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Anti-Hyperlipidemic Effect of Methanol Seed Kernel Extract of *Mangifera indica* on Wistar Rat Model

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

The aim of this study was to evaluate the anti-hyperlipidemic effect of methanol *Mangifera indica* seed kernel extract on hyperlipidemic Wistar rats. Mango seed kernels were dried at room temperature before being ground into fine powder. 500 g of mango seed kernel powder was soaked in 500 mL of 98 % 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 administered 2 ml of distilled water. **Groups II-V** were induced hyperlipidemia. However, while **Group II** was not treated with the extract (negative control), **Groups III** and **IV** were

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treated with 150 and 350 mg/kg body weight of mango seed kernel extract (MSKE) and **Group V** was administered the standard drug (atorvastatin). Treatment lasted for 21 days, after which rats were sacrificed and blood sample was collected and subsequently analyzed via standard procedures. Hyperlipidemia was characterized by increased levels of total cholesterol and Low Density Lipoprotein (LDL). Oral administration of MSKE 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 that the levels of high density lipoprotein (HDL) and triacylglyceride (TG) in the negative control (**Group II**) were significantly ($P<0.05$) low but increased following oral administration of extract in a dose dependent manner. It was also observed that MSKE of *M. indica* reduced the body weight of hyperlipidemic rats. In conclusion, it can be deduced from this study that MSKE has the potential to address hyperlipidemia

Keywords: Lipoprotein; hyperlipidemia; mangifera indica; atorvastatin; kernel.

1. INTRODUCTION

Hyperlipidemia is a secondary metabolic dysregulation orchestrated by enhanced levels of plasma lipids, including primarily total cholesterol, triglyceride, Low Density Lipoprotein and 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 a core risk factor for the untimely development of cardiovascular diseases like hypertension and coronary heart diseases [2,3] and have been traced to increased uptake of lipid via the gut or enhanced endogenous synthesis of the said molecules.

Hyperlipidemia is primarily orchestrated by alterations in lifestyle habits which is characterized by fat consumption in excess of 40% of total calories, unsaturated fat intake greater than 10% of total calorie as well as cholesterol intake in excess of 300 mg/day [4].

The use of plants with therapeutic significance has been an integral component of the human health care system which dates back to prehistoric times and interest in the practice has improved tremendously, evident by the fact that an estimated 80% of the global population depends on it to be relieved of one disease or the other [5].

Mango is of the genus *Mangifera* consisting of 30 species of tropical fruiting trees in the flowering family *Anacardiaceae*. Botanically, it is called *Mangifera indica*. Its application in Ayurvedic medicine dates back to more than 4000 years ago. Ayurveda was able to establish that the various parts of mango tree have varied medicinal properties. Parts of the tree are rich in mangiferin, a polyphenolic antioxidant and have served as, anti-lipid peroxidation agent, immunomodulatior, cardiotonic, hypotensive, wound healing agent [6].

Although, it has been established that different parts of *Mangifera indica* (mango) have unique therapeutic abilities, 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 defined.

2. MATERIALS AND METHODS

2.1 Collection and Processing of Plant Material

Mango *(Magnifera indica*) seed was identified at the herbarium unit of the Department of Biological Science, Gregory University Uturu Abia State. The seed kernels 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 fine powder. Exactly 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.

2.2 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 transparent plastic cages under standard laboratory conditions and were maintained at ambient temperature and relative humidity. They were fed grower mash (Vital

feeds Nigeria Ltd) and provided with water *ad-libtum*. Acclimatization lasted for two weeks, after which experiment commenced.

2.3 Preparation of High Fat Diets (HFD)

High fat diet was prepared by mixing 60 mL of cholesterol with 5 g of rat chow feed [7].

2.4 Animal Grouping

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

Group I: (Normal group): animals were orally administered with 2 ml of distilled 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, after which rats were anesthetized with chloroform, sacrificed and blood sample collected in plain bottles. The samples were subsequently centrifuged at 500 rpm for 10 minutes.

2.5 Biochemical Assays for Lipids

Cholesterol, HDL and triacyglyceride levels were estimated from serum by CHOD-PAP according to the method of Devi and Sharma (2004). LDL and HDL were calculated using the method by Johnson *et al*. (1997). While the artherogenic index was calculated using the method described by Muruganandan et al. [8].

