Biomed Pharmacother. 2018 Sep;105:1283-1290. doi: 10.1016/j.biopha.2018.06.108. Epub 2018 Jun 22.

Apigenin prevents metabolic syndrome in high-fructose diet-fed mice by Keap1-Nrf2 pathway.

Yang M[1], Jiang ZH[2], Li CG[2], Zhu YJ[2], Li Z[2], Tang YZ[2], Ni CL[2].

Author information

1 Key Laboratory of Hormones and Development (Ministry of Health), Tianjin Key Laboratory of Metabolic Diseases, Tianjin Metabolic Diseases Hospital & Tianjin Institute of Endocrinology, Tianjin Medical University, 300070, Tianjin, China. Electronic address: sanyoumu007@163.com.

2 Key Laboratory of Hormones and Development (Ministry of Health), Tianjin Key Laboratory of Metabolic Diseases, Tianjin Metabolic Diseases Hospital & Tianjin Institute of Endocrinology, Tianjin Medical University, 300070, Tianjin, China.

Abstract

Chronic dietary high fructose leads to various kinds of undesirable metabolic effects. Apigenin, a naturally occurring plant flavone, is plentiful in fruits and vegetables. The aim of this study was to identify the protective effects of apigenin on metabolic syndrome and elucidate potential underlying mechanisms. The animal model was established by 4-weeks high fructose feeding. Insulin resistance was estimated by oral glucose tolerance test and homeostasis model assessment-insulin resistance index. Liver function was evaluated by serum AST and ALT, hepatic histopathological alternation, and lipid accumulation in the liver. The alterations of lipid profile was evaluated by TG, TC, LDL-C and HDL-C levels in serum. Administration of apigenin exerted beneficial effects through improving insulin resistance, alleviating liver injury, and inhibiting the alterations of lipid profile in high fructose-fed mice. In addition, apigenin potently facilitated the accumulation of Nrf2 nuclear translocation and accompanied by increasing HO-1 and NQO1 protein expressions, which are responsible for attenuating oxidative stress. Molecular docking results demonstrated that potential interaction of apigenin with the Nrf2-binding site in the Keap1 protein. In summary, we demonstrated that apigenin prevented high fructose-induced metabolic syndrome probably by inhibiting binding of Keap1 to Nrf2, and thus Nrf2 nuclear translocation, subsequently resulting in increased the expressions of anti-oxidative genes including HO-1 and NQO1.

KEYWORDS: Apigenin; Keap1-Nrf2; Metabolic syndrome

PMID: 30021365 DOI: 10.1016/j.biopha.2018.06.108