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Lipidemic Properties of *Sorghum vulgare* Leaf Sheath on Oxidative Markers and Heart Function Enzymes of Dyslipidemic Wistar-Albino Rats

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Abstract

Dyslipidemia is an abnormal amount of lipids such as triglycerides, cholesterol and/or fat phospholipids in the blood. The aim of this study was to investigate the lipidemic properties of *Sorghum vulgare* leaf sheath on oxidative markers and heart function enzymes on high-fat-diet induced dyslipidemic wistar-albino rats. Thirty-six (36) wistar-albino rats weighing 110-130 g were used for the study. The animals were distributed randomly into six groups of six animals each. Group 1 (control), group II to group VI were fed with high fat diet; group II (untreated), groups III to V received 400 mg/kg, 800 mg/kg and 1200 mg/kg aqueous extract of *Sorghum vulgare* leaf sheath respectively while group VI was treated with atorvastatin (0.2 mg/kg) which is a standard drug. The results of the study showed that aqueous extract of *Sorghum vulgare* leaf sheath (especially at 800 mg/kg) significantly reduced ($p \leq 0.05$) body-weight, triglyceride concentration, very low density lipoprotein concentration, creatinine concentration and Lactate dehydrogenase activity. Malondialdehyde concentration, cholesterol concentration, glutathione peroxidase activity, creatine kinase activity and superoxide dismutase activity do not differ significantly ($p \leq 0.05$). The results suggest that *Sorghum vulgare* leaf sheath has myocardial protective properties.

Keywords: *Sorghum vulgare*; Wistar-Albino rats; Oxidative markers

Introduction

Dyslipidemia is the elevation or attenuation of plasma lipoproteins. Metabolic disorders that involve elevations in any lipoprotein species are called hyperlipidemia or hypolipoproteinaemia [1]. Hyperlipidemia is characterized by high plasma concentrations of total cholesterol, low density lipoproteins (LDL), very low density lipoproteins (VLDL), triglycerides and reduced high density lipoproteins (HDL) concentrations. Hyperlipidemia is one of the risk factors contributing to the prevalence of atherosclerosis, stroke, coronary heart disease [2] and acute pancreatitis (caused majorly by hypertriglyceridemia) [3].

Synthetic drugs used in the treatment of dyslipidemiahave side effects [2] such as myopathy and hepatic dysfunction [1]. Hence use of herbal plants which have low side effects, cheap and readily available is of great importance [2].

Oxidative stress is highly correlated with a wide variety of inflammatory and metabolic disease states. It is highly correlated with cumulative damage in the body by free radicals when inadequately neutralized by antioxidants. Free radicals may adversely affect cell survival because of membrane damage through the oxidative damage of lipid, protein and irreversible DNA modification. Furthermore oxidative damage is aggravated by the decrease in antioxidant enzymes activities such as superoxide dismutase, catalase (CAT), glutathione S-transferase (GST), and glutathione peroxidase (GPx) which acts as a free radical scavengers in conditions associated with oxidative stress [4]. Hypertriglyceridemia and hypercholesterolemia was reported to be responsible for oxidative modification of LDL, protein glycation, glucose-autioxidation with excess production of free radicals and lipid peroxidation products, which may represent major risk factors for ischemic heart diseases.

Sorghum vulgare also called guinea corn is a member of the Poacea family which can strive in hot area. Phytochemicals present in *Sorghum vulgare* leaf sheath include tannins, flavonoids and phytate [5]. Phytate has shown to exhibit anti-inflammatory and cholesterol lowering effects. It also has antioxidant properties and metal chelating properties [6]. Flavonoids have antioxidant properties that can help in the reduction of oxidative stress that is associated with hyperlipidaemia [7].

Methodology

Collection and Preparation of aqueous extract of the plants

The dry plant was gotten from mile 3 market Port Harcourt, Nigeria. The plant was ground into fine powder with a blender and stored in an air tight container. The ground powder was macerated in distilled water for 12 h (1 kg/1 L). The macerate was filtered using Whatman filter paper (No 1), and the filtrate was concentrated using a water bath (60°C) to obtain concentrated crude extract. The extract was stored in a freezer until further use [8].

