عنوان مقاله:

Crocin protects the renal tubular epithelial cells against high glucose-induced injury and oxidative stress via regulation of the SIRT۱/Nrf۲ pathway

محل انتشار:

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خلاصه مقاله:

Objective(s): Renal tubular damage is critical pathological feathers of diabetic nephropathy (DN). This study aimed to explore the protective activity and related mechanisms of crocin in renal epithelial cell injury induced by high glucose.Materials and Methods: Renal tubular epithelial HK-۲ cells were cultured with D-glucose to establish an in vitro DN model. Cell viability was evaluated by CCK-۸ assay. Apoptosis was detected by Annexin V-FITC kit. Oxidative stress was evaluated by colorimetry. RT-qPCR was carried out to determine the mRNA expressions of NF-E۲-related factor ۲ (Nrf۲) and its pathway genes. Western blot was applied to determine the protein expressions of Nrf۲ and related proteins.Results: High glucose (۵.۵, ۳۰, and ۵۰ mM D-glucose) decreased cell viability at ۷۲ hr, which was attenuated by crocin (۲۵ and ۵۰ μM). Crocin also attenuated the high glucose (۳۰ mM D-glucose) induced apoptosis of HK-۲ cells, decreased MDA content, and increased SOD activity in culture media. Crocin increased mRNA levels of Nrf۲, HO-۱, and NQO۱. Moreover, crocin increased protein expressions of Nrf۲, Sirtuin ۱ (SIRT۱), and p-Akt (Ser۴۷۳). Inhibition of Nrf۲ using siRNA, and inhibitors of SIRT۱ (nicotinamide, NAM, ۲۰ μM) and PI۳K/Akt (LY۲۹۴۰۰۲, ۵۰ μM) all attenuated the protective effect of crocin. Nrf۲ siRNA and NAM also partially attenuated the inhibitory effect on oxidative stress and increase in the Nrf۲ protein by crocin treatment.Conclusion: Crocin protects renal epithelial cells against injury induced by high glucose, and the mechanism is associated with partial activation of the SIRT۱-Nrf۲ pathway.

کلمات کلیدی:
Diabetic nephropathy, High glucose, NF-E۲-related factor ۲, Oxidative stress, Renal tubular epithelial cell, Sirtuin ۱

لینک ثابت مقاله در پایگاه سیویلیکا: