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

Abstract

The aim of this study was to evaluate the anti-hyperlipidemiceffect of the methanol seed kernel extract of *Mangiferaindica* on Wistar ratsfed high fat diets. 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 ratswere divided into five groups of five rats each.**Group I**: was the normal control and was fed only normal rat chow.**GroupsII-V**were induced hyperlipidemia. However, while **Group II** was not treated with the extract (negative control), **GroupsIII and IV** were treated with 150 and 350 mg/kg body weight of methanol seed kernel extract(MSKE) of *Mangiferaindica* and **Group V** the standard drug (atorvastatin). Treatment lasted for 21 days, after which rats were sacrificed and blood sample collectedwas subsequently analyzed via standard procedures.Hyperlipidemia was characterized by increased levels oftotal cholesterol and Low Density Lipoprotein (LDL). Oral administration of the methanol seed kernel extractof *Mangiferaindica* 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 (**GroupII**) 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.

Keywords: Lipoprotein, Hyperlipidemia,*Mangiferaindica*, Atorvastatin, Kernel

Introduction

Hyperlipidemia is a secondary metabolic dysregulation orchestrated by enhancedlevels of plasma lipid, includingprimarily total cholesterol, triglyceride and Low Density Lipoproteinanddecreased 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 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 dates back toprehistoric times and interest in the practice has improved tremendously, evident by the fact that an estimated 80%of theglobal populationdepends on it to be relieved of one disease or the other [3].

Mango is of the genus Mangiferaconsisting of 30 species of tropical fruiting trees in the flowering family*Anacardiaceae*. Botanically, it is called *Mangiferaindica*. Its application inAyurvedic medicine dates back tomorethan 4000 years ago. Ayurveda was able to establish that the various parts of the mangotree have varied medicinal properties. Parts of the tree are rich in mangiferin, a polyphenolicantioxidant, anti-lipid peroxidation, immunomodulation, cardiotonic, hypotensive, wound healing and anti-degenerative as well asanti-diabetic properties [4].

Although, it has been established that different parts of *Mangiferaindica* (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.

Materials and Methods

Collection and processing of plant material

Mango *(Magniferaindica*) seedwas identifiedinthe herbarium unit of the department of Biological Science, Ahmadu Bello University Zaria Kaduna State. The seed kernel was subsequently dislodgedand 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.Exactly 500g of powdered plant sample was introduced into a conical flask containing 500mLof methanol and shaken intermittently for 3 days. The extract was dried in water bath below 40^0C

Animals

Adult male Wistar rats weighing 120-160g were procured from the animal house of the Department of Science Laboratory Technology, AkanuIbiam Federal Polytechnic UnwanaAfikpo, Ebonyi State. The rats were housed in well ventilated transparent plasticcages under standard laboratory conditions and were maintained at ambient temperature and relative humidity. They were fedgrowermash (Vital feeds Nigeria Ltd) and provided with water*ad-libtum*. Acclimatization lasted for two weeks, afterwhich experiment commenced.

Preparation of High Fat Diets (HFD)

High fat diet was prepared by mixing 60mL 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 inthis study. Animals were divided into five groups of rats per group.

Group I (Normal group): animals wereallowed access to rat chow and water

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

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

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

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

Treatment lasted for 21 days, afterwhich rats were anesthetized by exposure to chloroform, sacrificed and blood sample collected in plain bottles were subsequently centrifuged at 500 rpm for 10 minutes.

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 (2005).

Body Weight Measurement

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

Statistical analysis

Data generated from the study were analyzed using statisticssoftware IBM SPSS Statistics 21. Data were expressed as mean ± standarddeviation (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 ± 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	150mg/kg MSKEMI	64.21 ± 5.54^{bc}	21.13 ± 2.32^{b}	41.20 ± 4.86^{b}	23.20 ± 1.64^{c}

Group IV	300mg/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 columnare 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	150mg/kg MSKE	102.43±5.34	147.13±4.57	104.12±3.28
Group IV	300mg/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.

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 1shows the lipid profile of hyperlipidemiarats administered with methanol seed kernel extract of *Mangiferaindica* (mango) indicating that feeding a high fat dieton animals significantly(P<0.05) 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 tolevels 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 its increased degradation of cholesterol in the body, allof which translate to declined serum dual-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 the aqueous leaf extract of *Mangiferaindica* 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 MSKEMI on the weight of hyperlipidemiarats, indicatingthat treatment with extract reduced the weight of hyperlipidemic rats.

Conclusions

Through this study, it has been revealed that methanol seed kernel extract has the potential to reverse anti-hyperlipid country.

References

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