

# Anti-hyperlipidemic Effect of Methanol Seed Kernel Extract of *Mangifera indica* on Wistar Rats fed High Fat Diet

## Abstract

The aim of this study was to evaluate the hyperlipidemic effect of methanol seed kernel extract of *Mangifera indica* on wistar rats. Mango seed kernels were dried at room temperature before being ground to fine powder. 500 g of mango seed kernel powder was soaked in 500 ml of methanol and shaken intermittently for 72 h after which the extract was concentrated. Twenty five adult male wistar rats were divided into five groups of five rats each. **Group I:** was the normal control and was fed only normal rat chow. **Groups II-V** was induced hyperlipidemia, however, while Group II was not treated with the extract (negative control), **Groups III and IV** were treated with 150 and 350 mg/kg body weight of methanol seed kernel extracts of *Mangifera indica* and Group V the standard drug (atorvastatin). Treatment lasted for 21 days after which rats were sacrificed and blood sample collected subsequently analysed via standard procedures. Hyperlipidemia was characterized by increased levels of total cholesterol and Low Density Lipoprotein (LDL). Oral administration of methanol seed kernel extract of *Mangifera indica* significantly ( $P < 0.05$ ) reduced the aforementioned indices to levels which though were significantly ( $P < 0.05$ ) higher than that reported for the normal control group. On the other hand, it was observed that the levels of high density lipoprotein (HDL) and triacylglyceride (TG) in the negative control (**Group II**) were significantly low but increased following oral administration of extract in a dose dependent manner. It was also observed that extract reduced the weight of hyperlipidemic rats.

**Keywords:** Lipoprotein, Hyperlipidemia, *Mangifera indica*, Atorvastatin, Kernel

## Introduction

Hyperlipidemia is a secondary metabolic dysregulation orchestrated by enhanced levels of plasma lipid including primarily total cholesterol, triglyceride and Low Density Lipoprotein alongside decreased High Density Lipoprotein reason for which atherosclerosis is initiated and progressed [1]. Increased serum levels of triglyceride, cholesterol and LDL have been identified as core risk factors for the untimely development of cardiovascular diseases like hypertension

and coronary heart disease [2] and have been traced to increased uptake of lipid via the gut or enhanced endogenous synthesis of the said molecules.

The use of plants with therapeutic significance has been an integral component of the human health care system since prehistoric times and interest in the practice has improved tremendously, evident by the fact that an estimated 80% global population depend on it to be relieved of one disease or the other [3].

Mango is of the genus *Mangifera* consisting of 30 species of tropical fruiting trees in the flowering family of *Anacardiaceae*. Botanically, it is called *Mangifera indica*. Its application in Ayurvedic medicine dates back to more 4000 years ago. Ayurveda was able to establish that the various parts of this tree have varied medicinal properties. Parts of the tree is rich in mangiferin, a polyphenolic antioxidant with, anti-lipid peroxidation, immunomodulation, cardiogenic, hypotensive, wound healing and anti-degenerative and anti-diabetic properties [4].

Although, it has been established that different parts of *Mangifera indica* (mango) have unique therapeutic ability, it is yet to be known whether or not the seed could be useful in the treatment of hyperlipidemia, hence, the imperativeness of this study is determined.

## **Materials and Methods**

### **Collection and processing of plant material**

Mango (*Mangifera indica*) seed were identified at the herbarium unit of the department of Biological Science, Ahmadu Bello University Zaria Kaduna State. The seed kernel were subsequently dislodged and afterwards dried at room temperature for ten days. The dried seed kernels were pulverized with the aid of mortar and pestle and sieved to obtain a fine powder .The

500 g of powdered plant sample was introduced into a conical flask containing 500 mL of methanol and shaken intermittently for 3 days. The extract was dried in water bath below 40°C

### **Animals**

Adult male wistar rats weighing 120-160 g were procured from the animal house of the Department of Science Laboratory Technology, Akanu Ibiam Federal Polytechnic Unwana Afikpo, Ebonyi State. The rats were housed in well ventilated cages under standard laboratory conditions and were maintained at ambient temperature and relative humidity respectively. They were fed growers mash (Vital feeds Nigeria Ltd) and provided with water. Acclimatization lasted for two weeks after which experiment commenced.

### **Preparation of High Fat Diets (HFD)**

The high fat diet was prepared by mixing 60 ml of cholesterol with 5 g of rat chow feed. (Hassarajani *et al.*, 2007).

### **Animal grouping**

A total number of twenty five adult male wistar rats were for this study. Animals were divided into five groups of rats per group.

**Group I** (Normal group): animals were allowed access to rat chow and water

**Group II** (Negative control): animals were induced hyperlipidemia without treatment

**Group III:** Hyperlipidemic rats were administered with 150 mg/kg of MSKE.

**Group IV:** Hyperlipidemic rats were administered with 350 mg/kg of MSKE.

**Group V:** Hyperlipidemic rats were administered with 4 mg/kg of atorvastatin.

Treatment lasted for 21 days treatment after which rats were anaesthetized by exposure to chloroform, sacrificed and blood sample collected in plain bottles and afterwards centrifuged at 500 rpm for 10 minutes.

