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Hypoglycemic, Hypocholesterolemic and Ocular-protective Effects of an Aqueous Extract of the Rhizomes of *Sansevieria senegambica* Baker (Agavaceae) on Alloxan-Induced Diabetic Wistar Rats

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ABSTRACT

In this study, the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the haematology, plasma biochemistry and ocular indices of oxidative stress were investigated in alloxan induced diabetic rats. Diabetes mellitus was induced by injection of alloxan (80 mg kg^{-1} body weight), via the tail vein. The extract was administered orally at 100, 200 and 300 mg kg^{-1} (both to normal and diabetic rats) and metformin at 50 mg kg^{-1}. On gas chromatographic analysis of the flavonoid fraction of the crude aqueous extract, twenty nine known flavonoids were detected, consisting mainly of 31.46% apigenin, 20.47% quercetin, 11.21% kaempferol, 5.75% (-)-epicatechin, 5.75% naringenin, 3.60% biochanin, 3.58% (+)-catechin. Compared to test control, the treatment significantly lowered ($p<0.05$) ocular malondialdehyde content, atherogenic indices, total white cell and monocyte counts; and plasma levels of glucose, triglyceride, total-, very low density lipoprotein-, low density lipoprotein- and non-high density lipoprotein cholesterols, total and conjugated bilirubins, total protein, sodium, urea, blood urea nitrogen, as well as plasma activities of alkaline phosphatase, alanine and aspartate transaminases. However, the treatment significantly increased ($p<0.05$) haematocrit, haemoglobin concentration, red cell, lymphocyte and platelet counts, mean cell volume, ocular vitamin C content and plasma levels of high density lipoprotein cholesterol, potassium, chloride and bicarbonate and ocular activities of catalase and superoxide dismutase. Besides confirming the presence of pharmacologically active compounds in the rhizome extract, this study also showed the hypoglycemic, hypolipidemic, anti-anemic, immune-modulating, ocular-, hepato-renal and cardio-protective potentials of the extract. All these, support the use of the rhizomes of *Sansevieria senegambica* in African traditional health care practices for the management of diabetes mellitus.

Key words: Apigenin, haematology, hypoglycemia, lipid profile, ocular oxidative stress, quercetin, *Sansevieria senegambica* Baker

INTRODUCTION

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia (elevated glucose in the plasma) resulting from defects in insulin secretion, insulin action, or both (Wardlaw, 1999; American Diabetes Association, 2004; Centers for Disease Control and Prevention, 2005). The global number of individuals with diabetes in 2000 was estimated to be 171 million (2.8% of the world's population), a figure projected to increase in 2030 to 366 million (6.5%), 298 million of

whom will live in developing countries (Wild *et al.*, 2004; WHO, 2004; Kengne *et al.*, 2005; Howes, 2006). The estimated prevalence of diabetes in Africa is 1% in rural areas, up to 5-7% in urban sub-Saharan Africa and between 8 and 13% in more developed areas (Sobngwi *et al.*, 2001; Amoah *et al.*, 2002; Kengne *et al.*, 2005; Howes, 2006). The increase in incidence of diabetes in the developing countries follows the trend of urbanization and lifestyle changes and probably, increases in obesity and decreasing amounts of exercise (Howes, 2006). In Nigeria and other African populations, the past two decades has witnessed the emergence of type 2 diabetes mellitus as a major health problem, affecting about 2-7% of these populations (Amos *et al.*, 1997). In 2006, the International Diabetes Federation (IDF) estimated that 10.8 million sub-Saharan Africans had type 2 diabetes mellitus. It also predicted that there will be 18.7 million diabetics in Africa by 2025 which represents an 80% increase and is much greater than the 55% increase in type 2 diabetes mellitus expressed worldwide (Imoisili and Sumner, 2009). The WHO (2004) report estimated that 1.7 million people in Nigeria had diabetes, with the projection that the number will triple by 2030.

