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ORIGINAL ARTICLE

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Aucubin inhibited lipid accumulation and oxidative stress via Nrf2/HO-1 and AMPK signalling pathways

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Abstract

Aucubin (AU) is the main active ingredient of *Aucuba japonica* which has showed many positive effects such as anti-inflammation and liver protection. Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease. In this research, we explored the effects of AU on the tyloxapol-induced NAFLD in mice and apolipoprotein C-III (apoC-III) induced-3T3L1 cells. Tyloxapol (300 mg/kg) was injected to C57BL/6 mice with aucubin. The differentiated 3T3-L1 cells were treated with or without aucubin after stimulation of apoC-III (100 μg/mL). In results, aucubin inhibited hyperlipidaemia, oxidative stress and inflammation by influencing the content of total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), very low density lipoprotein (VLDL), myeloperoxidase (MPO), superoxide dismutase (SOD), tumour necrosis factor receptor-α (TNF-α), interleukin-1β (IL-1β), and IL-6 in blood. AU activated NF-E2-related factor 2 (Nrf2), peroxisome proliferator-activated receptor α (PPARα), PPARγ and hemeoxygenase-1 (HO-1) and promoted the phosphorylation of adenosine 5′-monophosphate-activated protein kinase (AMPKα), AMPKβ, acetyl-CoA carboxylase (ACC) and protein kinase B (AKT). In conclusion, AU performed the function of hypolipidaemic by its obvious anti-inflammation and antioxidant activity, which may become a kind of new drug targeting at NAFLD.

KEYWORDS
Aucubin, NAFLD, Nrf2, tyloxapol

1 | INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is an increasingly recognized cause of liver-related diseases and death. It represents a series of liver diseases characterized by macrovesicular steatosis in the case of alcohol intake (less than 40 g of ethanol per week) that is generally considered to be harmful to the liver. Although the relationship between hepatic alveolar steatosis and inflammatory changes and fibrosis in obese patients has been known for decades, it is a fact that has been ignored clinically.[1] NAFLD increases risk of type 2 diabetes mellitus (T2DM), cardiovascular (CVD) and cardiac diseases, and chronic kidney disease (CKD).[2] NAFLD can develop into non-alcoholic steatohepatitis (NASH) and eventually progress into hepatocellular carcinoma (HCC).[3] It could activate some related inflammatory or stress-response molecules such as nuclear factor-kappa B (NF-κB), phosphatase and tensin homolog (PTEN)

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