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Full Length Research Paper

Total lipid profile and faecal cholesterol with aqueous fruit extract of *Solanum macrocarpum* in triton-induced hyperlipidaemic albino rats

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Studies were undertaken to investigate the effect of the aqueous fruit extract of *Solanum macrocarpum* on the total lipid profile [total cholesterol, triglycerides, high density lipoprotein-cholesterol (HDL-C) and low density lipoprotein-cholesterol (LDL-C)] and faecal cholesterol on triton-induced hyperlipidaemic rats. Total serum cholesterol, triglycerides and LDL-C were lowered in the hyperlipidaemic rats with increasing dose of the extract whilst HDL-C increased. These changes however were not significant (*P>0.05*) with the exception of LDL-C which was significant (*P<0.05*) at 24 h of the study when compared to the control. The increase in faecal cholesterol with increase in extract dose was significant (*P<0.005*) at 24 h when compared to the control. The results show that the aqueous fruit extract of the plant may be capable of reducing circulating lipids in triton-induced hyperlipidaemic rats probably by reducing absorption of lipids, thus reducing hyperlipidaemia.

Key words: *Solanum macrocarpum*, faecal cholesterol, triglycerides, total cholesterol, LDL-cholesterol, HDL-cholesterol.

INTRODUCTION

In recent years, an increasing percentage of people from industrialized countries have been using complementary and alternative medicines (CAM) (Long et al., 2007). Herbal medicine is used by up to 80% of the population in the developing countries (Jaouad et al., 2004). Extracts from the unripe fruits of *Solanum macrocarpum* (synonymns: *S. macrocarpon* L. sensu stricto and *S. daysphyllum* Schumach and Thonn) (Grubben and Denton, 2004) called "Gorongo" in Kanuri have been used by the "Kanuris" of Nigeria and other West African countries (like Sierra Leone) and East African countries' (like Kenya and Uganda) folk medicine (Grubben and Denton, 2004). In the traditional North East Arid zone of Nigeria, the unripe fruit of *S. macrocarpum* is known for its laxative, antihypertensive and hypolipidaemic effects. The phytochemistry of the plant revealed that *S. macrocarpum* fruit contains alkaloids (Sodipo et al., 2008a). The steroidal alkaloids are said to be responsible for lowering hyperlipidaemia (Anonymous, 2007; Sodipo et al., 2009c). Furthermore, saponins as found in the fruit of this plant (Sodipo et al., 2008a) are cholesterol-lowering agents (Cheeke, 1971; Sodipo et al., 2009c). Importantly, *S. macrocarpum* had been shown to display a wide spectrum of biological activities, with experimental support for the empiric ethnopharmacological use of this plant in folk medicine (Sodipo et al., 2008a, b; 2009a). Recently, we have demonstrated hepatoprotective effects of aqueous fruit extract of *S. macrocarpum* in diet-induced hypercholesterolaemic rats (Sodipo et al., 2009b). However, the mechanism of hyperlipidaemia had not bee extensively studied. Sodipo et al. (2009c) observed a favourable lipid profile in diet-induced

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Abbreviations: HDL-C, High density lipoprotein-cholesterol; **LDL-C,** low density lipoprotein-cholesterol; **CAM,** complementary and alternative medicines; **IP,** intraperitoneally; **RBC,** red blood cell; **ALT,** alanine amino transferase.

hypercholesterolaemic rats administered with the aqueous fruit extract, the exact mechanism of cholesterol lowering was not known. Taking into account these data, we have conducted our research on total lipid profile and faecal cholesterol after oral administration of triton-X 100 (a non-ionic surfactant that interferes with uptake of lipids) to induce hyperlipidaemia in rats in order to find out if the fruit of *S. macrocarpum* can indeed lower hyperlipidaemia as an increase in faecal cholesterol corresponds to a decrease in cholesterol and lipid absorption (Moore et al., 1968). Also, a plausible mechanism of the hypolipidaemic action of the plant, if there is any, could probably be fashioned out. It has been shown that intravenous injection of nonionic detergents such as triton WR-1339 (polymeric p-iso-octyl polyoxethylene phenol) in experimental animals, results in a progressive increase in the concentration of lipids in the blood (Otway and Robinson, 1967). In the experiment that would be described, triton X-100 (polyoxylethylene octyl phenyl ether) was administered orally to the rats and not parenterally like triton WR1339 because the pilot study revealed that 400 mg/kg of the triton-X administered intraperitoneally (ip) to 30 rats in the first day killed all of them, probably indicating high osmotic fragility and altered red blood cell (RBC) morphology, as to cause icterus, leading to the death of the rats. Oral administration of the triton however did not cause death in the rats (Sodipo, 2009).

