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Macrotyloma uniflorum extract improves lipid profile in high fructose fed rats

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Abstract

Backround and Objectives: High fructose feeding induces insulin resistance and hyperinsulinaemia in rats. A role for oxidative stress in the occurrence of insulin resistance has been suggested by several workers. The aim of this study was to investigate the hypolipidemic effect of *M. uniflorum* on high-fructose diet rats that showed characteristic features of insulin resistance. **Materials and Methods:** Male Wistar rats weighing 160–180 g were divided into four groups. The control group received the control diet containing starch. The second group control diet and was administered *M. uniflourm* at dose (1000 mg/kg body weight). The fructose group III was given a high-fructose diet (>60% of total calories). The fourth group were given fructose diet and were administered *M. uniflourm* at dose (1000 mg/kg body weight). **Results:** At the end of 45 days of the experimental period fructose-fed rats displayed resulted in a significant increase in the concentrations of cholesterol, triglycerides (TGs), free fatty acids (FFAs), and phospholipids in plasma, liver, kidney, and skeletal muscle. Reduced activities of lipoprotein lipase (LPL) and lecithin cholesterol acyl transferase (LCAT) and increased activity of the lipogenic enzyme hydroxymethylglutaryl–coenzyme A (HMG-CoA) reductase were observed in plasma and liver. High-density lipoprotein cholesterol (HDL-C) was significantly lowered and very low-density lipoprotein cholesterol (VLDL-C) and low-density lipoprotein cholesterol (LDL-C) were significantly elevated. **Conclusion:** This study demonstrates that of *M. uniflorum* can alter the lipid metabolism in HFFD treated rats and might be implications in the treatment of insulin resistance and hypolipidemic effect of HFFD rats.

Key words: fructose diet, hypolipidemic, insulin resistance, lipid profile, *M. uniflorum*

Abbreviations:

HFFD-High fructose fed diet; HDL- high-density lipoprotein cholesterol HMG CoA– hydroxymethylglutaryl–coenzyme A; LCAT- lecithin cholesterol acyl transferase; LDL - low-density lipoprotein cholesterol; VLDL – very low-density lipoprotein cholesterol.

INTRODUCTION

Diabetes mellitus (DM) is a metabolic disorder characterized by increased glucose level and insulin deficiency and/or defects of insulin action[1]. The consumption of fructose has increased significantly due to the widespread use of high fructose corn syrup as a sweetening agent in food industry. This has received the attention of many investigators since obesity and several aspects of the metabolic syndrome, such as hypertension, dyslipidemia, and glucose intolerance, are strongly correlated with the consumption of high fructose diet. Studies have confirmed that high fructose feeding causes alterations in glucose and lipid metabolism, a substantial decrease in peripheral insulin sensitivity, glucose intolerance, and hypertension[2].

A high-fructose diet (60 g/100 g diet) induces insulin resistance (IR) associated with hyperinsulinaemia and hyperglycaemia in animals[3]. However, dietary fructose is almost totally absorbed and metabolized rapidly by the liver, undergoing a markedly different metabolic fate from glucose metabolism, resulting in deleterious effects, such as IR, obesity and hyperuricemia[4]. We have previously shown that fructose worsens the adverse effects of dietary fats on serum glucose and lipids regulation[5]. The dyslipidemia observed in high fructose-fed rats include elevated triglycerides (TG), free fatty acids (FFA) and lipoprotein abnormalities. These alterations

are secondary to the development of insulin resistance[6]. Hypertriglyceridemia is a major risk factor for cardiovascular diseases[7]. In a review Lam *et al.*,[8] have noted that elevation of free fatty acids (FFA) can increase gluconeogenesis by influencing the gene expression and protein levels of enzymes involved in hepatic glucose metabolism. Pharmaceutical intervention aimed at overcoming fatty acid inhibition of glucose oxidation is found to be beneficial in the treatment of type 2 diabetes[9].

