# Postprint: Effects of Ethanol Extract of Moringa oleifera Leaves on Blood Lipids and Oxidative Stress in a Mouse Model of Non-alcoholic Fatty Liver Disease

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**Date:** 2018-10-26T00:00:00+00:00

## Abstract

High-fat diet-induced non-alcoholic fatty liver disease (NAFLD) mouse models were treated with low (5 mg/kg), medium (30 mg · kg-1), and high (60 mg · kg-1) doses of Moringa oleifera leaf ethanol extract (EE-MO). The results showed that high-dose EE-MO significantly reduced body weight and liver wet weight in NAFLD mice. EE-MO dose-dependently decreased serum TC, TG, HDL-C, and LDL-C levels in NAFLD mice. In addition to reducing the aforementioned biochemical parameters, high-dose EE-MO also significantly decreased serum FFA content. HE and Sudan III staining results demonstrated that hepatic steatosis and cellular damage were significantly ameliorated in model mice after EE-MO treatment. EE-MO exhibited ameliorative effects on lipid metabolism in the NAFLD mouse model. Furthermore, high-fat diet was found to induce ROS and MDA content in mouse liver and serum, increase SOD, POD, and CAT activities, and decrease GSH-Px activity. Low, medium, and high doses of EE-MO dose-dependently reduced ROS and MDA contents in the liver and serum of NAFLD mice, alleviating oxidative stress. Low-dose EE-MO had no significant effect on SOD, POD, CAT, and GSH-Px enzyme activities. After treatment with medium and high doses of EE-MO, SOD, POD, and CAT enzyme activities in NAFLD mice

## Full Text

## Figure Specifications

Figure 1 [Figure 1: see original paper] full-width, 65 mm height; Figure 2 [Figure 2: see original paper] half-column, 65 mm height; Figure 3 [Figure 3: see original paper] full-width, 125 mm height; Figures 4 [Figure 4: see original paper]-5 full-width, 105 mm height

# Effect of Ethanol Extract of Moringa oleifera Leaves on Lipid Profile and Oxidative Stress in a Mouse Model of Non-alcoholic Fatty Liver Disease

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## Abstract

This study investigated the effects of low (5 mg/kg), medium (30 mg · kg $^{1}$), and high (60 mg · kg $^{1}$) doses of ethanol extract of Moringa oleifera leaves (EE-MO) on a high-fat diet-induced mouse model of non-alcoholic fatty liver disease (NAFLD). The results demonstrated that high-dose EE-MO significantly reduced body weight and wet liver weight in NAFLD mice. EE-MO treatment dose-dependently decreased serum TC, TG, HDL-C, and LDL-C levels. In addition to reducing these biochemical parameters, high-dose EE-MO also significantly lowered serum FFA content. Histological analysis via HE and Sudan III staining revealed that EE-MO treatment markedly ameliorated hepatic steatosis and cellular injury in model mice, indicating that EE-MO improves lipid metabolism in the NAFLD mouse model.

Furthermore, the high-fat diet increased ROS and MDA contents while elevating SOD, POD, and CAT activities and suppressing GSH-Px activity in both liver and serum. Low, medium, and high doses of EE-MO dose-dependently reduced ROS and MDA levels in liver and serum, thereby alleviating oxidative stress. Low-dose EE-MO showed no significant effect on SOD, POD, CAT, or GSH-Px enzyme activities. However, medium and high doses of EE-MO significantly decreased SOD, POD, and CAT activities while increasing GSH-Px activity. These findings suggest that EE-MO may alleviate oxidative stress in NAFLD mice through the GSH-Px antioxidant enzyme pathway.

**Keywords:** Moringa oleifera, oxidative stress, non-alcoholic fatty liver disease, lipid profile

## Introduction

Moringa oleifera, belonging to the family Moringaceae, is known as the “miracle tree”and represents a tropical plant with unique economic value. Native to India, Moringa has been introduced and commercially cultivated in Chinese provinces including Yunnan, Guangdong, Guangxi, Fujian, Guizhou, and Taiwan since the last century. The plant is entirely utilizable: its root bark serves as a traditional medicinal material, young leaves and pods are nutritious vegetables, and seeds contain valuable vegetable oil that is in high demand internationally. Nutritional

analysis reveals that Moringa contains twice the protein of milk, four times the calcium, twice the potassium of bananas, twice the iron of spinach, seven times the vitamin C of citrus fruits, four times the vitamin A of carrots, and vitamin E levels 70-fold and 40-fold higher than spirulina and soybean flour, respectively (Liu and Li, 2004).

