Evaluation of the Influence of oral administration of*Sidaacuta* on Lipid Profile and Body Weight of Wistar Rats

Abstract

The aim of this study was to evaluate the influence of oral of *Sida acuta* on hepatic damage, lipid profile and body weight of wistar rats. Freshly harvested *Sida acuta* was dried under room temperature and afterwards ground to powder which was subsequently processed into extract. Thirty adult male wistar rats were divided into five groups of five rats. Groups I (normal control) was administered with 2 ml of distilled water, groups II–VI were induced hepatic damage. While group II was left untreated, groups III-V were administered with 100, 200 and 300 mg/kg of extract respectively. Group VI was administered silymarin (standard drug). Treatment lasted for 28 days after which animals were humanely sacrificed and blood sample collected for analysis. Treatment with extract significantly ($P<0.05$) reduced the activities of liver enzymes; aspartate aminotransferase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP). While the serum levels of TC, TG, HDL and LDL reported for group II were significantly ($P<0.05$) higher than those reported for the normal control, oral administration of extract significantly ($p<0.05$) reduced the level of TC, TG, HDL and LDL reported for groups III-V. Oral administration of extract did not adversely affect the body weight of animals. In conclusion, it can be deduced from the study that oral administration of *Sida acuta* reversed hepatic damage, and distorted lipid profile and did not result in weight loss.

Keywords: Lipid, Liver, Enzymes, Silymarin, Lemongrass

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Introduction

The liver is not only the largest organ of the body but also one the organs critical to the human organism. It plays cardinal roles inlipid synthesis and transportation among numerous functions [1]. Thus, it is not in doubt that impaired liver functioncan translate to abnormal lipid profile [2]; [3].

Human exposure to pollutants such as carbon black resulting from the incomplete combustion of fossil fuel as well as diesel exhaust particles discharged by the diesel powered trucks and

automobiles constitute a better part of the atmospheric particulate matter in urban centers has tremendously increased and have been positively correlated with increased human mortality through various diseases notably hepatic damage among others through oxidative stress [4].Conventionally, treatment of liver damage involves the use of synthetic drugs which may further hamper hepatic health and in some serious case [5].

The earth is endowed with diverse plants of therapeutic values that are globally utilized by an estimated 80% of the world's population in pursuance of health care needs [6].*Sida acuta*commonly referred to as the malvaceous weed is abundantly around improved pastures, wastes, disturbed places and roadsides [7].

Although native to Mexico and Central Africa, it can now be seen throughout the tropics and subtropics [8]. In addition to the fact that it has been used successfully in the treatment of diseases such as fever, headache, skin disease, diarrhea and dysentery [1] numerous pharmacological activities such anti-plasmodial, cytotoxic and antioxidant [9] are attributed to the said plant.

Materials and Methods

Collection of plant material

Fresh leaves of *Sida acuta* were collected from a bush within a residential area in AmaokweUturu, Isiukwuato Local Government Area of Abia State in the Southeastern part of Nigeria. The plant was transported in a dark polythene bag to the herbarium unit of the Department of Forestry, Michael Okpara University of Agriculture, Umudike Abia State, and Southeast Nigeria for identification.

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Preparation of Extract

The leaves of *Sida acuta* were properly washed with the aid of clean tap water in order to remove dirt. This was followed by drying at room temperature. Afterward, the leaves were ground and sieved to fine powder. Exactly 500 g of powdered plant sample was subsequently steeped in 3 L of distilled water for 24 hr. The mixture was filtered with a clean sieve and was concentrated to dryness in a water bath for 3 days at 50^{o}C.

Animals

Adult male Wistar rats weighing 120-150 g were bought from the Animal of the Department of Science Laboratory Technology, AkanuIbiam Federal Polytechnic Unwana, Afikpo, Ebonyi State. The rats were housed in plastic cages under standard laboratory conditions. They were allowed access to food and water *ad-libthum.* Animals were acclimatized for 14 days.

Median Lethal dose 50% (LD50)

The LD_{50} determination was conducted in two phases.In the first phase, nine (9) male rats were divided into three groups of three rats each. The groups were separately administered with 10, 100 and 1000 mg/kg of extract orally. Afterwards, animals were observed closely for 24 hr. for possible signs of toxicities. In the absence of mortality at the first phase, the second phase was initiated and three groups of one rat per group. The various groups of the second phase was separately administered with 1600, 2900 and 5000 mg/kg of extract, after which signs of toxicities were looked out for on the animals which were observed for 48 h for signs of toxicity according to Lorke [10].

Animal Grouping

Group I (Normal Control): animal were administered distilled water

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Group II (Negative Control) animals induced hepatic damage by oral administration of 600 mk/kg/d of paracetamol without treatment

Group III: Rats with hepatic damage administered 100 mg/kg of extract

Group IV: Rats with hepatic damage administered 200 mg/kg of extract

GroupV: Rats with hepatic damage administered 300 mg/kg of extract

Group VI: Rats with hepatic damage administered standard drug (silymarin)

Administration of extract lasted for 28 days after which animals were sacrificed and blood sample collected for analysis.

Liver function test

The activity of liver enzymes;aspartate aminotransferase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP) were determined by introducing 2 mL of blood into an EDTA tube and afterwards centrifuged at 4,000 rpm for 15 min. The resulting plasma was analyzed with the aid of kits

Determination of lipid profile

Cholesterol, HDL and triacyglyceride levels were estimated from serum by CHOD-PAP. LDL and HDL were calculated, while the artherogenic index was calculated using the method described by Muruganandan et al. [11].

Body weight measurement

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Determination of animal weight was performed at the commencement of the study which was considered day 0 and before termination of experiment.

