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# Ameliorates diabetic nephropathy by targeting of MEK1/2-ERK1/2-RSK2 signaling in streptozotocin-induced diabetic mice by plumbagin.

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**Objectives:** The aim of this research was to examine the renoprotective effects and molecular mechanisms of the plumbagin in the diabetic mice caused by streptozotocin (STZ). Plumbagin Ameliorates early diabetic nephropathy through diabetic mouse inhibition MEK1/2-ERK1/2-RSK2.

**Methods:** Intraperitoneally, the male C57BL/6 was injected in 200 mg / kg STZ. Plumbagin was given for 8 consecutive weeks, starting 1 week after injection of STZ. Body weight, urinary excretion over 24 hours, and fasting blood glucose were assessed. Histopathological analysis has investigated the kidney tissue. The Western blotting study determined total levels and phosphorylation of the protein kinase 1/2 stimulated with mitogen (MEK1/2), the extracellular signal-regulated kinase 1 and 2 (ERK1/2) and S6 kinase 2 ribosomal (RSK2).

**Results:** Plumbagin therapy decreased considerably the amount of albuminuria and serum creatinine, enhanced mesangial matrix expansion and expanded footing cycle duration, and decreased the urinary rates of N-acetyl-beta-D-glucosamines, neutrophil gelatinase-assound lipocalin, and STZ-led diabetic mice. Plumbagin also prevented MEK1/2, ERK1/2 and RSK2 from being inhibited by renal cortical phosphorylation.

**Conclusions:** Our findings indicate that plumbagin mitigates renal injury in diabetes mice induced by STZ. The inhibition of the MEK1/2-ERK1/2-RSK2 signaling route could be partially correlated with this influence.