Presently, there is renewed interest in the use of herbal products. This may be attributable to the down turn in the economy, as herbal medicine is perceived to be a cheaper means of treatment (Acuff *et al.*, 2007). Plant products can improve glucose metabolism and the overall condition of individuals with diabetes, not only by hypoglycemic effects but also by improving lipid metabolism, antioxidant status and capillary function (Bailey and Day, 1989). *Sansevieria senegambica* is one of a number of medicinal herbs that is used in traditional health care practices for the management of diabetes mellitus. *Sansevieria senegambica* (Family Agavaceae or Ruscaceae) belongs to the genus *Sansevieria*, whose common names include mother-in-laws tongue, devils tongue and snake plant. This genus consists of about sixty species of flowering plants, native to tropical and subtropical regions of the world (Evans, 2005). The leaves appear flattened toward the tip end with a slim point and a surface that is a matte-green with faint banding. It is grown as an ornamental plant (USDA, 2008). In Kaïn, northern Yatenga, Burkina Faso, the dried powder of *S. senegambica* is used in the preservation of grains (Jarvis *et al.*, 2000). In traditional health care practice, especially in Southern Nigeria, it is used for the management of bronchitis, inflammation, cough, boils and gonorrhea (Omobuwajo *et al.*, 2008), arresting the effects of snake bites, as well as in compounding solutions used as hair tonic and in the management of diabetes mellitus, hypertension and liver problems. The weight reducing, hypoglycemic and hypocholesterolemic activities of the leaves have been reported (Ikewuchi, 2010; Ikewuchi *et al.*, 2011a). The present study investigated the effects of an aqueous extract of the rhizomes of *S. senegambica* on haematology, plasma biochemistry and ocular indices of oxidative stress in alloxan-induced diabetic Wistar rats.

MATERIALS AND METHODS

Preparation of plant extract: Samples of fresh whole *S. senegambica* plants (Fig. 1) were procured from a horticultural garden at the University of Port Harcourt's Abuja campus and from behind the Ofrima complex, University of Port Harcourt, in Port Harcourt, Nigeria. After due identification at the University of Port Harcourt's Herbarium, the identity was confirmed/authenticated by Dr. Michael C. Dike of Taxonomy Unit, Department of Forestry and Environmental Management, Michael Okpara University of Agriculture, Umudike, Abia State, Nigeria; and Mr. John Ibe, the Herbarium Manager of the Forestry Department, National Root Crops Research Institute (NRCRI), Umuahia, Nigeria. The rhizome was removed, cleaned of soil,

Fig. 1: *Sansevieria senegambica* Baker

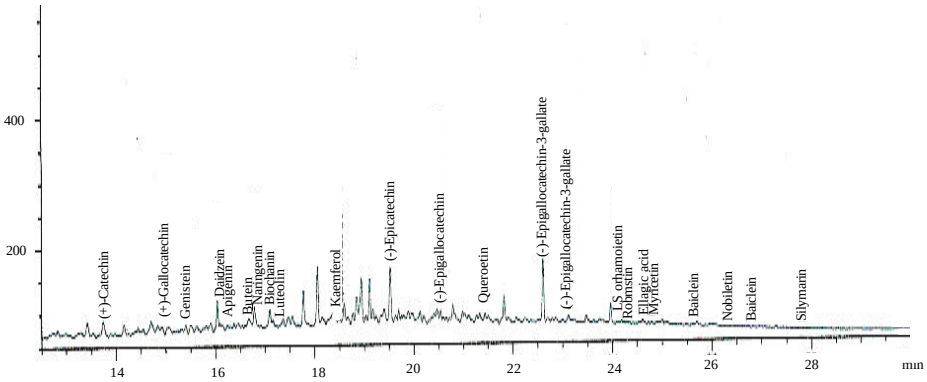

Fig. 2: Chromatogram of the flavonoid fraction of an aqueous extract of the rhizomes of *Sansevieria senegambica*

oven dried at 55°C and ground into powder. The resultant powder was soaked in hot, boiled distilled water for 12 h, after which the resultant mixture was filtered and the filtrate was stored in the refrigerator 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. The percentage recovery of the crude aqueous extract was 22.1%. The resultant residue of the crude aqueous extract was use for the phytochemical study, to determine its flavonoid composition.

Determination of the phytochemical content of the aqueous crude extract

Calibration, identification and quantification: Standard solutions were prepared in methanol. The linearity of the dependence of response on concentration was verified by regression analysis. Identification was based on comparison of retention times and spectral data with standards. quantification was performed by establishing calibration curves for each compound determined, using the standards. A sample chromatogram of the extract is shown in Fig. 2.