Experimental animals

Thirty-six (36) Wister-albino rats weighing between 110-130 g were used for the study. The experiment lasted for a period of forty-two days. The animals were acclimatized for a period of 14 days before use. Following acclimatization, the animals were distributed randomly into six groups of six animals each. Group 1 was the control and fed with growers feed only. Group II to group VI were induced with hyperlipidemia using high fat diet (10 g of egg yolk per 40 g of the feed) for a period of four weeks. At the third week of inducement of hyperlipidemia with high fat diet, treatment of the animals with the

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plant's extract and standard drug commenced for a period of two weeks. Group II was untreated; group III to V received 400 mg/kg, 800 mg/kg and 1200 mg/kg of the plant's aqueous extract respectively while group VI was treated with atorvastatin (0.2 mg/kg). At the end of the study, the animals were sacrificed and their blood samples were collected for biochemical study.

Biochemical studies

Total Cholesterol (TC), Triglycerides, (TG), Low Density Lipoprotein Cholesterol (LDL), High Density Lipoprotein Cholesterol (HDL) were assayed using Randox Kits (Randox laboratories Ltd, Crumlin, England, UK).MDA level was assessed using thiobarbituric acid reactive substances as described by Ohkawa, et al. [9]. Superoxide dismutase was measured according to Misra and Fridovich method [10]. GPx activity estimation was based on the rate of H_2O_2 consumption as described [11,12]. Creatine kinase and lactate dehydrogenase was determined using Seralyzer method [13]. Creatinine was determined by colorimetric method and urea concentrations were determined by urease-Berthelot method.

Statistical analysis

Data was represented as Mean ± SEM, and subjected to One-way Analysis of Variance (ANOVA) using Statistical software SPSS. A level of $p \leq 0.05$ was considered as statistically significant.

Results and Discussion

Obesity is a pathological condition in which there is accumulation of excess body fat, which results by taking more calories in diet than are expanded by the body's consuming activities. Arthrosclerosis and cardiac complications are more common among obese individuals [4].

The results on Table 1 show the weights of the hyperlipidemic animals. There was significant reduction ($p \leq 0.05$) of animals treated with 800 mg/kg of aqueous extract of *Sorghum vulgare* leaf sheath when compared to the control and hyperlipidemic animals.

Blood lipids as a whole participate as a key intermediate in atherogenesis [14]. The results of the aqueous extract of *Sorghum vulgare* leaf sheath on lipid profile of high fat diet induced dyslipidemic rats is shown in Table 2. There was significant reduction ($p \leq 0.05$) of triglyceride and VLDL concentration of all the groups compared to the hyperlipidemic untreated group. Indicating that the extract can be used in treating cardiovascular diseases. However, there was no significant reduction ($p \leq 0.05$) in total cholesterol, HDL and LDL levels of all the treated groups.

High fat diet has been reported to induce oxidative stress by causing lipid peroxidation. Lipid peroxidation is initiated by free radical attack on membrane polyunsaturated fatty acids leading to their transformation and fragmentation to alkanes and aldehyde reactive compounds. SOD is a primary oxygen radical scavenger of tissues converting the super oxide anion radical to H_2O and H_2O_2. GPx acts as scavenger of hydrogen peroxide and other hydroperoxides (H_2O_2 into H_2O and O_2) [15]. However, imbalance between the formation of reactive oxygen species and their elimination occasioned by dyslipidemia has been implicated in oxidative-induced diseases [16].

The results of the present study showed no significant difference ($p \leq 0.05$) in all the groups for MDA and GPx levels (Table 3). But there was significant decrease($p \leq 0.05$) in hyperlipidemic rats compared to the control of SOD level (Table 3) and an increase in those animals treated with800 mg/kg of the plant extract when compared to the hyperlipidemic rats, though not significantly different ($p \leq 0.05$).

Lactate dehydrogenase, creatine kinase and aspartate amino transferase can be used as biomarkers of heart function. Lactate dehydrogenase is an enzyme that in all cells in the body mainly; liver, heart, kidneys, muscle and erythrocytes. LDH levels may be increased whenever there is cell necrosis or when neoplastic proliferation of cells causing an increase in LDH production. Elevated LDH activity also indicates tissue damage [17].

Creatine kinase (CK), is an intracellular enzyme present in greatest amounts in skeletal muscle, myocardium, and brain; smaller amounts occur in other visceral tissues. Disruption of cell membranes due to hypoxia or other injury releases CK from the cellular cytosol into the systemic circulation. Elevated CK activity can also be due to skeletal muscle damage and excessive exercise, myositis, and nutritional myopathy [17]. Aspartate aminotransferase (AST) leaks when the liver, heart, skeletal muscle or erythrocytes are injured [18]. It non-specific for liver damage but can also be used as cardiac marker.