Statistical Analysis

Data generated from the study were expressed as mean ± standard deviation using SPSS (Ver. 23). Data were analysed using one way analysis of variance (ANOVA). Variation in mean values was compared using Turkey Test. *P-values* less than 0.05 was considered statistically significant.

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Table 1: Liver Enzyme activity in Rats with Hepatic Damage treated with Aqueous Extract of Lemon Grass

Treatment	AST (U/I)	ALT(U/I)	ALP(U/I)
Group I (Normal CTRL)	9.12±1.20 [a]	14.00±2.67[a]	75.80±2.24[a]
Group II Negative CTRL	34.00±4.10[c]	27.01±4.10[d]	116.01±4.21[c]
Group III (100 mg/kg	11.00±3.66 [b]	21.06±3.27[b]	84.00±2.00[b]

Ext.)			
Group IV (200 mg/kg Ext.)	11.80±4.34 [b]	22.07±4.36[bc]	82.00±1.43[b]
Group IV (300 mg/kg Ext.)	11.00±5.67[b]	21.30±4.66[b]	81.02±1.65[b]
Group IV (Std. silymarin)	9.08±2.20[a]	14.04±4.26[a]	76.00±6.52[a]

Results are expressed as mean ± standard deviation of three determinations. Values with different superscripts in a column are significantly different at P<0.05.

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Table 2: Lipid Profile of Wistar Rats administered Aqueous Extract of Lemon Grass

Treatment	**TC (mg/dl)**	**TG (Mg/dl)**	**HDL (Mg/dl)**	**LDL (Mg/dl)**
Group I (Normal CTRL)	203±11.53[a]	63±10.56[a]	71±4.56[d]	127±42.56[a]
Group II Negative CTRL	222±2.06[b]	73±7.06[b]	84±5.46[c]	136±45.56[c]
Group III (100 mg/kg Ext.)	206±6.31[ab]	64±5.01[ab]	75±4.65[b]	130±2.51[d]
Group IV (200 mg/kg Ext.)	203±4.21[a]	62±2.36[a]	70±2.46[c]	130±1.16[b]

Group IV (300 mg/kg Ext.)	204±5.05[a]	63±21.56[a]	72±3.52[b]	131±5.32[b]
Group IV (Std. silymarin)	202±6.30[a]	62±1.59[a]	73±12.46[d]	128±5.16[a]

Results are expressed as mean ± standard deviation of three determinations. Values with different superscript in a column are significantly ($p<0.05$) different

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Table 3: Body weight of wistar rats administered Aqueous Extract of Lemon Grass

Treatment	Initial wt. (g)	Final wt. (g)
Group I (Normal CTRL)	125.23±8.72	157.20±6.00
Group II Negative CTRL	135.64±4.50	130.40±5.63
Group III (100 mg/kg Ext.)	167.32±5.27	175.65±2.40
Group IV (200 mg/kg Ext.)	147.23±6.19	165.30±5.60

Group IV (300 mg/kg Ext.)	175.03±6.27	187.03±5.76
Group IV (Std. silymarin)	167.30±6.39	178.21±6.50

Results are expressed as mean ± standard deviation of three determinations. Values with different superscripts in a column are significantly different at P<0.05.

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Results and Discussions

Cellular damage either orchestrated naturally or artificially is mediated mainly by free radical generation. Table 1 shows that the activity of liver enzymes in rats with damaged hepatocytes treated with aqueous extract of *Sida acuta* indicating that the activity of AST, ALT and ALP in rats induced with hepatic damage was significantly ($p<0.05$) higher than that reported for the

normal control. However, following oral administration of 100, 200, and 300 mg/kg of *Sida acuta* extract, a significant reduction in the activity of the aforementioned enzymes was observed which however was significantly ($p<0.05$) lower than that reported for the group administered standard drug which in turn was not significantly ($p>0.05$) different from that reported for the normal control. This could be attributed to the influence of flavonoids and zinc which are appreciably present in the leaf of *Sida acuta*(Nwankpa et al., [12]. This result is consistent with the finding of Nwankpa et al. [12] which reported the antioxidant properties of*Sida acuta* extract. Damage to the liver will undoubtedly impair hepatic functions including hepatic lipid metabolism which can translate to a distorted lipid profile.Table 2 shows the lipid profile of wistar rats administered aqueous extract of *Sida acuta* indicating that total cholesterol, Triacylglycerol (TG), High Density Lippoprotein(HDL), and Low Density Lippoprotein levels in group II were significantly ($p<0.05$) higher than those reported for the normal control which in turn were not significantly ($p<0.05$) different from those reported for the groups administered *Sida acuta*extract as well as the group administered the standard drug (silymarin). The potential of the said extract to restore a normal lipid profile of the animals could be attributed to the presence of flavonoids reportedly present in the leaf of the plant Nwankpa et al. [12]. This is consistent with the finding of Ogbodo et al. [13] which demonstrated the anti-hyperlipidemic effect of the leaf extract of *Sidacorymbosa* a member of the malvaceae family to which *Sida acuta* belongs.Liver damage can cause symptoms such as poor appetite which can translate to weight loss. Table 3 shows the body weight of rats administered with aqueous extract of *Sida acuta*, showing that there was a significant increase in the weight of rats over treated periods. However, a contrary observation was made on the group II which was induced with hepatic

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damage without treatment. Weight increase observed on treated rats could be attributed to the reversal of damaged liver cells to healthy ones and hence improved consumption of meal.

Conclusion

Through this research effort, it can be deduced that oral administration of *Sida acuta* reversed hepatic damage, and distorted lipid profile and did not result in weight loss.

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