Modern pharmacological research has demonstrated that Moringa possesses antipyretic, anti-inflammatory, anti-lithiatic, diuretic, hypotensive, lipid-lowering, and antioxidant properties. In India and African countries, it is commonly used to treat diabetes, hypertension, cardiovascular disease, and obesity (Anwar et al., 2007; Fahey, 2005; Ghasi et al., 2000; Song et al., 2017). Yang et al. (2017) reported that different doses of Moringa aqueous extract reduced serum TC, TG, HDL-C, and LDL-C levels in high-fat diet-induced obese rats. Similarly, normal SD rats fed Moringa leaf powder showed significantly lower serum triglyceride and cholesterol levels compared to controls (Zhang et al., 2016). Moreover, Moringa extracts exhibit potent antioxidant activity. In vitro studies have shown that the ethanol extract of Moringa oleifera leaves (EE-MO) exhibits DPPH, ABTS, and OH radical scavenging rates comparable to the positive control BHT (Zhou et al., 2017). Water-soluble polysaccharides from Moringa stems and leaves scavenge hydroxyl and superoxide anion radicals in a dose-dependent manner (Liang and Zhen, 2013). In rat models of renal and myocardial ischemia-reperfusion injury, Moringa seed extract significantly reduced serum MDA content and increased glutathione peroxidase activity in serum, kidney, and heart tissues, thereby alleviating oxidative stress (Qu, 2016; Qu et al., 2017).

The progression of non-alcoholic fatty liver disease (NAFLD) is a gradual process encompassing simple steatosis, non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis, with some patients progressing to hepatocellular carcinoma. In recent years, with improving living standards and lifestyle changes, NAFLD incidence in China has risen significantly and shows a trend toward younger populations, making it the second most common liver disease after viral hepatitis. While the pathogenesis of NAFLD remains incompletely understood, it is believed to involve insulin resistance (IR), oxidative stress, inflammation, Kupffer cell activation, cytokine dysregulation, lipid metabolism disorders, and iron overload (Loomba and Sanyal, 2013). Among these, oxidative stress and lipid metabolism disorders play crucial roles in hepatocellular injury. This study investigates the effects of EE-MO on lipid profile and oxidative stress in a NAFLD mouse model to explore its therapeutic efficacy, elucidate potential mechanisms, and provide a basis for the rational development of Moringa-derived interventions.

## Materials and Methods

### 1.1 Preparation of EE-MO

Dried Moringa oleifera leaves were purchased from the Luosifen herbal medicine market in Kunming, Yunnan Province. A total of 2.5 kg of dried leaves were pulverized and extracted three times with 95% ethanol at room temperature (6 L per extraction, 24 h each). The extracts were combined, and ethanol was recovered using a rotary evaporator until no alcohol remained. The ethanol extract was then lyophilized to obtain a dry powder.

### 1.2 Animal Model Establishment and Experimental Design

Fifty-five SPF-grade mice were randomly divided into a normal control group (N-CK, n=10) and a model group (n=45). The NAFLD model was established using a high-fat diet composed of 88% normal chow, 10% lard, and 2% cholesterol, processed into pellet form (Zhong et al., 2000). The normal group received standard chow throughout the study, while the model group was fed the high-fat diet. Beginning at week 5, model group mice were injected intraperitoneally with 40% CCl  in soybean oil solution (2 μL · g $^{1}$) twice weekly for 3 weeks, while the normal group received pure soybean oil. After 8 weeks of modeling, five mice from the model group were randomly selected and euthanized for hepatic histopathology to confirm successful model establishment. From week 8 onward, the model group was subdivided into model control (M-CK), low-dose Moringa (D-LM), medium-dose Moringa (Z-LM), and high-dose Moringa (G-LM) groups. At 9:00 AM daily, D-LM, Z-LM, and G-LM groups received oral administration of 5, 30, and 60 mg · kg $^{1}$ EE-MO, respectively, while M-CK received equal volumes of normal saline. During the 2-week treatment period, N-CK mice continued on standard chow, while all model groups remained on the high-fat diet. After 2 weeks, all mice were euthanized for collection of blood and liver tissues for subsequent biochemical and histological analyses.

