Diabetic nephropathy

- Effect of Renal Denervation on Early and Late Stages of Diabetic Nephropathy
- Therapeutic potential of Cordyceps militaris cultivated with Ginkgo biloba seeds for alleviating western diet-induced type 2 diabetes and diabetic nephropathy
- Two novel pathogenic PDX1 variants in two Japanese patients with maturity-onset diabetes of the young
- METTL3 promotes podocyte pyroptosis in diabetic nephropathy through N(6)-methyladenosine modification of TRIM29 mRNA
- The role of estimated glucose disposal rate in predicting cardiovascular risk among general and diabetes mellitus population: a systematic review and meta-analysis
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Diabetic nephropathy (DN) is characterized by albuminuria, hypertension, and a progressive decline in glomerular filtration rate.

Bioinformatics analysis revealed that IGHG1, TRIM11 (tripartite motif protein 11), and TonEBP are highly expressed in diabetic nephropathy. In vitro cell experiments demonstrated that IGHG1 positively regulates the expression of TRIM11 and TonEBP (tonicity-responsive enhancer binding protein) in HK2 cells treated with high glucose. Furthermore, TRIM11 upregulates the expression of TonEBP through activation of the MEK/ERK (mitogen-activated protein kinase/extracellular signal-regulated kinase) signaling pathway in HK2 cells treated with high glucose. In vivo, animal experiments further confirmed that silencing IGHG1 could prevent the occurrence and development of diabetic nephropathy.

The silencing of IGHG1 alleviated diabetic nephropathy by inhibiting the TRIM11/MEK/ERK axis and by downregulating TonEBP [1)]

The 3-hydroxy-3-methylglutaryl coenzyme A is a well-known agent that is active in lowering total plasma and low-density lipoprotein cholesterol (LDL-C) levels in cases with hypercholesterolemia. Hence, in this study, proteinuria changes at the beginning and after the withdrawal of lovastatin in patients with type 2 DN (T2DN) were studied.

Lovastatin was administered for thirty male patients with T2DN and then was withdrawn. Twenty-four hours, urine creatinine and protein levels were determined.

The mean levels of total cholesterol and LDL-C were reduced without any change in the triglyceride (TG) level while the high-density lipoprotein cholesterol (HDL-C) level was increased. There was a

reverse linear correlation between the changes in the level of HDL-C and the changes in the level of 24 h urine protein after 90 days of lovastatin therapy (P = 0.007, r = -0.484).

Short-term 3-month lovastatin therapy has no effect on proteinuria levels in patients with T2DN despite the antihyperlipidemic effects and reverse correlation of proteinuria with HDL-C [2)].

RBP4 may be used as a predictive factor of diabetic nephropathy patients complicated with silent cerebral infarction (SCI). and is positively correlated with cognitive dysfunction. RBP4/Lp-PLA2/Netrin-1 pathway activation may be one of the occurrence mechanisms in diabetic nephropathy complicated with SCI [3)].

Resveratrol (RSV) has been shown to have a renoprotective effect against diabetic nephropathy, but the underlying mechanisms of this have not been fully elucidated. The aim of the current study was to explore the mechanisms responsible for the therapeutic effects of RSV in rat mesangial cells in vitro and in a rat model of diabetic nephropathy. The viability of CRL-2573 rat mesangial cells and their expression levels of p38, phosphorylated (p)-p38, transforming growth factor beta 1 (TGF-β1) and fibronectin were assessed in response to treatment with high glucose, with or without RSV. Diabetic nephropathy was also induced in Sprague-Dawley rats by streptozotocin treatment. At 8 weeks, basic biochemical parameters and histopathological abnormalities as well as the expression of p38, p-p38, TGF-β1 and fibronectin in rat kidneys were compared between control diabetic rats and those treated with 20 mg/kg RSV daily for 4 weeks. In the mesangial cell line, RSV inhibited high glucose-induced increases in cell viability and fibronectin expression by significantly reducing p38 mitogen-activated protein kinase (MAPK) activation and TGF-β1 expression ($P<0.05$). In diabetic rats, RSV significantly decreased blood glucose, serum creatinine and urinary albumin levels, as well as the kidney weight and ratio of kidney weight/body weight compared with the control group ($P<0.05$). Moreover, RSV ameliorated renal histological changes and downregulated the expression of p-p38, TGF-β1 and fibronectin in the kidneys of diabetic rats. These data suggested that RSV protected renal tissue from diabetes-induced injury and that this activity may be via inhibition of the p38 MAPK/TGF-β1 signaling pathway [4)].

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