rates in patients with type 1 and type 2 diabetes. Published reports examining the efficacy and safety of sulodexide for preserving renal function in CKD have yielded conflicting results. To systematically evaluate the efficacy and safety of sulodexide agents for CKD, we conducted a systematic review of the published trials (RCTs).
Methods: Data sources included MEDLINE (1966—2012), EMBASE (1988—2012), the Cochrane Central Register of Controlled Trials, conference proceedings, and article reference lists. We included clinical trials reporting the effects of sulodexide regimens for DM on all-cause mortality, CV deaths, and major CV events, proteinuria with an observation period. Studies were excluded if they were crossover trials. Systematic reviews were performed on the outcomes of all-cause mortality, proteinuria and renal survival and adverse events
Results: Seven RCTs and 4 observational studies involving 386 patients were included in the review. One RCTs reported all-cause mortality, CV deaths, and major CV events, renal survival data with Kaplan-Meier survival curves. Only 6 study follow-up period have more than six months. RCT whose follow-up period have more than 6 months showed sulodexide had no statistically significant effects on improved renal survival and reduction of proteinuria. Only trials whose follow-up period have less than 6 months showed sulodexide had statistically significant effects on reduction of proteinuria. Patients receiving sulodexide therapy did not have an increased risk of hypertension or gastrointestinal tract tract adverse events.
Conclusion: The current cumulative evidence suggests that sulodexide reduce proteinuria only short-term effect, but this effect is dose-related. With the extension of observation time, sulodexide renal protective effect disappeared.

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Mechanism of Genistein in Autophagy of High Glucose Ambient Mice Podocyte

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Objective: Autophagy worked to maintain cell homeostasis under various stress conditions. The renal cells under high-glucose conditions, nutrient signaling pathways altering impacted cells autophagy. So the cells were abnormal to variety stress, further aggravating the disorder of cells function, which in turn developed into diabetic nephropathy (DN). Genistein (GEN) is one of the major soy isoflavones isolated from the soybean. In this study, we evaluate the effects of GEN on the autophagy of high glucose-induced podocytes in vitro.
Methods: Qualified podocytes were used in experiments. (1) Firstly, podocytes were cultivated with the high glucose for 0, 2, 6, 12, 24, 48 and 72 hours. Using western blot method found out the maximal time of LC3II. (2) Normal glucose (NG) group, mannitol control (MC) group, high glucose control (HG) group, high glucose + GEN (G) group, high glucose + chloroquine (CQ) group and high glucose + CQ + GEN (CG) group were set. According to the results of experiment (1), the podocytes were incubated with 20 μM GEN for 6 hours, then the autophagosome was evaluated by electron microscopy. (3) Setting controls as above, LC3 and p-mTOR were detected by immunofluorescence. (4) The expression of LC3II and p-mTOR were detected by western blot.
Results: (1) The time-effect relationship experiment disclosed that the protein expression of LC3II/I significantly when cells were treated with HG for 6 hours ($P < 0.01$). (2) Electron microscopy showed that the autophagosomes were more in NG than in HG group. (3) The results of immunofluorescence and western blot showed that LC3 significantly expressed in HG, G, CQ and CG group than in NG group ($P < 0.01$). The results of p-mTOR is increased in CQ group ($P < 0.01$).
Conclusion: GEN could relieve the inhibition of autophagy by CQ, and it would enhance the autophagy of podocyte.

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Role of SIRT1 /PGC-1α in Mitochondrial Oxidative Damage and Apoptosis of Podocyte Stimulated by High Glucose and Effects of Resveratrol

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Objective: Discuss the possible mechanisms of mitochondrial oxidative damage and cell apoptosis in diabetic podocyte lesions and provide theoretical basis for early prevention of diabetic nephropathy through stimulating podocytes with high glucose and interventing with resveratrol.
Methods: Podocytes were randomly divided into four groups: normal glucose group (NG), high glucose group (HG), high glucose + resveratrol group (HG+Res), high glucose + EX527 group (HG+EX527). The four groups were cultured for 48 hours. The expression of SIRT1, PGC1α, NRF1, TFAM, Cyto C and DIABLO were detected by Western blot. The mitochondrial ROS production was detected by MitoSOX staining. The mitochondrial membrane potential was measured by JC-1 staining. Complex I and III enzyme activities in mitochondria were measured by colorimetry. Podocyte apoptosis was detected by annexin V/PI staining.
Results: The protein expression of SIRT1, PGC-1α, NRF1 and TFAM were reduced in HG than those in NG. They were up-regulated in HG+Res, while lower in HG+EX527 than HG. Cyto C and Diablo in cytoplasm of HG were increased than those of NG. They were down-regulated in HG+Res, while higher in HG+EX527 than HG. The mitochondrial ROS production and apoptosis rate of podocyte in HG were increased than those of NG, those in HG + Res were decreased compared with HG, while those of HG + EX527 were increased. The Complex I and III enzyme activities and the mitochondrial membrane potential in HG were decreased than those of NG, those in HG+Res were increased, whereas those in HG+EX527 were decreased.
Conclusion: (1) Mitochondrial ROS synthesis was increased, mitochondrial respiratory function was decreased, mitochondrial apoptotic pathways was activated in podocyte treated with high glucose, which suggested that mitochondrial dysfunction might participate in the oxidative damage and apoptosis of podocyte in diabetic nephropathy. (2) Resveratrol could relieve the podocyte oxidative damage and apoptosis in podocyte treated with high glucose maybe through the mitochondrial protective mechanisms mediated by the activation of SIRT1/PGC-1α/NRF1/TFAM pathway.

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