Determination of flavonoid composition of the crude aqueous extract: The extraction was carried out according to the method of Kini *et al.* (2009). The sample was extracted with methanol and the resultant extract was subjected to gas chromatographic analysis. Chromatographic analyses were carried out on an HP 6890 (Hewlett Packard, Wilmington, DE, USA), GC apparatus, fitted with a flame ionization detector and powered with HP Chemstation Rev. A 09.01 (1206) software, to quantify and identify compounds. The column was a capillary HP INNOWax Column (30 m×0.25 mm×0.25 μm film thickness). The inlet and detection temperatures were 250 and 320°C. Split injection was adopted with a split ratio of 20:1. Nitrogen was used as the carrier gas. The hydrogen and compressed air pressures were 22 and 35 psi. The oven was programmed as follows: initial temperature at 50°C, first ramping at 8°C min^{-1} for 20 min, maintained for 4 min, followed by a second ramping at 12°C min^{-1} for 4 min, maintained for 4 min.

Experimental design the antidiabetic study: Male wistar albino rats (180-210 g) were collected from the animal house of the Department of Physiology, University of Nigeria, Enugu Campus. Studies were conducted in compliance with the applicable laws and regulations for handling experimental animals. The rats were weighed and sorted into nine groups (Table 1) of five animals each, so that their average weights were approximately equal. The animals were housed in plastic cages at the animal house of the Department of Biochemistry, University of Port Harcourt. After a 1-week acclimatization period on guinea growers mash (Port Harcourt Flour Mills, Port Harcourt, Nigeria), the animals were fasted overnight and their baseline fasting blood glucose level determined using multi Carein™ glucose strips and glucometer (Biochemical Systems International, Arezzo, Italy), by collecting blood via tail cut. Diabetes was induced by injection of a freshly prepared solution of alloxan (80 mg kg^{-1} body weight) in normal saline, via the tail vein of five groups while the other four groups were injected with normal saline alone. The dosage of administration of alloxan was adopted from Radwan (2001). Three days after administration of the alloxan, the animals were again fasted and blood collected via tail cutting blood (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 150% of the control). Then the rats were kept for 3 days to stabilize the diabetic condition (Maroo *et al.*, 2002) before commencing the treatment which lasted for ten days. The Diabetmin™ (metformin HCl) and extracts were administered daily by intra-gastric gavages. The dosages of administration of the extracts were adopted and modified from Ikewuchi (2010) and Ikewuchi *et al.* (2011a). The animals were allowed food and water *ad libitum*. The fasting glucose levels were taken on days 5 and 10. The animals

Table 1: Experimental design for the anti-diabetic screening

ID	Treatment (mg kg^{-1})
Normal	Normal saline and water
Test control	Alloxan and water
Reference treatment (Reference)	Alloxan and metformin (50 body weight)
Treatment control I (SRC1)	Normal saline and extract (100)
Treatment control II (SRC2)	Normal saline and extract (200)
Treatment control III (SRC3)	Normal saline and extract (300)
Treatment I (SR1)	Alloxan and extract (100)
Treatment II (SR2)	Alloxan and extract (200)
Treatment III (SR3)	Alloxan and extract (300)

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 and EDTA sample bottles. Whole blood was immediately used to determine the triglyceride levels, using multiCarein strips (Biochemical Systems International, Arezzo, Italy). Then the eyes were removed and stored for the determination of the ocular markers of oxidative stress. The heparin anti-coagulated blood samples were centrifuged at 1000 g for 10 min, after which their plasma was collected and stored for subsequent analysis while the EDTA anti-coagulated blood samples were used for the haematological analysis.

Assay of the plasma biochemical indices: The plasma glucose concentration was determined using the multiCarein™ glucose strips and glucometer (Biochemical Systems International, Arezzo, Italy). Plasma triglyceride concentration was determined using multiCarein™ triglyceride strips and glucometer. Plasma total and high density lipoprotein cholesterol concentrations were assayed enzymatically with Randox commercial test kits (Randox Laboratories Ltd., Crumlin, England, UK). 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 500 nm.