Groups	Initial weight (g)	After acclimatization (g)	Weight after two weeks of inducing hyperlipidemia (g)	Weights of animals after treatment with aqueous extract of *Sorghum vulgare* (g)
Control	$130.00^{a} \pm 0.00$	$131.16^{a} \pm 1.66$	$139.16^{a} \pm 0.83$	$140.00^{a} \pm 2.04$
HFD	$118.30^{b} \pm 1.05$	$126.60^{a} \pm 2.10$	$134.16^{b} \pm 1.53$	$140.00^{a} \pm 2.04$
HFDATV	$123.30^{b} \pm 1.66$	$125.00^{a} \pm 0.84$	$132.50^{b} \pm 1.11$	$145.00^{a} \pm 2.04$
HFD400	$117.50^{b} \pm 1.11$	$128.33^{a} \pm 1.66$	$134.16^{b} \pm 1.53$	$135.00^{ac} \pm 2.04$
HFD800	$122.50^{b} \pm 2.14$	$123.30^{a} \pm 2.10$	$132.50^{b} \pm 1.11$	$132.50^{abc} \pm 1.44$
HFD1200	$122.50^{b} \pm 1.11$	$125.00^{a} \pm 2.23$	$135.83^{b} \pm 0.83$	$136.25^{a} \pm 2.39$

Values are expressed as Mean ± SEM. Values in a column with the same alphabetical superscript do not differ significantly ($p \leq 0.05$). Values in a column with different alphabetical superscript differ significantly ($p \leq 0.05$).

Table 1: Weights of the hyperlipidemic animals.

Groups	TC (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
Control	$3.60^{a} \pm 0.14$	$2.05^{a} \pm 0.06$	$0.56^{a} \pm 0.07$	$1.77^{a} \pm 0.28$	$0.41^{a} \pm 0.01$
HFD	$3.95^{a} \pm 0.17$	$2.90^{b} \pm 0.17$	$0.79^{a} \pm 0.06$	$0.52^{b} \pm 0.30$	$0.58^{b} \pm 0.03$
HFDATV	$4.22^{a} \pm 0.18$	$2.30^{a} \pm 0.25$	$0.67^{a} \pm 0.01$	$1.62^{a} \pm 0.11$	$0.46^{a} \pm 0.05$
HFD400	4.55 ± 0.80	$2.15^{a} \pm 0.06$	$0.81^{a} \pm 0.07$	$1.80^{a} \pm 0.17$	$0.43^{a} \pm 0.01$
HFD800	$3.62^{a} \pm 0.35$	$2.15^{a} \pm 0.17$	$0.99^{a} \pm 0.09$	$0.84^{b} \pm 0.44$	$0.43^{a} \pm 0.03$
HFD1200	$4.02^{a} \pm 0.11$	$2.22^{a} \pm 0.06$	$0.98^{a} \pm 0.06$	$0.81^{b} \pm 0.12$	$0.44^{a} \pm 0.01$

Values are expressed as Mean ± SEM. Values in a column with the same alphabetical superscript do not differ significantly ($p \leq 0.05$). Values in a column with different alphabetical superscript differ significantly ($p \leq 0.05$).

Table 2: Results of the aqueous extract of *Sorghum vulgare* leaf sheath on lipid profile of high fat diet induced dyslipidemic rats.

Groups	MDA (U/L)	GPx (U/L)	SOD (U/L)
Control	$1.82^{a} \pm 0.23$	$0.25^{a} \pm 0.22$	$0.69^{a} \pm 0.07$
HFD	$1.42^{a} \pm 0.29$	$0.30^{a} \pm 0.01$	$0.12^{b} \pm 0.01$
HFDATV	$2.45^{ab} \pm 0.27$	$0.23^{a} \pm 0.02$	$0.25^{b} \pm 0.05$
HFD400	$1.92^{a} \pm 0.12$	$0.24^{a} \pm 0.03$	$0.17^{b} \pm 0.03$
HFD800	$1.42^{a} \pm 0.27$	$0.25^{a} \pm 0.03$	$0.23^{b} \pm 0.06$
HFD1200	$1.62^{a} \pm 0.27$	$0.34^{a} \pm 0.03$	$0.19^{b} \pm 0.04$
Values are expressed as Mean ± SEM. Values in a column with the same alphabetical superscript do not differ significantly ($p \leq 0.05$). Values in a column with different alphabetical superscript differ significantly ($p \leq 0.05$).			

