional Chinese medicine, had been reported to have beneficial effects on diabetic nephropathy (DN), but its action mechanism is still unclear. The study was designed to elucidate the potential mechanism of TSF in treating DN.
Methods: Type 2 DN rat models were established by high-fat diet-fed and low-dose-streptozotocin injection. The rats were treated with or without TSF by gavage for 20 weeks and examined by 24h-albuminuria, histology, immunohistochemistry, molecular analyses.
Results: The results showed that TSF treatment significantly inhibited urinary excretion of albumin, and attenuated renal histological injuries in the rats. TSF treatment also inhibited renal inflammation which was associated with inactivation of NF-κB signaling. In addition, TSF treatment also suppressed expressions of fibronectin, collagen I, and collagen IV.
Conclusion: The present study revealed that TSF might attenuate renal inflammation of type 2 DN by inhibiting NF-κB signaling pathway.
Acknowledgements: This work was supported by the National Natural Science Fund (No.81173422, 81473526) and Beijing Municipal Natural Science (No.7142143).

http://dx.doi.org/10.1016/j.hkjn.2015.08.021

0091
The Expression of Electron Transfer Flavoprotein β Mediating Apoptosis of Renal Tubular Epithelial Cells in Diabetic Nephropathy

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Objective: In diabetic nephropathy, it has been proven lipotoxicity is an important pathological mechanism of progressive tubulointerstitial injury. Electron transport flavoprotein (ETF) is an electron acceptor in fatty acid β oxidation, which is composed by α and β subunits. Our previous studies have found ETFβ point mutations in conserved sequences by renal cortex proteome in diabetic nephropathy. In this study, the expression and distribution of ETFβ and its role in diabetic kidney damage were explored to provide new ideas for further insight into the pathogenesis of diabetic nephropathy.
Methods: Expression of ETFβ in kidney was detected by Western blot and immunohistochemistry in a model of spontaneous type 2 diabetic OLETF rats. The effects of ETFβ at low expression on fatty acid-induced apoptosis by knocking down the gene were detected in NRK 52E cells. Apoptosis and related apoptosis pathways induced by fatty acid or knock down of ETFβ combined with fatty acids stimulate were compared to analyze the influence of ETFβ on lipotoxic apoptosis.
Results: Decreased ETFβ expression occurred only in 56-week-old rats which was correlated with massive proteinuria and severe tubular damage. We found increased ROS generation, decreased mitochondrial membrane potential, increased expression of BAX, and decreased Bcl-2, increased Cleaved caspase-3, and a lot of apoptotic cells in NRK 52E cells induced by palmitate. After ETFβ gene was knocked out, the above change by PA stimulation in mitochondrial apoptosis pathway was furtherly aggravated.
Conclusion: The expression of ETFβ in kidney cortex was significantly decreased with the progression of renal injury in diabetic nephropathy rats. Knock down of ETFβ under palmitate stimulation can significantly increase apoptosis rate. Thus, we speculate decreased ETFβ expression may be involved in renal tubular epithelial cells apoptosis and the progression of diabetic nephropathy.

http://dx.doi.org/10.1016/j.hkjn.2015.08.022

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Haematuria and Outcomes in Patients with Diabetic Chronic Kidney Disease

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Objective: Microscopic haematuria (denoted as haematuria) is considered a risk factor for end-stage renal disease (ESRD) in glomerulonephritis. Haematuria is not infrequent in patients with diabetic chronic kidney disease (CKD) and is associated with albuminuria and the duration of diabetes. However, the clinical significance of haematuria in patients with diabetic CKD has been rarely studied.
Methods: We included 1958 patients with type 2 diabetes and CKD stages 1–5 from nephrology outpatient department. They were divided into 3 groups according to haematuria in 3 consecutive urinalyses: no haematuria (0–2 red blood cells [RBCs]/high-power field [hpf] $\geq$ 2 times), mild haematuria (2–5 RBCs/hpf $\geq$ 2 times), and moderate haematuria ($\geq$5–10 RBCs/hpf $\geq$ 2 times).
Results: The average estimated glomerular filtration rate was 24.5 (12.0–40.0) ml/min/1.73 m^2 and the average urine protein-to-creatinine ratio (UPCR) was 1448 (471–3622) mg/g. Among 102 patients received biopsy, 42 patients (41.2%) had biopsy-proven diabetic nephropathy. We observed that haematuria was associated with age, UPCR, duration of diabetes, mean blood pressure, albumin, glycated haemoglobin, and C-reactive protein. The hazard ratio (HR) of moderate haematuria for ESRD was 1.39 (95% confidence interval [CI]: 1.10–1.76, $P < 0.001$). The odds ratios (ORs) of moderate haematuria for rapid renal progression wad 1.81 (95% CI: 1.29–2.53, $P < 0.001$). Moreover, the HRs of mild haematuria and moderate haematuria for mortality were 1.43 (95% CI: 1.12–1.84, $P = 0.005$) and 1.73 (95% CI: 1.27–2.36, $P < 0.001$), respectively. In the subgroup analysis, we observed that the association between haematuria and ESRD was more prominent in patients with CKD stages 1–3 or UPCR <500 mg/g.
Conclusion: Microscopic haematuria in patients with diabetic CKD is associated with risks for ESRD and mortality.

http://dx.doi.org/10.1016/j.hkjn.2015.08.023

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The Study of Syndrome Differentiation Based on Renal Biopsy Pathology of Diabetic Kidney Disease

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Objective: Research the distribution of syndrome differentiation in Traditional Chinese Medicine (TCM) on diabetic kidney disease (DKD) and its relationship with pathological stage of DKD.