MATERIALS AND METHODS

Plant collection and identification

The plant material (*S. macrocarpum* Linn.) used in this study was obtained from Alau in Konduga Local Government, Borno State, Nigeria, between October and November, 2007. The plant was identified and authenticated by Prof. S.S. Sanusi of the Department of Biological Sciences, University of Maiduguri, Maiduguri, Nigeria. Specimen voucher No. 548 was deposited at the Research Laboratory of the Department of Chemistry.

Extraction

The fruit of *S. macrocarpum* with the calyx removed was air dried and pulverized by grinding using pestle and mortar. The 2.2 kg of the ground fruit was subjected to exhaustive Soxhlet-extraction in distilled water at 100°C to give the extract yield of 15.3% $^{w}/_{w}$ (Mittal et al., 1981; Fernando et al., 1991; Lin et al., 1999). The resultant solution was concentrated *in vacuo* and it was stored in a specimen bottle at room temperature until when required.

Animals

Thirty male albino rats of Wistar strain weighing 160 to 200 g were used in this study. The animals were obtained from the Animal House Unit of the Department of Veterinary Physiology and Pharmacology, University of Maiduguri.

The animals were housed under standard laboratory condition in plastic cages. They were fed commercial growers' mash feed (ECWA Feeds, Jos, Nigeria) and water was provided *ad libitum*. All the animals were handled according to the International Guiding Principles for Biomedical Research Involving Animals (CIOMS, 1985) as certified by the Animal Ethics Committee of the Faculty of Veterinary Medicine, University of Maiduguri Nigeria.

Administration of triton and extract

Thirty albino rats were made hyperlipidaemic by feeding them orally (po) for 1 week with normal feed diet and triton-X (Sigma Chemical Co. St. Louis, M.O. USA) at a dose of 400 mg/kg in saline suspension from the stock concentration of 535 g/ml. The rats were divided into 5 groups of 6 animals each. After seven (7) days, the rats were administered with graded doses of the fruit extract. Group I was the control and it was given distilled water only. Groups II, III, IV and V were administered with geometrical doses (25, 50, 100 and 200 mg/kg) of the fruit extract IP from a stock concentration of 200 mg/ml. IP route was used because LD_{50} (IP LD_{50} = 1,280 mg/kg) could only be determined through this route. When the extract was administered through oral route no mortality was recorded. After 24, 48, and 72 h, respectively of the effect of the extract on the hyperlipidaemic rats, the total lipid profile and faecal cholesterol were determined (Adapted from Williamson et al., 1996).

Before the rats were fed with triton-X, their weight was taken. The weights were subsequently taken after 1 week of triton administration.

Determination of total lipid profile

Two rats from each of the groups were humanely sacrificed after 24, 48 and 72 h, respectively of the effect of the extract on hyperlipidaemic rats by cutting their throat with a sterile blade. Blood was collected into clean, sterile, labelled centrifuge tubes without an anticoagulant and centrifuged at a rate of 12,000 revolutions per minute (rpm) for 10 min. The clear, yellow serum was then separated from settled cellular elements and subjected to determination of total lipid profile. The total lipid profile estimated from the serum was total cholesterol, triglycerides, HDL-C and LDL-C. Total cholesterol was assayed by Tindar's reaction (Evans and Stein, 1986; NIH, 1990) using commercial kits from Fortress Diagnostics Limited; Antrim. Serum triglycerides level was determined as described by Fossati and Prencipe (1982), Cole et al. (1997) using commercial kit (Human cholesterol using Liquicolour Test Kit, Germany) (Grove, 1970). The serum LDC-L level was calculated by Friedwald's formular (Friedwald et al., 1972; Hardman and Limbird, 2001; Sood, 2006). The calculation for serum LDL-C is given by:

Serum LDL-C = Total cholesterol – Trigylcerides ÷ 2.2 – HDL – C.