Plasma VLDL- and LDL-cholesterol concentrations were calculated using the Friedewald equation (Friedewald *et al*., 1972) as follows:

$$[\text{LDL cholesterol}](\text{mmol L}^{-1}) = [\text{Total cholesterol}] - [\text{HDL cholesterol}] - \frac{[\text{Triglayceride}]}{2.2} \quad (1)$$

$$[\text{LDL cholesterol}](\text{mmol L}^{-1}) = \frac{[\text{Triglayceride}]}{2.2} \quad (2)$$

While the plasma non-HDL cholesterol concentration was determined as reported by Brunzell *et al*. (2008):

$$[\text{Non-HDL cholesterol}] = [\text{Total cholesterol}] - [\text{HDL cholesterol}] \quad (3)$$

The atherogenic indices were calculated as earlier reported by Ikewuchi and Ikewuchi (2009a, b, 2010) using the following formulae:

$$\text{CardiacRisk Ratio (CRR)} = \frac{[\text{Total cholesterol}]}{[\text{HDL cholesterol}]} \quad (4)$$

$$\text{Atherogenic Coefficient (AC)} = \frac{[\text{Total cholesterol}] - [\text{HDL cholesterol}]}{[\text{HDL cholesterol}]} \quad (5)$$

$$\text{Atherogenic Index of Plasma (AIP)} = \log \frac{[\text{Triglyceride}]}{[\text{HDL cholesterol}]} \quad (6)$$

The plasma activities of alanine and aspartate transaminases and alkaline phosphatase, were determined using Randox test kits (Randox Laboratories Ltd., Crumlin, England, UK). The activities of alanine and aspartate transaminases were, respectively measured by monitoring at 546 nm, the concentrations of pyruvate and oxaloacetate hydrazones formed with 2,4-dinitrophenylhydrazine. The activity of alkaline phosphatase was determined by monitoring the degradation of p-nitrophenylphosphate to p-nitrophenol, at 405 nm.

Plasma total and conjugated bilirubin, urea and creatinine concentrations were determined using Randox test kits (Randox Laboratories Ltd., Crumlin, England, UK). The wavelength for the determination of conjugated bilirubin and urea was 546 nm and that of total bilirubin was 578 nm. Plasma total protein was determined by the Biuret method using Randox test kits and the concentration of the resultant coloured complex was measured at 560 nm.

Plasma sodium and potassium concentration was determined by flame photometry, according to AOAC Official Method 956.01 (AOAC, 2006). Plasma calcium concentration was determined by the cresol phthalein complexone method (Baginski *et al*., 1973) and the concentration of the resultant complex was measured at 575 nm. Plasma chloride and bicarbonate concentrations were determined by titrimetric methods (Cheesbrough, 2004).

Determination of the haematological indices: Haematological indices were determined using Medonic M16 Haematological Analyser (Nelson Biomedical Limited., UK).

Assay of ocular indices of oxidative stress: Each eye was homogenized in 4 mL of 0.001 mol L^{-1} phosphate buffer (pH 7.4). The resultant homogenate was centrifuged at 1000×g for 15 min and the supernatants were collected and stored in the refrigerator for the assays. The protein contents of the homogenates were determined by the biuret method, using Randox test kits.

The method adopted for the analysis of malondialdehyde was that of Hunter *et al*. (1963) as modified by Gutteridge and Wilkins (1982). The concentration of the resultant malondialdehyde-thiobarbituric acid complex (or adduct) was measured at 532 nm. Ascorbic acid content was estimated by iodine titration as reported by Ikewuchi and Ikewuchi (2011) and Ikewuchi *et al*. (2011b). Catalase activity was determined according to the method of Beers and Sizer (1951). The concentration of the residual hydrogen peroxide was measured at 420 nm. Superoxide dismutase activity was determined according to the method of Misra and Fridovich (1972). The degree of inhibition of the auto-oxidation of adrenaline (which reflects the activity of superoxide dismutase) was determined by measuring the concentration of the resultant adrenochrome, at 520 nm. The amount of enzyme that produced 50% inhibition was defined as one unit of the enzyme activity.

Statistical analysis of data: All values are reported as the Mean±SD (standard deviation). The values of the variables were analysed for statistically significant differences using the Student's t-test, with the help of SPSS Statistics 17.0 package (SPSS Inc., Chicago Ill). The $p<0.05$ was assumed to be significant.

