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1,8 cineole protects type 2 diabetic rats against diabetic nephropathy via inducing the activity of glyoxalase-I and lowering the level of transforming growth factor-1β

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Abstract

Purpose: Diabetes leading to the production and circulation of glycation products along with the reduction of the activity of glyoxalase-I (GLO-I) contribute to diabetic nephropathy. Therefore, we studied the effect of 1,8 cineole (Cin) on the formation of diverse glycation products and the activity of GLO-I as well as renal histopathological alterations in the type-2 diabetic rat.

Methods: Type 2 diabetes was induced in rats with a combination of streptozotocin and nicotinamide (55 + 200 mg/kg). Two groups of rats, normal and diabetic, were treated intragastrically with Cin (200 mg/kg) once daily for 2 months. Fasting blood sugar, insulin resistance index, lipid profile, the activity of GLO-I, glycation products (Glycated albumin, Glycated LDL, Methylglyoxal, and advanced glycation end products), and oxidative stress (Advanced oxidation protein products, malondialdehyde, oxidized LDL, and reduced glutathione), inflammatory markers (Tumor necrosis factor-α and Transforming growth factor-1β), creatinine in the serum (Cre), and proteinuria (PU) in the urine of all rats was determined as well as renal histopathological alterations were investigated.

Results: Cin reduced biochemical (Cre and PU) and histopathological (glomerulosclerosis) indicators of renal dysfunction in the diabetic rat compared to untreated diabetic rats. Moreover, the treatment decreased different glycation, oxidative stress, and pro-inflammatory markers *($p < 0.001$)*. Further, Cin had an advantageous effect on glucose and lipid metabolism.

Conclusions: Cin ameliorated diabetic nephropathy via reduction of TGF-1β following to decrease the formation of different glycation products, oxidative stress, and inflammatory process with the induction of the activity of glyoxalase-I in type 2 diabetic rats.

Keywords: 1,8 cineole; Diabetic nephropathy; Glycation; Glyoxalase-I; TGF-1β.

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