Effect of Aqueous Extract of *Sansevieria senegambica* Baker on Plasma Chemistry, Lipid Profile and Atherogenic Indices of Alloxan Treated Rats: Implications for the Management of Cardiovascular Complications in Diabetes Mellitus.

Catherine Chidinma Ikewuchi, M.Sc.

Department of Biochemistry, Faculty of Science, University of Port Harcourt, PMB 5343, Choba, Rivers State, Nigeria.

E-mail: okaraonye@yahoo.com

ABSTRACT

Effect of aqueous extract of *Sansevieria senegambica* on plasma marker enzymes, plasma chemistry, glucose and lipid profiles, and atherogenic indices were studied in alloxan treated rats. Diabetes mellitus was induced by injection of alloxan (140mg/kg body weight), via the tail vein. The extract was administered orally at 150 and 200mg/kg, and metformin at 50mg/kg. Compared to test control, the 150mg/kg treatment lowered (significantly, $p<0.05$) plasma glucose (on day 2), LDL cholesterol and conjugated bilirubin levels, and not significantly, alanine transaminase activity, cardiac risk ratio, atherogenic index of plasma and coefficient, but significantly ($p<0.05$) increased plasma HDL cholesterol and calcium levels; without affecting plasma chemistry and other marker enzymes. These results indicate the extract's dose dependent hypoglycemic and cardioprotective (against dyslipidemic conditions) potential, as well as protection against hypocalcaemia.

(Keywords: *Sansevieria senegambica, alloxan treatment, atherogenic indices, lipid profile, plasma chemistry, plasma glucose, plasma marker enzymes*)

INTRODUCTION

The plant *Sansevieria senegambica* belongs to the genus Sansevieria, whose common names include mother-in-laws tongue, devils tongue, and snake plant. This genus consists of about sixty (60) species of flowering plants in the family Agavaceae (or Ruscaceae), native to tropical and subtropical regions of the world [Evans, 2005]. *Sansevieria senegambica* has short upright curved leaves flattening toward the tip end with a slim point, and a surface that is a matte-green with faint banding. This good compact waxy leaved species has reddish rhizomes and violet-white floral [http://www.glasshouseworks.com/succ-s.html]. It is grown as an ornamental plant [USDA, 2008]. In southern Nigeria, it is used in traditional medical practice, for curing bronchitis, inflammation, cough and boils, and managing diabetes mellitus and hypertension. It is also used in arresting snake bites, as well as in compounding solutions used as hair tonic. The present study was designed to investigate the effect of aqueous extract of *Sansevieria senegambica* on plasma marker enzymes, plasma chemistry, glucose and lipid profiles, and atherogenic indices of alloxan treated rats: with a view to clarifying the basis of its use in the management of diabetes mellitus.

MATERIAL AND METHODS

Collection of Animals and Preparation of Plant Extract

Albino rats were collected from the animal house of the Department of Physiology, University of Nigeria, Enugu Campus, Enugu, Nigeria. Samples of fresh *Sansevieria senegambica* plants were procured from: a horticulturist by Air Force Gate, Aba Road; Alikor Estate Choba; a horticultural garden by Uniport main gate, Abuja campus; behind Ofrima complex, Abuja campus, Uniport, all in Port Harcourt, Nigeria. After due identification at the University of Port Harcourt Herbarium, Port Harcourt, Nigeria, they were cleaned and the leaves were removed, oven dried at 55^0C and ground into powder. The resultant powder was soaked in boiled distilled water for

12h, after which the resultant mixture was filtered and the filtrate, hereinafter referred to as the aqueous extract was stored for subsequent use. A known volume of this extract was evaporated to dryness, and the weight of the residue used to determine the concentration of the filtrate, which was in turn used to determine the dose of administration of the extract to the test animals.

Experimental Design

The rats were randomly sorted into five groups of five animals each, so that the average weights were approximately equal. The animals were housed in plastic cages. After a one-week acclimatization period on guinea growers mash (Port Harcourt Flour Mills, Port Harcourt, Nigeria), the animals were fasted overnight and diabetes was induced by injection of a freshly prepared solution of alloxan (140mg/kg body weight) in normal saline, via the tail vein of four groups, while the control rats were injected with normal saline alone.

Seven days after administration of the alloxan, the animals were again fasted and blood collected via tail cutting [Burcelin *et al*., 1995], for the determination of their fasting glucose levels. It was found that the rats had moderate diabetes, having hyperglycemia (that is, with blood glucose of over 180% of the control). Then the rats were kept for 3 days to stabilize the diabetic condition [Jyoty *et al*., 2002] before commencing the treatment, which lasted for two days.