Plant materials have played an important role in the traditional treatment of diabetes, particularly the type 2 form. In many regions of the world, herbal remedies continue to be more accessible and affordable than conventional drugs and represent the first line of treatment available to a diabetes patient. Concurrently, within societies with well-developed, modern health care systems, demand is growing for herbal remedies to complement prescribed, modern therapies for many diseases, including diabetes[10]. The health benefits of horse gram are being recognized in the western world recently, but have been known for its ability to prevent and cure various diseases by Indian "Ayurvedic" system since centuries. Horsegram (*Macrotyloma uniflorum* (Lam.) Verdc) is an important rainfed minor pulse crop. It is a potential grain legume having excellent nutritional and remedial properties with better climate resilience to adapt harsh environmental conditions, grown almost all over the world including East and Northeast Africa, , India, China, Philippines, Bhutan, Pakistan, Sri Lanka and Queensland in Australia[11,12]. They seeds have been used for the treatment of heart conditions, asthma, kidney stones, bronchitis, leukoderma, urinary discharges, hepatoprotective role and obesity[13,14]. Therefore the present study aimed to investigate the role of *M.* uniflorum on the following plasma parameters were taken into consideration: lipid profile total cholesterol, high density lipoproteins (HDL), low density lipoproteins (LDL), triglycerides, glucose, urea, uric acid, creatinine, albumins, total protein concentrations, and alkaline phosphatase activity.

MATERIALS AND METHODS

Animals: Thirty six male adult Wistar strain albino rats, weighing 160-180g were purchased from "Sri Venkateswara Enterprises", Bangalore, Karnataka, India. In 2017 experimental rats were housed in clean sterile polypropylene cages under the constant environmental and nutritional conditions throughout the period of experiment. During the course of the experiments, the temperature was maintained between 22ºC ± 2ºC. The rats were fed on a standard pellet diet and HFFD during experimental period and water *ad libitum.* The experiment was carried out according to the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), New Delhi, India. The work is carried out by biochemistry lab in Bharathidasan university Consitutent college, orathanadu, Tamilnadu, India.

Chemicals: Fructose, bovine serum albumin, G-6-P, γ-glutamyl paranitroaniline, nicotinamide adenine dinucleotide (NAD^+, NADH) nicotinamide adenine dinucleotide phosphate ($NADP^+$, NADPH), reduced glutathione, oxidized glutathione, adenosine triphosphate, adenosine monophosphate and 1,2,4-aminonapthol sulphonic acid were obtained from Sigma Chemical Company, ST. Louis, MO, USA. All other chemicals and reagents used were of highest purity and of analytical grade marketed by Glaxo Laboratories, Mumbai, SD Fine Chemicals, Mumbai and Sisco Research Laboratories, Pvt. Ltd., India.

After one week of acclimatization the animals were divided into two batches. One batch was provided with a control diet containing starch as the source of carbohydrate (groups I and II) and the other was fed a fructose-enriched diet for 45 days (Groups III-VI). Different composition of diet (Table 1) given to all the rats for 45 days followed by *M. uniflorum* was given orally for 15 days. Blood samples from all the groups of animals were collected from the tail vein on the 10th, 20th and 30th days and estimated glucose levels to ensure diabetic status.

Experimental Design: The rats were divided into six groups, each group consisting of six rats. *M. uniflorum* seed powder dissolved in water and given to rats twice daily for a period of 15 days, after 45 days of control and HFFD.

Group I : Normal control rats (for 45 days)

Group II : Control rats treated with *M. uniflorum* seeds (1000 mg/kg) twice daily for 15 days

Group III : High Fructose fed rats (>60% fructose for 45 days)

Group IV : HFFD + *M. uniflorum* seeds (1000 mg/kg) twice daily for 15 days

Collection of Samples: At the end of the 45th day, all the rats were fasted overnight and sacrificed by cervical decapitation under mild ether anesthesia. Blood was collected tube with heparin and plasma was separated by centrifugation. The liver tissue was immediately removed and washed in ice- cold saline to remove blood. The tissues were sliced and homogenized in 0.1 M Tris- HCl buffer (pH 7.0). The homogenates were centrifuged at 1000 rpm for 10 min at 4ºC in a cold centrifuge.

Table 1: Composition of diets fed to rats for the determination of insulin resistance

Ingredient (g/100 g)	Control diet	High-fructose diet
Corn starch	60	-
Fructose	-	60
Casein	20	20
Methionine	0.7	0.7
Groundnut oil	5	5
Wheat bran	10.6	10.6
Salt mixture†	3.5	3.5
Vitamin mixture‡	0.2	0.2

†Composition of the mineral mix (g/kg): $MgSO_4.7H_2O$, 30.5; NaCl, 65.2; KCl, 105.7; KH_2PO_4, 200.2; $3MgCO_3.Mg(OH)_{2.3}H_2O$, 38.8; $FeC_6H_5O_7.5H_2O$, 40.0; $CaCO_3$, 512.4; KI, 0.8; NaF, 0.9; $CuSO_4.5H_2O$, 1.4; $MnSO_4$, 0.4; and $CONH_3$, 0.05.