### 1.3 Biochemical Analysis of Blood and Liver Tissues

Serum levels of TC, TG, HDL-C, LDL-C, and FFA, as well as ALT and AST activities, were measured using commercial kits according to manufacturer protocols. TC and TG assay kits were purchased from Shanghai Bioengineering Technology Co., Ltd. HDL-C, LDL-C, and FFA kits were obtained from Dalian TAKARA. ALT and AST activity kits were sourced from Sigma-Aldrich Trading Co., Ltd. (Shanghai, China).

### 1.4 Antioxidant Enzyme Activity Assays

SOD activity was determined using the nitroblue tetrazolium method. POD activity was measured via the guaiacol method. CAT activity was assessed using the UV rate method, while GSH-Px activity was determined by spectrophotometric colorimetry.

### 1.5 MDA and ROS Content Measurement

Malondialdehyde (MDA) content, a lipid peroxidation product, was measured using the thiobarbituric acid spectrophotometric method. ROS content was determined by the luminol chemiluminescence method (Faulkner and Fridovich, 1993).

### 1.6 Hematoxylin-Eosin (HE) Staining

Liver tissue fragments were fixed in 4% paraformaldehyde phosphate buffer for 24 h, then rinsed with running water for 4 h. Samples were dehydrated through graded ethanol series (50%, 70%, 80%, 90%, and 100%), cleared in xylene (two changes, 15 min each), and embedded in paraffin. Sections were cut, dewaxed in xylene I and II, rehydrated through descending ethanol concentrations (95%, 85%, 70%, 50%), and rinsed in distilled water for 2 min. Specimens were stained with hematoxylin for 5–15 min and 1% eosin for 1–5 min, then dehydrated, cleared, and examined microscopically.

### 1.7 Sudan Red III Staining

Performed according to the method described by Ma et al. (2010).

## Results

### 2.1 Effects of EE-MO on Body and Liver Weight in NAFLD Mice

As shown in Table 1 , after 8 weeks of high-fat feeding, M-CK group mice exhibited significantly higher body weight compared to N-CK. Following 2 weeks of low-dose EE-MO intervention, body weight showed no significant difference from M-CK. Medium and high-dose EE-MO treatment for 2 weeks resulted in a decreasing trend in body weight; although Z-LM remained non-significant compared to M-CK, G-LM showed significantly lower body weight. Liver weight followed a similar pattern, with M-CK significantly higher than N-CK. After EE-MO intervention, D-LM and Z-LM showed no significant difference from M-CK, whereas G-LM demonstrated significantly reduced wet liver weight.

**Table 1** Effects of EE-MO treatment on body weight (g) and wet liver weight (g) in NAFLD mice model

| Parameters | N-CK | M-CK | D-LM | Z-LM | G-LM |
|---|---|---|---|---|---|
| Body weight | 8.26±0.93 | 9.00±0.25 | 9.06±0.80 | 8.98±0.13 | 8.83±0.03 |
| Wet liver weight | 0.23±0.01 | 0.39±0.01 | 0.38±0.08 | 0.33±0.05 | 0.30±0.02 |

Note:  vs. N-CK,  vs. M-CK, $P<0.05$.

### 2.2 Effects of EE-MO on Serum TC, TG, HDL-C, LDL-C, and FFA in NAFLD Mice

As shown in Figure 1A, after 8 weeks of high-fat diet, M-CK mice exhibited significantly elevated serum TC and TG compared to N-CK. EE-MO dose-dependently reduced serum TC and TG levels. Serum LDL-C and HDL-C showed similar trends, with M-CK significantly higher than N-CK, and EE-MO treatment significantly decreasing both parameters (Figure 1B). High-fat diet also significantly increased serum FFA in M-CK versus N-CK. While D-LM and Z-LM showed no significant difference from M-CK, G-LM demonstrated significantly reduced FFA (Figure 1C).

**Figure 1** Effects of EE-MO treatment on serum TC, TG, HDL-C, LDL-C, and FFA contents in NAFLD mice model. vs. N-CK, * vs. M-CK, $P<0.05$.

### 2.3 Effects of EE-MO on Serum ALT and AST Activities in NAFLD Mice

As shown in Figure 2, NAFLD mice exhibited significantly increased serum ALT and AST activities following high-fat diet induction. EE-MO treatment dose-dependently reduced these enzyme activities, with the following order: D-LM > Z-LM > G-LM.

**Figure 2** Effects of EE-MO treatment on serum ALT and AST activities in NAFLD mice model. vs. N-CK, * vs. M-CK, $P<0.05$.