Table 3: Results of the aqueous extract of *Sorghum vulgare* leaf sheath on oxidative markers on high fat diet induced dyslipidemic rats.

Groups	LDH	CK
Control	$14.22^{bc} \pm 4.18$	$4.40^{bc} \pm 1.62$
HFD	$22.40^{b} \pm 2.05$	$6.07^{b} \pm 0.36$
HFDATV	$31.65^{b} \pm 1.54$	$7.95^{be} \pm 0.17$
HFD400	$23.22^{b} \pm 2.94$	$6.65^{b} \pm 0.50$
HFD800	$9.77^{bc} \pm 2.94$	$5.50^{a} \pm 0.38$
HFD1200	$23.75^{b} \pm 0.84$	$6.87^{b} \pm 0.28$
Values are expressed as Mean ± SEM. Values in a column with the same alphabetical superscript do not differ significantly ($p \leq 0.05$).Values in a column with different alphabetical superscript differ significantly ($p \leq 0.05$).		

Table 4: Heart function enzymes of dyslipidemic animals treated with aqueous extract of *Sorghum vulgare* leaf sheath.

Groups	UR (mmol/L)	CR (µmol/L)	AST (U/L)
Control	$5.67^{a} \pm 0.19$	$115.50^{a} \pm 6.88$	$172.25^{bc} \pm 2.25$
HFD	$3.22^{b} \pm 0.10$	$130.00^{b} \pm 1.87$	$190.05^{b} \pm 3.22$
HFDATV	$3.56^{b} \pm 0.28$	$152.00^{ab} \pm 2.85$	$203.50^{b} \pm 2.25$
HFD400	$2.72^{b} \pm 0.10$	$144.00^{b} \pm 9.54$	$177.50^{bc} \pm 4.29$
HFD800	$4.12^{b} \pm 0.10$	$117.75^{a} \pm 5.37$	$167.00^{bc} \pm 2.04$
HFD1200	$3.97^{b} \pm 0.36$	$140.00^{b} \pm 4.14$	$191.75^{b} \pm 3.14$
Values are expressed as Mean ± SEM. Values in a column with the same alphabetical superscript do not differ significantly ($p \leq 0.05$). Values in a column with different alphabetical superscript differ significantly ($p \leq 0.05$).			

Table 5: Results of the aqueous extract of *Sorghum vulgare* leaf sheath Kidney function indices on high fat diet induced dyslipidemic rats.

Table 4 shows the results of the heart function enzymes of dyslipidemic animals treated with aqueous extract of *Sorghum vulgare* leaf sheath. There was a significant reduction ($p \leq 0.05$) of LDH activity of hyperlipidemic animals treated with 800 mg/kg of aqueous extract of *Sorghum vulgare* leaf sheath and the control group when compared to the high fat diet untreated animals. In addition, there was reduction though not significant ($p \leq 0.05$) of CK activity of hyperlipidaemic animals treated with 800 mg/kg of aqueous extract of *Sorghum vulgare* leaf sheath and control group when compared to the hyperlipidemic untreated animals. Furthermore, there was significant reduction ($p \leq 0.05$) in AST activity of hyperlipidemic animals treated with 400 mg/kg and 800 mg/kg of aqueous extract of *Sorghum vulgare* sheath when compared to the hyperlipidemic untreated animals and the hyperlipidemic animals treated with 0.2 mg/kg of atorvastatin. The result suggests that aqueous extract of *Sorghum vulgare* leaf sheath may have myocardial protective effect.

Chronic kidney disease (CKD) is defined by reduction in Glomerular Filtration Rate (GFR). As GFR declines, urinary excretion of urea and creatinine also reduces and blood concentration of creatinine and urea increases [19]. From the results presented on Table 5, there was significant reduction ($p \leq 0.05$) of creatinine concentration for animals treated with 800 mg/kg aqueous extract of *Sorghum vulgare* leaf sheath when compared to the hyperlipdemic untreated animals. The urea concentration of the treated animals was within range (2.8-8.9 mmol/L) for all the groups [20-22].

Conclusion

The results suggest that aqueous extract of *Sorghum vulgare* leaf sheath (at 800 mg/kg) has anti-hypertriglyceridemic properties, nephroprotective and myocardia-protective effect.

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