The first treatment group (Reference) received daily by intra-gastric gavages, 50mg/kg body weight of Diabetmin^{TM} (metformin HCl); the second group (SST15) received daily by intra-gastric gavages 150mg/kg body weight of the *Sansevieria senegambica* extract; the third group (SST20) received 200mg/kg body weight of the extract; while the fourth group (test control) and the control group received appropriate volumes of water by the same route.

The animals were allowed food and water *ad libitum*. The fasting glucose levels were taken each day, before and 3hr after administration. The animals were allowed normal feed and water *ad libitum*. At the end of the treatment period, the rats were weighed, fasted overnight and anaesthetized by exposure to chloroform. While under anesthesia, they were painlessly sacrificed and blood was collected from each rat into heparin sample bottles. Whole blood was immediately used to determine the triglyceride levels (using test strips), while the heparin anti-coagulated blood samples were centrifuged at 1000g for 10min, after which their plasma was collected and stored for subsequent analysis.

Determination of the Plasma Glucose Concentrations

The plasma glucose concentration was determined using the multiCarein^{TM} glucose strips and glucometer. The glucose contained in the sample reacts with the glucose oxidase enzyme in the glucose electrode strips to produce an electric current. The magnitude of the current produced by the electrodes is directly proportional to the glucose concentration.

Determination of the Plasma Lipid Profiles/Indices

Plasma triglyceride concentration (TG) was determined using multiCarein^{TM} triglyceride strips and glucometer (Biochemical Systems International, Arezzo, Italy). The test is based on lipase/glycerol kinase/glycerol phosphate oxidase/peroxidase/chromogen reaction. The intensity of the colour developed from the reaction is proportional to the concentration of triglycerides in the blood. Plasma total and high density lipoprotein cholesterol concentrations (TC and HDLC) were assayed enzymatically with Randox commercial test kits (Randox Laboratories, Crumlin, England).

In the presence of magnesium ions, low density lipoproteins (LDL and VLDL) and chylomicrons fractions are precipitated quantitatively by the addition of phosphotungstic acid. After centrifugation, the cholesterol concentration of the high density lipoprotein (HDL) fraction, which remains in the supernatant, can be determined, as in total cholesterol. The cholesterol released by enzymatic hydrolysis is oxidized with the concomitant release of hydrogen peroxide, whose breakdown leads to the conversion of 4-aminoantipyridine to quinoneimine (the indicator) whose concentration can be determined spectrophotometrically at 500nm.

Plasma VLDL- and LDL-cholesterol (LDLC and VLDLC) concentrations was calculated using the

Friedewald equation [Friedewald *et al*., 1972] as follows:

- LDLC (mmol/L) = TC – HDLC – $\frac{TG}{2.2}$

- VLDLC (mmol/L) = $\frac{TG}{2.2}$

While the plasma non-HDL cholesterol (non-HDLC) concentrations were determined as reported by Brunzell *et al*. [2008]:

- Non-HDLC = TC – HDLC

The atherogenic indices were calculated as earlier reported by Ikewuchi and Ikewuchi [2009], using the following formulae:

- Cardiac Risk Ratio = $\frac{TC}{HDLC}$

- Atherogenic Coefficient = $\frac{TC - HDLC}{HDLC}$

- Atherogenic Index of Plasma = $\log\frac{TG}{HDLC}$

Enzyme Assays

The plasma activities of alanine transaminase (ALT), aspartate transaminase (AST), and alkaline phosphatase (ALP), were determined using Randox Test kits (Randox Laboratories, Crumlin, England). ALT was measured by monitoring at 546nm, the concentration of pyruvate hydrazone formed with 2,4-dinitrophenylhydrazine. AST was measured by monitoring at 546nm, the concentration of oxaloacetate hydrazone formed with 2,4-dinitrophenylhydrazine. The activity of alkaline phosphatase (ALP) was determined by monitoring the degradation of p-Nitrophenylphosphate to p-nitrophenol, at 405nm.