2.6 Body Weight Measurement

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

2.7 Statistical Analysis

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

3. RESULTS AND DISCUSSION

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 MSKE indicating that feeding a high fat diet on animals significantly ($P<0.05$) increased the level of total cholesterol and Low Density Lipoprotein (LDL). However,

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 ± 2.37^{d}	19.11 ± 5.09^{a}	56.12 ± 2.41^{d}	24.62 ± 4.01^{bc}
Group II	Negative Control	76.10 ± 3.89^{e}	13.21 ± 2.01^{ab}	58.50 ± 3.29^{d}	19.12 ± 1.85^{a}
Group III	150 mg/kg MSKEMI	64.21 ± 5.54^{bc}	21.13 ± 2.32^{b}	41.20 ± 4.86^{b}	23.20 ± 1.64^{c}
Group IV	300 mg/kg MSKEMI	63.10 ± 2.50^{a}	26.12 ± 2.30^{c}	43.5 ± 4.498^{bc}	28.21 ± 1.03^{b}
Group V	Atovarstatin	60.30 ± 2.30^{b}	22.12 ± 3.21^{b}	23.21 ± 1.98^{a}	28.91 ± 3.24^{a}

Results are expressed as mean ± standard deviation of three determinations. Values with the same superscript in the 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
Group I	Normal control (NC)	145.62±2.34	180.12±3.78	150.65±6.30
Group II	Negative Control	110.11±6.39	161.29±3.75	130.63±5.80
Group III	150 mg/kg MSKE	102.43±5.34	147.13±4.57	104.12±3.28
Group IV	300 mg/kg MSKE	128.56±3.46	185.60±5.01	150.21±3.46
Group V	Atovarstatin	120.22±2.32	176.34±3.60	138.34±6.90

Results are expressed as mean ± standard deviation of three determinations

oral administration of MSKE 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 that the levels of high density lipoprotein (HDL) and triacylglyceride (TG) in the negative control (Group II) were significantly (P<0.05) 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 increased elimination of LDL-C from the blood and increased degradation of cholesterol in the body, all of which translate to declined serum dual-C levels which may have also reduced serum cholesterol (TC) levels [9]. This is consistent with the findings of Khyati et al. [10] which showed that the aqueous leaf extract of *Mangifera indica* significantly (P<0.05) reduced TC, TG, LDL-C, VLDL and significant increased HDL-C (p<0.05). Table 2 shows the effect of treatment with MSKE on the weight of hyperlipidemic rats, indicating that treatment with extract reduced the weight of hyperlipidemic rats.

4. CONCLUSION

Through this study, it has been revealed that mango seed kernel extract has the potential to reverse hyperlipidemia.

CONSENT

It is not applicable.

ETHICAL APPROVALS

Animal Ethical committee approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Ghule BV, Ghante MH, Saoji AN, Yeole PG. Hypolipidemic and antihyperlipidemic effects of *Lagenaria siceraria* (Mol.) fruit extracts, Indian J Exp Biol. 2006;44:905-909.
2. Ansarullah Jadeja RN, Thounaojam MC, Patel V, Devkar RV, Ramachandran AV, Antihyperlipidemic potential of a polyherbal preparation on triton WR 1339 (Tyloxapol) induced hyperlipidemia: A comparison with lovastatin. Int J Green Pharm. 2009;3: 119-124.
3. Xu QY, Liu yH, Zhang Q, Ma B, Yang ZD, Liu L, et.al. Metabolomic Analysis of Simvastatin and Fenofibrate Intervention in High-Lipid Diet-Induced Hyperlipidemia Rats. Acta Pharmacol Sin. 2014; 35: 1265-1273.
4. Durrington PN. Hyperlipaemia. Cambridge:Butterworth-Heinemann Ltd. 1995
5. Shaik A, Elumalai A, Eswaraiah MC. An updated review on hepatoprotective medicinal plants. Journal of Drug Delivery & Therapeutics. 2012;2(2):1-3.
6. Shah KA, Patel MB, Shah SS, Chauhan KN, Parmar PK, Patel NM. Antihyperlipidemic activity of *Mangifera indica* leaf extract on rats fed with high cholesterol diet. Der Pharmacia Sinica. 2010;1(2):156-161.
7. Hassarajani S, Souza TD, Mengi SA,Chattopadhayay.Efficacy study of the bioactive fraction (F3) of Acorus calamus in hyperlipidemia. Indian J Pharmacol. 2007;39:196-200.
8. Muruganandan S, Scrinivasan K, Gupta S, Gupta PK, Lal J. Effect of mangiferin on hyperglycemia and atherogenicity in streptozotocin diabetic rats. J Ethnopharmacol. 2005;97:497- 501.
9. Brown MS, Goldstein JL. N Engl J Med. 1981;305:515.
10. Khyati AS, Mandev BP, Shreya SS, Kajal NC, Parul KP, Natavarlal MP. Antihyperlipidemic activity of *Mangifera indica* leaf extract on rats fed with high cholesterol diet. Der Pharmacia Sinica. 2010;1(2):156-161.

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