‡One kilogram of vitamin mix contained: thiamine mononitrate, 3 g; riboflavin, 3 g; pyridoxine HCl, 3.5; nicotinamide, 15 g; d-calcium pantothenate, 8 g; folic acid, 1 g; d-biotin, 0.1 g; cyanocobalamin, 5 mg; vitamin A acetate, 0.6 g; α-tocopherol acetate, 25 g; and choline chloride, 10 g.

Biochemical estimation: Lipids in plasma and tissues were extracted by the method of Folch and colleagues[15]. Total lipids were extracted with chloroform methanol mixture 2:1 (v/v) after homogenizing the tissue. Aliquots of the lipid extracts were evaporated to dryness and used for the estimation of lipids. Estimation of cholesterol, TGs, phospholipids, FFAs, and concentration of cholesterol in plasma lipoproteins were carried out following the procedures described earlier[16].

High-density lipoprotein–cholesterol (HDL-C) was estimated using a commercial kit (Agappe Diagnostics Pvt, Ltd). Very low-density lipoprotein–cholesterol (VLDL-C), and lowdensity lipoprotein–cholesterol (LDL-C) were obtained by the following calculations:

VLDL-C = triglycerides/5

LDL-C = total cholesterol - (HDL-C + VLDL-C).

Lipoprotein lipase (LPL) was assayed in plasma by the method of Korn[17]. Lecithin cholesterol acyl transferase (LCAT) was assayed in plasma by the method of Hitz and colleagues[18]. Hydroxymethylglutaryl–coenzyme A (HMGCoA) reductase activity was measured by the method of Rao and Ramakrishnan[19].

Statistical analysis: Values or mean ± SD for six rats in the each group and statistically significant differences between mean values were determined by one way analysis of variance (ANOVA) followed by DMRT values of $P < 0.05$ was considered to be significant. Statistical Package for Social Studies (SPSS Inc., Chicago, IL)) 19.0 versions were used for this analysis.

RESULTS

The concentration of plasma lipids in control and experimental animals were given in Table 2. Fructose-fed rats had elevated levels (P<0.05) of cholesterol, TG, FFA, Phospholipids in plasma and liver as compared to control rats. Supplementation of *M. uniflorum* significantly (P<0.05) reduced the levels of cholesterol, TG, FFA and phospholipids group IV (HFFD +*M. uniflorum*) as compared to HFFD rats. There is no difference between group I and II.

Concentration of plasma total cholesterol and that in lipoprotein fractions was given in Table 3. In HFFD rats (group III) Significant increases in (P<0.05) VLDL-C and LDL-C concentrations and a decrease in HDL-C as compared to control rats (group I) . In *M. uniflorum* supplemented fructose-fed rats LDL-C and VLDL-C were lower while HDL-C was higher as compared to fructose-fed rats. But no differences between group I and II.

The activities of LPL, LCAT in plasma, and HMG-CoA reductase in liver were presented in the Table 4. The activities of LPL andLCAT were significantly lowered (P<0.05) in plasma and liver of fructose-fed rats. There was an increased activity (P<0.05) of HMG-CoA reductase in fructose-fed group. The activities of these enzymes were restored to baseline control values when rats were treated with *M. uniflourm*. There is no difference between group I and II.

M. uniflorum seed extract (1000 mg/kg of B.W) is effective dose for all parameters significant effect in HFFD rats as compared to control rats. *M. uniflorum* in normal control rats didn't show any significant.