Determination of Plasma Chemistry

Plasma total and conjugated bilirubin, urea and creatinine concentrations were determined using Randox test kits (Randox Laboratories, Crumlin, England). Direct (conjugated) bilirubin reacts with diazotized sulphanilic acid in alkaline medium to form a blue coloured complex, whose intensity can be monitored at 546nm. Total bilirubin is determined in the presence of caffeine, which releases albumin bound bilirubin, by the reaction with diazotized sulphanilic acid, with intensity of the resultant solution monitored at 578nm. Urease hydrolyzes urea to ammonia, whose concentration can be measured photometrically at 546nm, by Berthelot's reaction. In the presence of a strong alkali, creatinine reacts with picric acid to form picramic acid which imparts a yellow-red color on the solution, whose intensity can be monitored at 492nm. The amount of the complex formed is directly proportional to the creatinine concentration.

Plasma total protein was determined by the Biuret method, while plasma albumin was determined using bromcresol green (BCG) dye binding method [Holme and Peck, 1998]. Bromcresol green, a yellow dye, binds selectively to albumin at pH 4.2, to form an intense blue protein-dye complex with a maximum absorbance at 630nm. Alkaline copper solutions react with peptide bonds in protein to produce a violet color whose intensity at 560nm, is directly proportional to the amount of protein present.

Determination of Plasma Electrolytes

Plasma sodium and potassium concentrations were determined by flame photometry. When elements or their compounds are heated at high temperatures, they gain energy and become excited, and so, when they fall back to their ground or original state, produces an emission spectrum which is characteristic of the element. The intensity of the emission is within certain limits, proportional to the concentration of the element in the solution. Plasma calcium concentration was determination by the cresol phthalein complexone method (Baginski *et al*., 1973). Color was developed with cresol phthalein complex at pH 12. The magnesium interference was eliminated by complexing with 8-hydroxyquinoline, after measuring the optical density a 575nm. An excess of ethyleneglycol (diamine) tetra acetic acid (EDTA) was added for washing the calcium and the optical density was measured again. The difference is proportional to calcium level.

Plasma chloride concentration was determined by the titrimetric method (Cheessbrough, 2004).

Mercuric nitrate was titrated against chloride to form mercuric chloride in the presence of an indicator diphenyl carbazone. Light violet color was observed when the entire chloride ion in the sample was used up and excess mercuric nitrate produces a violet color. The end point of reaction is proportional to chloride concentration. Plasma bicarbonate concentration was determination by the titrimetric method (Cheessbrough, 2004). HCl reacts with bicarbonate and liberates carbon (IV) oxide leaving excess unreacted HCl in solution. NaOH was titrated against the HCl in the presence of phenol red indicator, to a neutralization point of orange color. The amount of bicarbonate is inversely proportional to unreacted HCl.

Statistical Analysis of Data: All values are quoted as the mean ± SD. The values of the various parameters for the test and control groups were analyzed for statistical significant differences using the student's t-test, with the help of SPSS Statistics 17.0 package. P<0.05 was assumed to be significant.

RESULTS AND DISCUSSION

The effect of aqueous extract of *S. senegambica* on plasma glucose levels of alloxan treated rats is given in Table 1. Before administration on day 1, the plasma glucose levels of the test groups were not significantly (p<0.05) different from the control, test control and reference treatment. After treatment, the plasma glucose levels of the test groups were significantly higher than the control, but no different from the test control and reference treatment group. There were no significant differences in the plasma glucose levels of all the groups, before administration on day 2. The plasma glucose level of SST15 (after administration on day 2) was significantly lower than test control and SST20, but not different from that of the reference group. The percentage decrease in plasma glucose levels of the test groups were higher (though not significantly) than the test control and reference treatment on day 1. On day 2, the percentage decrease in plasma glucose level of SST15 was significantly (p<0.05) higher than control, test control and reference treatment.

Table 2 shows the effect of aqueous extract of *S. senegambica* on plasma lipid profiles of alloxan treated rats. There were no significant differences in the plasma total cholesterol levels of the animals. The plasma triglyceride and VLDL cholesterol levels of the test groups were not significantly different from the control, test control and reference groups. The plasma LDL cholesterol level of SST15 was significantly (p<0.05) lower than the test control and SST20, but not different from control and reference group. The plasma HDL cholesterol level of SST15 was significantly (p<0.05) the highest, while that of test control was significantly the least. The plasma non HDL cholesterol levels of the test groups were not significantly different from the other three groups.

Table 1: Time Course of the Effect of Aqueous Extract of *Sansevieria senegambica* on Plasma Glucose Levels of Alloxan Treated Rats.

