Lauric acid Ameliorates Non-alcoholic Fatty Liver Disease in Rats: Role of PI3K, IRS-1, Sirt1&AMPK signaling pathways

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Abstract

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major chronic liver illness characterized by hepatic lipid build up. This study investigated the role of lauric acid, a key component of coconut oil with potential anti-inflammatory, anti-oxidant, and insulin sensitizing properties against NAFLD induced by feeding rats a high-fat diet (HFD). In the present study, to induce NAFLD in the rats, an HFD was administered (five times per week) for 8 weeks. Lauric acid groups were received lauric acid (250 and 500 mg/kg; orally), concurrently with HFD for 8 weeks. The biochemical and the hepatic histological studies were performed. Lauric acid ameliorated serum levels of TG, TC, ALT, AST, blood glucose and insulin.

Moreover, lauric acid significantly elevated the levels of SOD, GSH, catalase and IL-10. Additionally, it lowered MDA, ROS, MPO, 4-HNE IL-1β and TNF-α levels in the liver homogenate. Furthermore, lauric acid significantly down-regulated the expression of TGF-β1, and up-regulated the expression of IRS1, AMPK, PI3K and SIRT1 genes in the liver. In parallel, lauric acid has ameliorated the liver apoptosis by decreasing the expression of annexin V. Lauric acid histological results showed normal appearance of the central vein with considerably enhanced hepatocytes and full absence of fat vacuoles. Overall, these data proposed that lauric acid improve the lipid deposition and ameliorate the inflammation in NAFLD rats via regulating the PI3K, IRS-1, Sirt1& AMPK signaling pathways which could be an effective candidate for the NAFLD treatment.

Keywords: NAFLD, HFD, lauric acid, Liver, annexin V, AMPK