Determination of faecal cholesterol

Faeces from two rats in each of the groups were collected after 24, 48 and 72 h, respectively that the extract had acted on the hyperlipidaemic rats into clean, cellophane bags and deep frozen. They were homogenized and extracted with chloroform/methanol in the ratio 2:1 (Folch et al., 1957) for analysis of faecal cholesterol. The faecal cholesterol determination was like that of the serum total cholesterol (Tindar's reaction) (Evans and Stein, 1986; NIH, 1990) using commercial kits from Fortress Diagnostic Limited; Antrim.

Statistical analysis

Data were expressed as the mean ± SD and student t-test. The

Table 1. Change in body weight of male albino rats after being administered triton-X (400 mg/kg) orally for 7 days.

Group	Mean body weight ± S.D. (g)	
	Days of treatment	
	0	7
One	114.33 ± 11.76[a]	129.00 ± 9.22[b]
Two	98.83 ± 11.55[a]	111.83 ± 11.18[a]
Three	140.83 ± 37.57[a]	151.67 ± 36.82[a]
Four	137.33 ± 30.89[a]	147.00 ± 30.44[a]
Five	175.33 ± 31.10[a]	194.83 ± 37.12[a]

Within rows, means with the same superscript are not statistically significant ($p > 0.05$) when compared with day 0 using student t-test. 0 day = before triton-X administration, 7 days = after oral administration of triton.

results obtained were subjected to Analysis of Variance (ANOVA) using Graph Pad Software (1998).

RESULTS

Effect of extract on body weight of rats

The effect of triton-X on mean body weight of albino rats is shown in Table 1. The increase in body weight observed in the rats was not statistically significant (*P>0.05*) when compared with day zero in all the groups except in group 1 where the increase was significant (*P<0.05*).

Effect of extract on total lipid profile of hyperlipidaemic rats administered triton-X orally for 7 days

The decrease in triglycerides, total cholesterol and increase in HDL-C were not significant (*P>0.05*) throughout the period of study with the exception of the decrease in LDL-C which was significant (*P<0.05*) at 24 h as shown in Table 2.

Effect of extract on faecal cholesterol of hyperlipidaemic rats administered triton-X orally for 7 days

The increase in faecal cholesterol with increase in extract dose was only significant at 48 h of study (*P<0.05*) as shown in Table 3.

DISCUSSION

The increase in mean body weight of the rats after triton-X administration on the rats in groups two-five, orally for 7 days was not significant (*P>0.05*) except for group one where the weight increase was significant (*P<0.05*). This probably implies that triton-X at the dosage employed, 400 mg/kg or for the length of time given, had a slight effect of inducing hyperlipidaemia in rats. The decrease in total cholesterol, triglycerides and increase in HDL-C were not significant (*P>0.05*) throughout the period of study with the exception in the decrease in LDL-C which was significant (*P<0.05*) at 24 h. Triglycerides are true fats, esters of glycerol and fatty acids that belong to the organic group of compounds called lipids. Upon hydrolysis, they yield glycol and fatty acids. Low levels of triglycerides imply that there is no risk factor related to atheroschlerotic disease that causes thickening of the walls of the larger arteries which may lead to heart attack. Also, an increase in serum triglycerides leads to hyperlipidaemia (Mukherjee, 1988). In the present study, though the decrease recorded in trigylycerides was not statistically significant, it may still probably be said that the aqueous fruit extract of *S. macrocarpum* lowers hyperlipidaemia. Cholesterol is the main lipid found in blood, bile and brain tissues (Sood, 2006). Normal serum cholesterol varies with age, diet and from country to country (Odutola, 1992). Cholesterol is classified as a sterol because it is required for formation of steroids and cellular membranes (Sood, 2006). Increased levels of cholesterol lead to coronary artery disease, hyperproteinaemia, diabetes, cirrhosis, haemolytic jaundice, acute infection, malnutrition and hyperthyroidism (Mukherjee, 1988; Odutola, 1992). Increased levels of oestrogens can also lead to hypercholesterolemia (Odutola, 1992).