### Biochemical assays for lipids

Cholesterol, HDL and triacylglyceride levels were estimated from serum by CHOD-PAP according to the method of Devi and Sharma, (2004), respectively. LDL and HDL were calculated using the method by Johnson *et al.* (1997). While the artherogenic index was calculated by using the method described by Muruganandan et al. (2005)

### Body Weight Measurement

The weight of the rats was determined prior to hyperlipidemia induction, after induction and after treatment. The changes in bodyweight was calculated and recorded.

### Statistical analysis

Data generated from the study was analyzed using statistics software IBM SPSS Statistics 21. Data were expressed as mean  $\pm$  standard deviation (SD). The results were considered as significant at  $P < 0.05$ . Mean values were compared using one way analysis of variance (ANOVA).

**Table 1: Lipid Profile of Hyperlipidemic Rats administered with Methanol Leaf Extract of *M. indica***

Groups	Treatment	TC (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	TG (mg/dl)
Group I	Normal control (NC)	60.23 $\pm$ 2.37 <sup>d</sup>	19.11 $\pm$ 5.09 <sup>a</sup>	56.12 $\pm$ 2.41 <sup>d</sup>	24.62 $\pm$ 4.01 <sup>bc</sup>
Group II	Negative Control	76.10 $\pm$ 3.89 <sup>e</sup>	13.21 $\pm$ 2.01 <sup>ab</sup>	58.50 $\pm$ 3.29 <sup>d</sup>	19.12 $\pm$ 1.85 <sup>a</sup>
Group III	150 mg/kg MLEMI	64.21 $\pm$ 5.54 <sup>bc</sup>	21.13 $\pm$ 2.32 <sup>b</sup>	41.20 $\pm$ 4.86 <sup>b</sup>	23.20 $\pm$ 1.64 <sup>c</sup>

<b>Group IV</b>	300 mg/kg MLEMI	63.10±2.50 <sup>a</sup>	26.12±2.30 <sup>c</sup>	43.5±4.498 <sup>bc</sup>	28.21±1.03 <sup>b</sup>
<b>Group V</b>	Atovarstatin	60.30±2.30 <sup>b</sup>	22.12±3.21 <sup>b</sup>	23.21±1.98 <sup>a</sup>	28.91±3.24 <sup>a</sup>

Results are expressed as mean ± standard deviation of three determinations. Values with the same superscript in column are significantly at P≤0.05.

**Table 2: Effect of Methanol Leaf Extract of *M. indica* on the weight of Hyperlipidemic Rats**

Groups	Treatment	Wt. before induction	Wt. after induction	Wt. after treatment
<b>Group I</b>	Normal control (NC)	145.62±2.34	180.12±3.78	150.65±6.30
<b>Group II</b>	Negative Control	110.11±6.39	161.29±3.75	130.63±5.80
<b>Group III</b>	150 mg/kg MSKE	102.43±5.34	147.13±4.57	104.12±3.28
<b>Group IV</b>	300 mg/kg MSKE	128.56±3.46	185.60±5.01	150.21±3.46
<b>Group V</b>	Atovarstatin	120.22±2.32	176.34±3.60	138.34±6.90

Results are expressed as mean ± standard deviation of three determinations.

## Result and Discussions

For the levels of lipid to be elevated, it is either, the amount of lipid absorbed through the gut is increased or the endogenous synthesis of lipid is enhanced. Thus, in order to reduce hyperlipidemia, it is either the endogenous synthesis is blocked or absorption is decreased. Table 1 shows the lipid profile of hyperlipidemic rats administered with methanol leaf extract of *Mangifera indica* (mango) indicating that feeding high fat diet on animals significantly increased the level of total cholesterol and Low Density Lipoprotein (LDL). However, oral administration of methanol mango seed kernel extract significantly (P<0.05) reduced the aforementioned indices to the levels which though were significantly (P<0.05) higher than that reported for the

normal control group. On the other hand, it was observed that the levels of high density lipoprotein (HDL) and Triacylglyceride (TG) in the negative control (Group II) were significantly low but increased following oral administration of extract in a dose dependent manner. This may be attributed to enhanced inhibition of intestinal absorption of cholesterol, interference with lipoprotein production, increased expression of hepatic LDL receptors and their protection which culminate to leading to increased elimination of LDL-C from the blood and its increased degradation of cholesterol in the body all of which translate to declined serum LDL-C levels which may have also reduced serum cholesterol (TC) levels (Brown and Goldstein, 1981). This is consistent with the findings of Khyati et al (2010) which showed that aqueous leaf extract of *Mangifera indica* significantly ( $P < 0.05$ ) reduced TC, TG, LDL-C, VLDL and significant increase in HDL-C ( $p < 0.05$ ). Table 2 shows the effect of treatment with MLEMI on the weight of hyperlipidemic rats indicating that treatment with extract reduced the weight of hyperlipidemic rats.

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