RESULTS

Table 2 shows the flavonoid composition of an aqueous extract of the rhizomes of *Sansevieria senegambica*. Twenty nine known flavonoids were detected, consisting mainly of 31.46% apigenin,

Table 2: The composition of the flavonoid fraction of an aqueous extract of the rhizomes of *Sansevieria senegambica*

Compounds	Retention time (min)	Composition (mg kg^{-1})
(+)-Catechin	13.741	546.101
(+)-Gallocatechin	15.035	157.565
Genistein	15.620	166.459
Daidzein	16.041	411.965
Apigenin	16.248	4796.769
Butein	16.673	383.515
Naringenin	16.785	876.981
Biochanin	17.095	549.012
Luteolin	17.364	115.338
Kaempferol	18.496	1709.270
(-)-Epicatechin	19.524	877.405
(-)-Epigallocatechin	20.472	198.659
Quercetin	21.439	3120.495
(-)-Epicatechin-3-gallate	22.606	14.810
(-)-Epigallocatechin-3-gallate	23.118	92.683
Isorhamnetin	24.097	35.694
Robinetin	24.188	274.127
Ellagic acid	24.611	333.741
Myricetin	24.789	101.161
Baicalein	25.695	206.173
Nobiletin	26.350	222.456
Baicalin	26.951	41.630
Silymarin	27.802	14.147
Total		15246.154

Table 3: The time course of the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma glucose profiles of normal and alloxan-induced diabetic rats

		Day 5		Day 10	
Treatment groups	Day 0 (mg dL^{-1})	Value (mg dL^{-1})	% Reduction	Value (mg dL^{-1})	% Reduction
Normal	96.00±2.92^{a,e}	98.35±4.46^{a}	-2.61±7.29^{a}	104.20±5.07^{a}	-8.73±8.17^{a,c}
Test control	155.40±5.73^{c}	208.00±5.10^{c,*}	-33.94±4.13^{c}	179.50±8.20^{c,*}	-15.67±7.36^{c}
Reference	125.90±1.95^{d}	89.33±2.10^{d,f,*}	29.05±0.64^{d}	110.00±7.52^{a,d,*}	12.57±6.88^{d}
SRC1	99.80±5.40^{e}	56.00±9.82^{e,*}	44.16±7.22^{e}	83.00±6.60^{e,*}	16.66±7.58^{d}
SRC2	99.00±1.22^{a,e}	85.00±3.46^{f,*}	14.13±3.67^{f}	85.25±6.14^{e,*}	13.87±6.47^{d}
SRC3	92.50±6.10^{a}	98.60±7.33^{a,d}	-7.26±13.57^{a,b}	83.50±7.83^{e,*}	9.72±6.31^{d}
SR1	167.25±5.21^{f}	156.33±5.76^{g,*}	6.45±4.63^{b}	129.00±9.51^{b,*}	22.72±7.57^{b,d}
SR2	318.93±3.16^{b}	176.40±4.83^{b,*}	44.68±1.80^{e}	133.80±8.35^{b,*}	58.03±2.96^{e}
SR3	125.20±3.56^{d}	102.00±3.16^{a,*}	18.49±2.93^{f}	87.33±2.24^{e,*}	30.22±1.85^{b}

Values are Mean±SD, n = 5, per group. $^{a, b}$Values in the same column with different superscripts letters are significantly different at $p<0.05$. *$p<0.05$ compared to corresponding values on day 0. % reduction: percentage reduction from the corresponding values on day 0

20.47% quercetin, 11.21% kaempferol, 5.75% (-)-epicatechin, 5.75% naringenin, 3.60% biochanin, 3.58% (+)-catechin, 2.70% daidzein, 2.52% butein, 2.19% ellagic acid, 1.80% robinetin, 1.46% nobiletin, 1.35% baicalein, 1.30% (-)-epigallocatechin, 1.09% genistein and 1.03% (+)-gallocatechin.