DISCUSSION

High level of total cholesterol is one of the major risk factors for coronary heart diseases and it is well known that hyperlipidemia and the incidence of atherosclerosis are increased in diabetes[20]. Insulin resistance in T2DM is also associated with hyperlipidemia and atherosclerosis[21]. Fructose-fed rats in the present study exhibited hypercholesterolemia and hypertriglyceridemia. The fructose-induced dyslipidemia may be attributed to the increased de novo hepatic lipogenesis through providing large amounts of hepatic triose-phosphate for fatty acid synthesis[22]. In our earlier studies as well as previous researcher studies also reported that fructose administration can have profound the effects on plasma and tissue lipids levels[23]. The present study investigated the biochemical important effects of *M. uniflourm* on HFFD induced metabolic complications in rats. *M. uniflourm* significantly reduced fructose induced increment in the body weight and relative organ weights (liver and adipose tissue weight). With regard to reports that long-lasting fructose consumption could cause adaptive changes in healthy animals that in turn masked syndromes of such disturbances as in lipid metabolism[24].

Significant increases (P<0.05) in VLDL-C and LDL-C concentrations and a decrease in HDL-C were observed in fructose-fed rats as compared to control rats. In *M. uniflorum* supplemented fructose-fed rats. LDL-C and VLDL-C were lower while HDL-C was higher as compared to fructose-fed rats. High-fructose feeding caused increased plasma triacylglycerol (TG) and VLDL-C, lipid indices and decreased HDL-C. These results were consistent with the previous study[25]. Fructose feeding may lead to hypertriglycerdemia by increasing the formation of glycerol-3-phosphate, a precursor of lipid synthesis. Hypertriglyceridemia may also arise due to defect in removal of VLDL from plasma or increased secretion of VLDL in the liver. Lipoprotein lipase is an important enzyme responsible for the hydrolysis of TG from chylomicrons and LDL. Adminstration of *M. uniflorum* may depend on its TG, LDL and VLDL lowering effect and its insulin sensitivity effects and prevents fructose-induced cholesterol accumulation

In our study shows the . The activities of LPL and LCAT were significantly lowered (P<0.05) in plasma and liver of fructose-fed rats. There was an increased activity (P<0.05) of HMG-CoA reductase in fructose-fed group. The activities of LPL andLCATwere lowered in plasma and liver of fructose-fed rats. There was an increased activity of HMG-CoA reductase in fructose-fed group. The activities of these enzymes were restored to baseline control values when rats were treated with *M. uniflourm*. The lowered HDL-C concentration in fructose-fed rats can be attributed to the decreased LPL and LCAT activities in plasma. LCAT, the enzyme that catalyzes esterification of cholesterol with FFAs, along with LPL is responsible for HDLC synthesis. It plays an important role in cholesterol and TG transport and metabolism. The decreased activity of LCAT indicates impairment in HDL-C synthesis as well as TG metabolism in fructose-fed rats. The effect of fructose

feeding on LCAT, LPL, and HMG-CoA reductase produces changes in lipid components, mainly in the concentrations of cholesterol, TGs, HDL-C, and VLDL-C. Previous researchers[26] demonstrated higher plasma cholesterol and LDL-C concentration in healthy and hyperinsulinemic subjects after 4 weeks of high fructose diet. In the present study, the *M. uniflourm* treatment to fructose-fed rats resulted in a favorable lipid profile. There is also accumulating evidence for the hypolipidemic effects.

Insulin has a regulatory effect of on FFA metabolism. A defect in the ability of insulin to regulate the FFA metabolism could contribute to increased FFA levels in fructose - fed rats. Elevated concentration of plasma FFA may play a key role in the pathogenesis of type 2 diabetes, by impairing peripheral glucose utilization and by promoting hepatic glucose overproduction[27]. Cellular and membrane phospholipids are the major targets of damaging free radicals and therefore depletion of phospholipids in liver of high fructose-fed rats could attributed to oxidative stress[28]. Previous researchers studies have shown that fructose facilitates oxidative damage in tissues[29]. Further a positive correlation between the levels of lipid peroxidation products and IR has been documented[30].

Fructose is a highly lipogenic nutrient. Excessive FFA delivery to muscle from the circulation can be a source of muscle TG accumulation. The unregulated fructose metabolism generates both glycerol and acyl portions of acyl-glycerol molecules, the substrates for TG synthesis. Increase in acyl CoA carboxylase and diacylglycerol acyl transporter activities has been reported in liver of a similarmodel system, the fructose-fed hamster[31]. The increase in muscle TG store could also be linked to impaired removal due to decreased tissue lipoprotein lipase activity in FRU rats[25]. Supplemention of *M. uniflourm* increased the levels of LPL, LCAT and HMG CoA. It may be *M. uniflourm* having activity of hepatic lipase is blocked in these rats due to hepatic IR.