### 2.4 Effects of EE-MO on Serum and Hepatic ROS and MDA Contents in NAFLD Mice

High-fat diet significantly increased serum ROS content (Figure 3A). While D-LM showed no significant difference from M-CK, Z-LM and G-LM exhibited significantly reduced serum ROS, with G-LM lower than Z-LM. Hepatic ROS was similarly elevated in M-CK versus N-CK. Low-dose EE-MO significantly reduced hepatic ROS compared to M-CK, while medium and high doses further decreased ROS in a dose-dependent manner (Figure 3B). Serum and hepatic MDA contents paralleled ROS changes, with high-fat diet increasing MDA and EE-MO dose-dependently reducing both serum and hepatic MDA (Figure 3C and D).

**Figure 3** Effects of EE-MO treatment on ROS and MDA contents in serum and liver of NAFLD mice model. vs. N-CK, * vs. M-CK, $P<0.05$.

### 3.5 Effects of EE-MO on Antioxidant Enzyme Activities in NAFLD Mice

As shown in Table 2 , M-CK exhibited significantly higher serum and hepatic SOD, CAT, and POD activities than N-CK, indicating antioxidant enzyme induction by high-fat diet. D-LM showed no significant difference from M-CK in

these enzyme activities. Z-LM significantly reduced serum and hepatic SOD, CAT, and POD activities compared to M-CK. G-LM further decreased these activities below Z-LM and M-CK levels, with hepatic SOD, CAT, and POD restoring to N-CK levels. Conversely, GSH-Px activity was significantly suppressed in M-CK versus N-CK. While D-LM showed no difference from M-CK, Z-LM and G-LM significantly increased GSH-Px activity in both serum and liver, with G-LM higher than Z-LM. Medium and high doses of EE-MO reversed the high-fat diet-induced suppression of GSH-Px activity.

**Table 2** Effects of EE-MO treatment on antioxidant enzyme activities in NAFLD mice model

| Tissues | Antioxidant enzymes | N-CK | M-CK | D-LM | Z-LM | G-LM |
|---|---|---|---|---|---|---|
| **Serum** | SOD (U · mL $^{1}$) | 2.05±0.26 | 5.02±0.15 | 5.08±0.23 | 4.38±0.02 | 2.07±0.02 |
| | CAT (U · mL $^{1}$) | 4.98±0.50 | 11.25±1.23 | 13.56±1.61 | 6.56±0.78 | 5.25±0.21 |
| | POD (U · mL $^{1}$) | 3.23±0.08 | 8.45±0.49 | 9.09±0.88 | 7.06±0.98 | 3.58±0.28 |
| | GSH-Px (U · mL $^{1}$) | 11.98±0.20 | 5.65±0.12 | 5.59±0.32 | 12.88±2.01 | 14.35±1.00 |
| **Liver** | SOD (U · mg $^{1}$) | 1.26±0.07 | 7.32±0.27 | 7.36±1.45 | 5.25±0.22 | 2.23±0.08 |
| | CAT (U · mg $^{1}$) | 0.32±0.03 | 3.56±0.09 | 3.93±0.23 | 2.45±0.05 | 1.02±0.01 |
| | POD (U · mg $^{1}$) | 0.78±0.05 | 6.32±0.59 | 6.56±0.57 | 3.98±0.21 | 2.24±0.03 |
| | GSH-Px (U · mg $^{1}$) | 0.25±0.01 | 0.02±0.002 | 0.07±0.001 | 0.28±0.005 | 0.39±0.01 |

Note: vs. N-CK, vs. M-CK, $P<0.05$.

### 3.6 Histological Analysis of Liver Tissues Following EE-MO Intervention

As shown in Figure 4, N-CK mice exhibited regularly arranged hepatic cords radiating from central veins, with uniform hepatocyte size and predominantly mononuclear cells (some binucleated). After 8 weeks of high-fat diet, M-CK mice showed diffuse hepatocellular steatosis, disorganized hepatic cords, and diffuse hepatocyte swelling obscuring lobular architecture, with ballooning degeneration. Low-dose EE-MO intervention (D-LM) resulted in persistent extensive steatosis, enlarged hepatocytes, disorganized cords, and numerous cytoplasmic fat vacuoles. Medium-dose treatment (Z-LM) showed reduced but still present steatosis, with swollen hepatocytes containing fewer fat vacuoles and mildly disorganized cords. High-dose treatment (G-LM) restored regular hepatic cord arrangement around central veins, with only minimal steatosis and round nuclei.