Time/ Parameter	Control	Test control	Magnitude Reference	SST15	SST20
Day 1					
• B.A. (mg/dL)	58.30±7.01[a]	95.38±51.84[a,b]	70.50±5.60[b]	106.63±42.80[a,b]	171.88±124.36[a,b]
• A.A. (mg/dL)	42.80±5.12[a]	61.75±2.86[b]	57.25±5.07[b]	72.75±11.69[b]	59.75±8.23[b]
• %Decrease	25.98±9.95[a]	21.38±26.27[a]	18.13±10.95[a]	24.36±19.95[a]	46.81±27.16[a]
Day 2					
• B.A. (mg/dL)	91.00±6.52[a]	181.25±86.09[a]	99.25±22.19[a]	122.25±33.01[a]	184.50±103.319[a]
• A.A. (mg/dL)	98.60±13.09[a,c]	138.50±38.82[c]	110.75±18.38[a,b,c]	80.00±16.69[a]	171.00±57.43[b,c]
• %change	-8.25±10.36[a]	16.31±16.67[c]	-15.49±27.90[a,c]	33.22±8.19[b]	17.65±55.00[a,b]

Values are mean ± SD, n=5, per group. B.A. = before administration. A.A. = after administration. Values in the same row with the different superscripts are significantly different at p<0.05: ‡ p<0.05 compared to B.A. value on day 1. % decrease = percentage change from B.A. value for the day.

Table 2: Effect of Aqueous Extract of *Sansevieria senegambica* on Plasma Lipid Profile of Alloxan Treated Rats.

Lipid	Plasma concentration (mmol/L)				
	Control	Test control	Reference	SST15	SST20
Triglyceride	1.28±0.49^{a,b}	0.77±0.07^{a}	0.92±0.18^{b}	1.27±0.58^{a,b}	1.09±0.19^{b}
Total cholesterol	11.19±1.45^{a}	11.79±1.74^{a}	11.56±1.18^{a}	11.64±2.25^{a}	11.31±1.86^{a}
HDL cholesterol	5.12±0.99^{a,c}	3.87±0.28^{a}	5.04±1.16^{c}	7.40±1.69^{b}	3.84±1.11^{a,c}
VLDL cholesterol	0.58±0.22^{a,b}	0.35±0.03^{a}	0.42±0.08^{b}	0.58±0.26^{a,b}	0.50±0.09^{b}
LDL cholesterol	5.48±2.01^{a,b}	7.57±1.54^{a,b}	6.61±1.96^{a,b}	3.66±2.03^{a}	6.97±1.17^{b}
Non HDL cholesterol	6.06±1.84^{a}	7.92±1.54^{b}	7.03±1.88^{a,b}	4.24±2.10^{a,b}	7.47±1.18^{a,b}

Values are mean ± SD, n=5, per group. Values in the same row with the different superscripts are significantly different at p<0.05.

The significantly lower plasma LDL cholesterol levels observed in the animals administered 15mg/100g body weight of the extract, suggests ability to reduce cardiovascular risk. Decreases in plasma LDL cholesterol have been considered to reduce risk of coronary heart disease [Rang *et al*., 2005; Shen, 2007]; while high plasma levels of LDL cholesterol is a risk factor for cardiovascular disease [Ademuyiwa *et al*., 2005; Lichtennstein *et al*., 2006] and often accompanies diabetes mellitus [Rang *et al*., 2005; Brunzell *et al*., 2008; Shen, 2007], obesity [Krauss *et al*., 2006] and hypertension [Shepherd, 1998; Zicha *et al*., 1999].

In this study, the increased plasma HDL cholesterol levels produced by the administration of the extract at 15mg/100g body weight, portends reduction of cardiovascular risk. According to clinical data, increases in plasma HDL cholesterol concentration decreases cardiovascular risk [Rang *et al*., 2005; Assmann and Gotto, 2004]. High HDL exerts a protective effect by decreasing the rate of entry of cholesterol into the cell via LDL and increasing the rate of cholesterol release from the cell [Marcel *et al*., 1980]; and inhibiting the oxidation of LDL as well as the atherogenic effects of oxidized LDL by virtue of its antioxidant [Brunzell *et al*., 2008; Assmann and Gotto, 2004; Ademuyiwa *et al*., 2005] and anti-inflammatory property [Ademuyiwa *et al*., 2005].

Conversely, decreased plasma HDL cholesterol is a risk factor for cardiovascular diseases [Rang *et al*., 2005; Martirosyan *et al*., 2007; Lichtennstein *et al*., 2006; Lewis and Rader, 2005] and is often found in hypertension [Shepherd, 1998; Zicha *et al*., 1999], obesity [Krauss *et al*., 2006] and diabetes mellitus [Rang *et al*., 2005; Brunzell *et al*., 2008; Shepherd, 1998; Shen, 2007].