A regulatory protein, called sterol regulatory element binding protein, binds to sterol responsive elements found on multiple genes, and activates a cascade of enzymes involved in lipid biosynthesis pathway such as HMG-CoA reductase and fatty acid synthase. The activity of this protein in liver is reported to be enhanced in insulin resistant fructosefed mice[32] and this explains the increased levels of cholesterol and fatty acids during fructose feeding.

Treatment of *M. uniflourm* reduction in Total cholesterol, TGs and FFAs could be attributed to the insulin-potentiating actions of spices, since the regulatory enzymes of lipid metabolism are influenced by insulin.

CONCLUSION

The results of the present study suggest that *M. uniflorum* has modulatory effects on lipid metabolism in fructose-fed insulin-resistant rats. Risk factors for cardiovascular disease, such as dyslipidemia, hypertension, and glucose intolerance, tend to cluster within individuals and insulin resistance is a common feature in these conditions. The authours spectulated that *M. uniflorum* seed extract (1000 mg/kg of B.W) is many advantageous biochemical effects and can serve as a promising component of functional food and modulate the lipid profile and exhibit potent hypolipidimic, active aginest HFFD rats.

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Table 2: Concentration of cholesterol, TG, FFA, and phospholipids in plasma of the experimental animals.

Parameters	Control	Control (1000 mg/ kg)	HFFD control	HFFD+MUF (1000 mg/ kg)
Total cholesterol Plasma (mg/dL)	92.8±4.9 [a]	91.5±4.7 [a]	134.5±18.7 [b]	95.1±9.4 [c]
Liver (mg/100 mg tissue)	281.1±20.2[a]	279.9±19.8[a]	349.6±24.2 [b]	289.2±20.7 [c]
TG Plasma (mg/dL)	96.5±7.14 [a]	95.7±7.45 [a]	161.6±12.25 [b]	99.72±7.52 [c]
Liver (mg/100 mg tissue)	242.9±20.3 [a]	241.1±20.4 [a]	298±22.17 [b]	245.74±22.16 [c]
FFA Plasma (mg/dL)	96.1±7.5 [a]	95.4±8.4 [a]	146.8±10.8 [b]	99.7±8.5 [c]
Liver (mg/100 mg tissue)	128.1±9.8 [a]	127.4±9.6 [a]	168.5±14.2 [b]	130.2±10.8 [c]

Each value is mean ± S.D. for six rats in each group.
Values not sharing a common superscripts differ significantly at $p < 0.05$ (DMRT).

Table 3: Concentration of HDL, LDL and VLDL in plasma of the experimental animals.

Parameters	Control	Control (1000 mg/ kg)	HFFD control	HFFD+MUF (1000 mg/ kg)
HDL (mg/dL)	52.5±3.6 [a]	52.5±4.7 [a]	34.2±1.7 [b]	52.1±3.4 [c]
LDL (mg/dL)	12.6±0.2[a]	12.0±19.8[a]	63.6±24.2 [b]	12.9±20.7 [c]
VLDL (mg/dL)	18.2±0.9 [a]	18.2±0.7 [a]	32.6±2.7 [b]	17.9±0.9 [c]

Each value is mean ± S.D. for six rats in each group.
Values not sharing a common superscripts differ significantly at $p < 0.05$ (DMRT).

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Table 4: Activities of lipoprotein lipase (LPL) and lecithin cholesterol acyl transferase (LCAT) in plasma and hydroxymethylglutaryl (HMG)-CoA reductase in liver

Parameters	Control	Control (1000 mg/ kg)	HFFD control	HFFD+MUF (1000 mg/ kg)
LCAT (μmoles of cholesterol/hr/L)	79.5±3.3 [a]	78.2±3.2 [a]	60.4±3.7 [b]	78.9±3.4 [c]
LPL (μmoles of glycerol liberated/hr/L)	6.2±0.4[a]	6.0±0.4[a]	4.6±24.2 [b]	6.1±0.4 [c]
HMG-CoA reductase (Ratio of HMG-CoA to mevalonate)	3.6±0.9 [a]	3.6±0.7 [a]	2.2±2.7 [b]	3.5±0.9 [c]

Each value is mean ± S.D. for six rats in each group.
Values not sharing a common superscripts differ significantly at $p < 0.05$ (DMRT).