**Figure 4** HE staining of liver tissues from NAFLD mice model after treatment with different EE-MO dosages.

### 3.7 Effects of EE-MO Intervention on Hepatic Steatosis

Sudan III staining revealed no significant orange-red coloration in N-CK livers, indicating absence of steatosis. M-CK livers showed extensive orange-red stain-

ing, confirming severe steatosis. Low-dose EE-MO reduced staining area and intensity compared to M-CK, though most tissue remained orange-red. Medium and high doses further decreased staining area and intensity, with no significant difference between Z-LM and G-LM groups (Figure 5 [Figure 5: see original paper]).

**Figure 5** Sudan red III staining of livers from NAFLD mice model after treatment with different EE-MO dosages.

## Discussion

NAFLD prevalence is extremely high in Europe and America, and its incidence in China is rising with the popularity of unhealthy lifestyles and increasing obesity. Studies indicate that approximately 25% of NAFLD patients progress to cirrhosis within 10 years, necessitating early intervention. The "two-hit" hypothesis is widely accepted as the classic pathogenic mechanism of NAFLD. The first hit involves lipid accumulation in hepatocyte cytoplasm, triggering cytotoxic events. The second hit comprises oxidative stress reactions—ROS-induced inflammatory responses in hepatocytes that constitute a critical factor in NAFLD pathogenesis (Nd, 2003). This study first examined EE-MO effects on oxidative stress in the NAFLD mouse model. High-fat diet induced ROS and MDA accumulation in serum and liver, confirming oxidative stress events. EE-MO dose-dependently reduced ROS and MDA accumulation, alleviating oxidative stress.

Analysis of classic antioxidant enzymes (SOD, CAT, POD) revealed that high-fat diet significantly increased their activities, likely as a compensatory response to ROS accumulation. Low-dose EE-MO showed no significant effect on these enzyme activities. Medium and high doses significantly reduced SOD, POD, and CAT activities, with G-LM restoring them to N-CK levels. Despite high-fat diet-induced increases in antioxidant enzyme activities, medium and high-dose EE-MO reduced them below N-CK levels, indicating that EE-MO does not alleviate oxidative stress by enhancing SOD, POD, or CAT activities. Conversely, high-fat diet suppressed GSH-Px activity, while EE-MO treatment dose-dependently increased GSH-Px activity in serum and liver. GSH-Px is another crucial peroxide-decomposing enzyme that, under normal conditions, maintains hepatic redox balance by catalyzing GSH-mediated reduction of hydrogen peroxide to protect cellular membranes. Previous studies have shown decreased GSH-Px activity in STZ-induced NAFLD rat models, with pharmacological intervention restoring normal levels (Videla et al., 2004; Wang et al., 2013). Our findings demonstrate that EE-MO dose-dependently reverses high-fat diet-induced GSH-Px suppression, suggesting a key role for GSH-Px in mitigating oxidative stress in this NAFLD model. Additionally, EE-MO contains secondary metabolites such as flavonoids, polysaccharides, and anthocyanins that may scavenge ROS through non-enzymatic pathways (Chen, 2006; Chen et al., 2007; Liu and Li, 2004).

Beyond oxidative stress, dyslipidemia represents another critical factor in NAFLD pathogenesis. In the first-hit hypothesis, insulin resistance leads to hepatic steatosis, triggering lipid metabolism disorders and liver injury. This study also examined EE-MO effects on TC, TG, HDL-C, LDL-C, and FFA. High-fat diet elevated all these parameters, consistent with previous reports (Chao et al., 2014; Li et al., 2013). While low and medium doses showed limited effects on FFA, high-dose EE-MO significantly reduced FFA content. All EE-MO doses decreased serum TC, TG, LDL-C, and HDL-C, reducing hepatic lipid accumulation as confirmed by HE and Sudan III staining. These results demonstrate that EE-MO effectively ameliorates dyslipidemia in the NAFLD mouse model.

This study is the first to demonstrate that EE-MO treatment effectively alleviates both oxidative stress and lipid metabolism disorders in NAFLD, with medium and high doses showing superior efficacy to low doses. However, the treatment duration was limited to 2 weeks. Although no mortality occurred during this period, the potential toxicity of high-dose EE-MO warrants further investigation.

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*Note: Figure translations are in progress. See original paper for figures.*