The effect of aqueous extract of *S. senegambica* leaves is shown in Table 3. The cardiac risk ratio, atherogenic coefficient and atherogenic index of plasma of SST15 were lower, though not significantly, than the other groups. Low atherogenic indices are protective against coronary heart disease [Usoro *et al*., 2006]. Atherogenic indices are powerful indicators of the risk of heart disease: the higher the value, the higher the risk of developing cardiovascular disease and vice versa [Martirosyan *et al*., 2007; Brehm *et al*., 2004; Dobiásová, 2004; Usoro *et al*., 2006].

Effects of aqueous extract of *Sansevieria senegambica* on plasma marker enzymes of alloxan treated rats is shown in Table 4. There were no significant differences in the plasma aspartate transaminase activities of all the groups. The plasma alanine transaminase activity of SST15 was significantly (p<0.05) higher than control and lower than test control and SST20, but not different from reference. The plasma alkaline phosphatase activity of SST20 was significantly (p<0.05) higher than reference, but not different from control, test control and SST15. These results indicate that the extract has no effect on the integrity and function of the liver of the diabetic.

Table 5 shows the effect of aqueous extract of *Sansevieria senegambica* on plasma chemistry of alloxan treated rats. The plasma creatinine and urea levels of SST15 were significantly (p<0.05) lower than reference group, but not different from control, test control and SST20. The plasma total bilirubin concentration of SST15 was significantly (p<0.05) lower than control, but not different from test control, reference treatment and SST20.

Table 3: Effect of Aqueous Extract of *Sansevieria senegambica* on Atherogenic Indices of Alloxan Treated Rats.

Index	Magnitude				
	Control	Test control	Reference	SST15	SST20
Cardiac risk ratio	2.26±0.58^{a,c}	3.04±0.32^{b,d}	2.39±0.46^{a,b}	1.65±0.45^{b}	3.07±0.48^{c,d}
Atherogenic coefficient	1.26±0.58^{a,c}	2.04±0.32^{b,d}	1.39±0.46^{a,b}	0.65±0.45^{b}	2.07±0.48^{c,d}
Atherogenic index of plasma	-0.62±0.13^{a}	-0.70±0.04^{a,b}	-0.74±0.09^{a,b}	-0.80±0.12^{b}	-0.53±0.15^{a}

Values are mean ± SD, n=5, per group. Values in the same row with the different superscripts are significantly different at p<0.05.

Table 4: Effect of Aqueous Extract of *Sansevieria senegambica* on Plasma Marker Enzymes of Alloxan Treated Rats.

Enzyme	***Activity (U/L)***				
	Control	***Test control***	***Reference***	***SST15***	***SST20***
Aspartate transaminase	17.96±14.19^{a}	26.90±15.48^{a}	17.88±5.87^{a}	21.66±10.74^{a}	26.26±25.47^{a}
Alanine transaminase	8.34±2.62^{a}	29.28±5.51^{b}	25.18±16.85^{a,b,c}	14.03±4.94^{c}	25.98±8.99^{b}
Alkaline phosphatase	17.11±9.19^{a,b}	31.05±22.62^{a,b}	23.46±14.64^{a}	52.79±37.87^{a,b}	44.02±24.34^{b}

Values are mean ± SD, n=5, per group. Values in the same row with the different superscripts are significantly different at p<0.05.

Table 5: Effect of Aqueous Extract of *Sansevieria senegambica* on Plasma Chemistry of Alloxan Treated Rats.