In this present study, the decrease in serum total cholesterol on extract administration at all hours of study, (24, 48 and 72 h), though not significant, is in agreement with the hypocholesterolaemia recorded with the fruits of *S. melongena* L., *Solanum gilo* Raddi and *S. macrocarpum* Linn. In New Zealand rabbits and Wistar strain albino rats, respectively which were fed with cholesterol-rich diet (Odetola et al., 2004; Sodipo et al., 2009c). The aqueous fruit extract of *S. macrocarpum* could probably then be said to have a cholesterol-lowering effect on the triton-induced hyperlipidaemic rats. At present though, plant products are more important in the dietary aspect of controlling hyperlipidaemia rather than as drug treatment (Williamson et al., 1996). Dietary cholesterol has little influence on serum total cholesterol in most people. Nonetheless, high levels of cholesterol in serum may be as a result of high dietary intake known as exogenous hyperlipidaemia (Odutola, 1992). In the present study, the decrease in LDL-C was significant (*P<0.05*) at 24 h of study. LDL-Cs is derived from the metabolism of VLDL-C and they have a very low half life ($t^1/_2$), 3 to 4 days (Hardman and Limbird, 2001). The results buttressed the fact that the aqueous fruit extract of *S. macrocarpum* could probably lower the hyperlipidaemia induced in the rats. Flavonoids present in the fruit (Sodipo et al., 2008a) prevent the oxidation of the LDL-C which is atherogenic (Chander et al., 2005; Khan, 2008). Also the phytochemistry of the plant revealed that

Table 2. Effect of the aqueous fruit extract of *S. macrocarpum* on total lipid profile of hyperlipidaemic rats administered orally with Triton-X for 7 days.

Hours after extract administration	Extract dose (mg/kg)	Total cholesterol (mmol/L)	Triglycerides (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)
		Mean ± S.D.			
24	Control (distilled water)	2.50 ± 0.00[a]	1.35 ± 0.21[a]	1.05 ± 0.07[a]	1.00 ± 0.14[a]
	25.00	2.10 ± 0.14[a]	1.20 ± 0.00[a]	1.10 ± 0.14[a]	0.45 ± 0.07[b]
	50.00	2.10 ± 0.42[a]	1.15 ± 0.07[a]	1.10 ± 0.14[a]	0.15 ± 0.07[b]
	100.00	1.85 ± 0.21[a]	1.15 ± 0.07[a]	1.10 ± 0.14[a]	0.10 ± 0.00[b]
	200.00	1.70 ± 0.00[a]	1.00 ± 0.00[a]	1.15 ± 0.07[a]	0.10 ± 0.00[b]
48	Control (distilled water)	2.40 ± 0.28[a]	1.25 ± 0.21[a]	0.85 ± 0.21[a]	1.20 ± 0.14[a]
	25.00	2.35 ± 0.21[a]	1.10 ± 0.00[a]	0.85 ± 0.07[a]	0.85 ± 0.35[a]
	50.00	2.25 ± 0.07[a]	1.10 ± 0.14[a]	0.95 ± 0.07[a]	0.85 ± 0.07[a]
	100.00	2.25 ± 0.21[a]	1.05 ± 0.07[a]	1.05 ± 0.07[a]	0.75 ± 0.50[a]
	200.00	2.05 ± 0.07[a]	0.80 ± 0.14[a]	1.05 ± 0.17[a]	0.75 ± 0.07[a]
72	Control (distilled water)	2.35 ± 0.07[a]	1.35 ± 0.07[a]	0.80 ± 0.28[a]	1.10 ± 0.14[a]
	25.00	2.25 ± 0.07[a]	1.10 ± 0.14[a]	0.85 ± 0.07[a]	0.95 ± 0.07[a]
	50.00	2.25 ± 0.07[a]	1.10 ± 0.28[a]	1.00 ± 0.14[a]	0.75 ± 0.36[a]
	100.00	2.15 ± 0.07[a]	1.00 ± 0.28[a]	1.05 ± 0.07[a]	0.70 ± 0.00[a]
	200.00	2.10 ± 0.14[a]	0.80 ± 0.20[a]	1.15 ± 0.07[a]	0.65 ± 0.07[a]

Means with different superscripts are statistically significant ($p < 0.05$) among the groups.

S. macrocarpum fruit contains alkaloids (Sodipo et al., 2008a). Reports have shown that the Solanacea contain steroidal alkaloids (Olaniyi et al., 1998).