Parameter	***Magnitude***				
	Control	***Test control***	***Reference***	***SST15***	***SST20***
Creatinine (mg/dL)	0.63±0.27^{a,b}	0.85±0.57^{a,b}	0.65±0.13^{a}	0.49±0.05^{b}	0.54±0.34^{a,b}
Urea (mg/dL)	20.40±8.23^{a}	34.80±9.15^{a,b}	46.34±12.12^{b}	23.18±1.46^{a,c}	58.57±23.96^{b,c}
Total bilirubin (mg/dL)	1.08±0.47^{a}	0.82±0.38^{a,b}	0.37±0.25^{b}	0.34±0.11^{b,c}	0.56±0.24^{a,c}
Conjugated bilirubin (mg/dL)	0.73±0.32^{a}	0.52±0.24^{a,c}	0.22±0.19^{c,d}	0.27±0.11^{b,d}	0.43±0.17^{a,b}
Unconjugated bilirubin (mg/dL)	0.35±0.18^{a}	0.31±0.27^{a,b}	0.16±0.10^{a,b}	0.07±0.03^{b}	0.12±0.07^{a,b}
Unconjugated/conjugated bilirubin ratio	0.470±0.155^{a}	0.644±0.502^{a}	0.716±0.430^{a}	0.318±0.304^{a}	0.272±0.068^{a}
Total protein (mg/dL)	6.12±0.08^{a}	6.00±0.30^{a}	6.00±0.23^{a}	6.13±0.23^{a}	6.18±0.15^{a}
Albumin (mg/dL)	3.70±0.32^{a}	3.95±0.27^{a}	4.00±0.16^{a}	3.95±0.22^{a}	3.93±0.15^{a}
Bicarbonate (mmol/L)	20.75±0.83^{a}	26.00±1.22^{b}	24.75±0.83^{b}	22.25±2.28^{a,b}	22.75±2.05^{b}
Calcium (mmol/L)	2.36±0.26^{a,b}	2.21±0.13^{a}	2.41±0.07^{b}	2.34±0.10^{a,b}	2.47±0.08^{b}
Chloride (mmol/L)	100.75±2.05^{a}	100.25±0.83^{a}	100.0±2.55^{a}	99.75±2.49^{a}	100.75±4.26^{a}
Potassium (mmol/L)	4.80±0.070^{a,c}	3.90±0.39^{b,d}	4.18±0.24^{b,d}	4.65±0.46^{a,b}	4.48±0.40^{c,d}
Sodium (mmol/L)	141.50±1.12^{a}	141.00±1.22^{a}	141.25±2.28^{a}	141.50±2.60^{a}	144.50±2.69^{a}

Values are mean ± SD, n=5, per group. Values in the same row with the different superscripts are significantly different at p<0.05.

The plasma conjugated bilirubin level of SST15 was significantly (p<0.05) lower than control and test control, but not different from reference group and SST20. The plasma unconjugated bilirubin level of SST15 was significantly (p<0.05) lower than control, but not different from test control, reference treatment and SST20.

There were no significant differences in the plasma total protein, albumin, chloride, sodium and unconjugated/conjugated bilirubin ratio of all the groups. The plasma bicarbonate level of SST20 was significantly (p<0.05) higher than control, but not different from test control, reference treatment and SST15.

The plasma calcium concentration of SST20 was significantly ($p<0.05$) higher than test control, but not different from control, reference treatment and SST15. The plasma potassium level of the test groups were not significantly different from the control, test control and reference treatment group. The extract improved the lowered plasma calcium level produced by the diabetic condition. The significance of this cannot be overemphasized.

Many neuromuscular and other cellular functions depend on the maintenance of the ionized calcium concentration in the extracellular fluid [Crook, 2006]. Calcium fluxes are also important mediators of hormonal effects on target organs through several intracellular signaling pathways, such as the phosphoinositide and cyclic adenosine monophosphate systems [FAO, 2004; Crook, 2006]. The extract may have achieved this by affecting parathyroid hormone secretion. This hormone increases the renal tubular reabsorption of calcium, promotes intestinal calcium absorption by stimulating the renal production of 1,25-dihydroxyvitamin D or calcitriol [1,25-(OH)2D], and, if necessary, resorbs bone [Brown and Hebert, 1997, Crook, 2006]. Collectively, these results indicate that the extract does not significantly alter the liver and kidney functions, as well as plasma electrolyte balance (except for calcium).

CONCLUSIONS

In conclusion, the treatment did not produce significant differences in the plasma marker enzyme activities and plasma chemistry compared to test control, except for the plasma alanine transaminase activity, plasma calcium and conjugated bilirubin concentrations. All of these results indicate a dose dependent control of plasma glucose level by the extract, as well as a possible protective mechanism against the development of cardiovascular complications, via dyslipidemic conditions, and the development of hypocalcaemia.

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ABOUT THE AUTHOR

Catherine Chidinma Ikewuchi is a Lecturer at the Department of Biochemistry, Faculty of Science, University of Port Harcourt, Port Harcourt, Nigeria. She holds a Master of Science (M.Sc.) degree in Biochemistry from the University of Port Harcourt, Port Harcourt, Nigeria. Her research interests are in the area of Nutritional Biochemistry and Toxicology.

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