The steroidal alkaloids are said to be responsible for lowering hyperlipidaemia (Anonymous, 2007; Sodipo et al., 2009c). Furthermore, saponins as found in the fruit of this plant (Sodipo et al., 2008a) are cholesterol-lowering agents (Cheeke, 1971; Sodipo et al., 2009c). The result in this study of significant decrease in LDL-C, tallies with that of Chander et al. (2005) who showed that administration of Indian black tea to triton WR 1339 (polymeric p-iso-octyl polyoxyethylene phenol)-induced hyperlipidaemic rats caused a decrease in the plasma LDL-C. In the present study, there was an increase in HDL-C throughout the period of study, though the increase was not significant. It has been estimated that for any 0.026 mmol/ml (1 m/dl) increase in HDL-C there is a 3% decrease in the risk of mortality from cardiovascular disease (Okonofua et al., 1990; Igweh et al., 2005). The increase in faecal cholesterol with increase in extract dose was significant at 48 h ($P<0.05$) when compared to the control in the hyperlipidaemic rats. This implies that with increase in extract dose, cholesterol excretion in the faeces increased. The lowered LDL-C previously observed could be attributed to the increase in total faecal cholesterol indicating that the extract could have probably altered lipid absorption. This agrees with the work of Moore et al. (1968) who demonstrated that the hypocholesterolaemic action of dietary unsaturated fatty acids in man is associated with an increase in the faecal loss of bile acids and neutral sterols. Saponins as found in this plant (Sodipo et al., 2008a) are known to increase bile acid and other sterol production (Mahato and Gongulu, 1982; MacDonald, et al., 2005) which is subsequently excreted.

This is in conformity with the hypothesis that increased faecal excretion of cholesterol corresponds to a decreased absorption (Moore et al., 1968). Literature has shown that green tea leaves (*Camellia sinensis*) lowered cholesterol and inhibited lipid absorption and also decreased serum alanine amino transferase (ALT) activity in

Table 3. Effect of the aqueous fruit extract of *S. macrocarpum* on faecal cholesterol of hyperlipidaemic rats administered orally with triton-X for 7 days.

Hours after extract administration	Extract dose (mg/kg)	Faecal cholesterol (mmol/L) Mean ± S.D.
24	Control (distilled water)	0.20 ± 0.28[a]
	25.00	0.25 ± 0.07[a]
	50.00	0.30 ± 0.00[a]
	100.00	0.35 ± 0.07[a]
	200.00	0.36 ± 0.07[a]
48	Control (distilled water)	0.25 ± 0.00[a]
	25.00	0.30 ± 0.00[b]
	50.00	0.35 ± 0.07[b]
	100.00	0.35 ± 0.07[b]
	200.00	0.43 ± 0.04[b]
72	Control (distilled water)	0.20 ± 0.07[a]
	25.00	0.25 ± 0.25[a]
	50.00	0.40 ± 0.50[a]
	100.00	0.40 ± 0.00[a]
	200.00	0.43 ± 0.04[a]

Means with different superscripts are statistically significant ($p < 0.05$) among the groups.

ovariectomised rats and obese mice, respectively (Wang et al., 2006; Bruno et al., 2008). It there-fore follows that since the aqueous fruit extract of *S. macrocarpum* increased faecal cholesterol excretion in hyperlipidaemic rats, the absorption probably decreased and this might explain the hypolipidaemic and hypocholesterolaemic effects of the extract as claimed in traditional medicine. Also the increase in faecal cholesterol excretion might be due to its decreased absorption which may be due to the inhibition of pancreatic lipolytic enzymes such as lipase and phospholipase A_2 as demonstrated in green tea leaves (Bruno et al., 2008). Stimulation of fatty acid oxidation ingreen tea leaves (Bruno et al., 2008), leading to an increase in *β*-oxidation in mice fed high-fat diet, may also be a plausible mechanism in non-accumulation of cholesterol and hence increased excretion of fat in the faeces in hyperlipidaemic rats administered aqueous fruit extract of *S. macrocarpum*. The increase in faecal excretion of cholesterol was only significant at 48hrs of study, probably implying that the maximal hypolipidaemic effect was felt at 48hrs of extract administration.

Conclusion

The present study shows that the aqueous fruit extract of *S. macrocarpum* may be capable of reducing lipids in triton-induced hyperlipidaemic rats probably by reducing absorption of lipids and increasing faecal cholesterol excretion, thus reducing hyperlipidaemia, thus buttressing the claim in traditional medicine that the unripe fruit lowers hyperlipidaemia.

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