The time course of the hypoglycemic effect of an aqueous extract of the rhizomes of *Sansevieria senegambica* on normal and alloxan-induced diabetic rats is presented in Table 3. On day 0, the

Table 4: Effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma lipid profile of normal and alloxan-induced diabetic rats

	Concentration (mmol L^{-1})					
Treatment groups	Triglyceride	Total cholesterol	HDL cholesterol	VLDL cholesterol	LDL cholesterol	Non-HDL cholesterol
Normal	0.84±0.09^{a,d}	1.74±0.08^{a}	1.11±0.04^{a}	0.38±0.04^{a,d}	0.25±0.10^{a,d}	0.63±0.11^{a,e}
Test control	1.04±0.06^{c}	2.24±0.14^{c,d}	0.73±0.05^{c}	0.47±0.03^{c}	1.05±0.12^{c}	1.53±0.10^{c}
Reference	0.88±0.03^{d}	1.89±0.11^{a}	1.24±0.07^{d}	0.40±0.01^{d}	0.25±0.08^{d}	0.65±0.07^{a,e}
SRC1	0.80±0.06^{a}	1.98±0.17^{a,d}	0.86±0.20^{b,c}	0.37±0.03^{a}	0.75±0.03^{e}	1.11±0.05^{d}
SRC2	0.81±0.04^{a,d,e}	1.71±0.21^{a}	1.14±0.21^{a,b,d}	0.37±0.02^{a,b,d}	0.20±0.10^{a}	0.57±0.09^{e}
SRC3	0.82±0.04^{a,e}	2.14±0.09^{c,d}	1.23±0.17^{a,d}	0.37±0.02^{a,b}	0.54±0.17^{b,f}	0.91±0.18^{b,f}
SR1	0.70±0.12^{b,e}	2.22±0.11^{c}	1.13±0.06^{a,b}	0.32±0.05^{b,e}	0.77±0.19^{e,f}	1.09±0.15^{b,d}
SR2	0.84±0.04^{a,d,e}	1.85±0.11^{a,b}	1.12±0.11^{a,b,d}	0.38±0.02^{a,b,d}	0.35±0.07^{a,b}	0.73±0.07^{a,f}
SR3	0.68±0.02^{b}	1.78±0.10^{a,b}	1.09±0.14^{a,b,d}	0.31±0.01^{e}	0.39±0.14^{b}	0.69±0.15^{a,e}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same column with different superscripts are significantly different at $p<0.05$

Table 5: Effects of an aqueous extract of the rhizomes *Sansevieria senegambica* on the atherogenic indices of normal and alloxan-induced diabetic rats

Treatment groups	Cardiac risk ratio	Atherogenic coefficient	Atherogenic index of plasma
Normal	1.57±0.11^{a}	0.57±0.11^{a}	-0.12±0.05^{a,d,e}
Test control	3.17±0.15^{c}	2.17±0.15^{c}	0.17±0.05^{c}
Reference	1.52±0.06^{a}	0.52±0.06^{a}	-0.15±0.04^{d}
SRC1	2.37±0.43^{b}	1.37±0.43^{b}	-0.02±0.12^{d,e}
SRC2	1.51±0.15^{a}	0.52±0.15^{a}	-0.14±0.07^{a,b,d,e}
SRC3	1.77±0.28^{a,d}	0.77±0.28^{a,d}	-0.17±0.07^{a,b,d}
SR1	1.97±0.19^{b,d,e}	0.97±0.19^{b,d,e}	-0.21±0.06^{b}
SR2	1.65±0.12^{a}	0.65±0.12^{a}	-0.13±0.03^{a,e}
SR3	1.66±0.19^{a,e}	0.66±0.19^{a,e}	-0.20±0.07^{b}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same column with different superscripts are significantly different at $p<0.05$

plasma fasting glucose concentration of the alloxan treated animals were significantly higher ($p<0.05$) than the untreated animals (normal, SRC1, SRC2 and SRC3). On days 5 and 10, the plasma fasting glucose levels of the animals administered the extracts were significantly lower ($p<0.05$) than corresponding test controls and values on day 0. The percentage reductions in plasma fasting glucose levels of the treated rats on days 5 and 10, were significantly higher ($p<0.05$) than the corresponding values of the test control group.

The effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma lipid profiles of normal and alloxan-induced diabetic rats is shown in Table 4. The plasma triglyceride, total, VLDL-, LDL- and non-HDL cholesterol levels of the test control group were significantly higher ($p<0.05$) than those of the other groups; while their plasma HDL cholesterol levels was significantly lower ($p<0.05$).

Table 5 shows the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the atherogenic indices of normal and alloxan-induced diabetic rats. The atherogenic indices (cardiac risk ratio, atherogenic coefficient and atherogenic index of plasma) of the test control group, were significantly higher ($p<0.05$) than those of the other groups.

The effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the levels of plasma markers of liver integrity and function in normal and alloxan-induced diabetic rats is given in Table 6. The plasma activities of alkaline phosphatase, alanine and aspartate transaminases,

Table 6: Effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma hepatospecific markers of normal and alloxan-induced diabetic rats

	Magnitude							
Treatment groups	Alkaline phosphatase activity (U L^{-1})	Alanine transaminase activity (U L^{-1})	Aspartate transaminase activity (U L^{-1})	Total bilirubin (µmol L^{-1})	Conjugated bilirubin (µmol L^{-1})	Unconjugated bilirubin (µmol L^{-1})	Unconjugated /conjugated bilirubin ratio	Total protein (mg dL^{-1})
Normal	176.64±2.75^{a}	13.66±0.82^{a}	21.58±0.93^{a}	2.93±0.22^{a,f}	2.27±0.19^{a,d}	0.66±0.12^{a}	0.29±0.06^{a,e}	50.96±1.84^{a}
Test control	567.87±6.40^{b}	26.61± 0.88^{c}	27.05±0.76^{c}	3.64±0.21^{c}	2.71±0.22^{c}	0.93±0.04^{c}	0.35±0.04^{a,e}	59.99±0.92^{c}
Reference	316.19±4.49^{c}	21.00±0.63^{d}	25.92±2.04^{c,d}	3.52±0.08^{d}	2.21±0.17^{a,d}	1.30±0.13^{d}	0.59±0.11^{c,d}	57.61±0.93^{d}
SRC1	462.88±14.56^{d}	21.56±0.88^{d}	23.37±1.89^{d,e}	2.96±0.05^{a,f}	2.15±0.18^{d}	0.81±0.13^{a,c}	0.38±0.09^{a,c,d,e}	48.09±2.43^{a}
SRC2	382.26±6.33^{e}	16.67±0.75^{e}	16.25±1.73^{b}	2.71±0.15^{e}	1.72±0.15^{e}	0.99±0.02^{b,e}	0.58±0.05^{d}	53.86±1.28^{e}
SRC3	223.56±5.21^{f}	13.52±0.92^{a}	16.66±1.55^{b}	3.25±0.18^{b}	2.34±0.18^{a,d}	0.91±0.06^{b,c}	0.39±0.04^{e}	58.06± 1.20^{d}
SR1	485.76±2.48^{g}	23.51±0.88^{b}	24.65±0.86^{d}	3.10±0.18^{a,b}	2.34±0.19^{a,c,d}	0.76±0.02^{a}	0.33±0.03^{a}	55.68±0.77^{b,e}
SR2	383.64±2.21^{e}	25.00±0.71^{b}	18.19±0.60^{b}	2.78±0.14^{e,f}	1.31±0.14^{b}	1.46±0.10^{d}	1.13±0.16^{b}	49.43±0.68^{a}
SR3	240.12±6.22^{h}	15.98±0.63^{e}	22.16±1.07^{a,e}	3.33±0.20^{b,d}	2.30±0.18^{a}	1.03±0.06^{e}	0.45±0.04^{c}	56.14±0.66^{b,d}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same column with different superscripts are significantly different at $p<0.05$

Table 7: Effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma electrolyte profiles of normal and alloxan-induced diabetic rats

	Concentration						
Treatment groups	Urea (mmol L^{-1})	Blood urea nitrogen (mg dL^{-1})	Calcium (mmol L^{-1})	Sodium (mg dL^{-1})	Potassium (mg dL^{-1})	Chloride (meq L^{-1})	Bicarbonate (meq L^{-1})
Normal	20.70±1.08^{a}	58.15±3.03^{a}	2.06±0.08^{a,c}	129.15±1.82^{a,f}	5.23±0.07^{a,e}	98.83±0.84^{a,e}	18.20±0.84^{a,d}
Test control	28.70±1.00^{c}	80.62±2.82^{c}	1.89±0.06^{b,d}	148.67±1.75^{c}	5.00±0.07^{c}	84.75±0.43^{c}	15.25±0.50^{c}
Reference	12.65±0.52^{d}	35.55±1.45^{d}	1.97±0.06^{c,d}	130.00±1.58^{a,d,f}	9.20±0.10^{d}	114.66±0.85^{d}	18.00± 0.71^{d}
SRC1	17.58±0.81^{e}	49.37±2.27^{e}	1.94±0.07^{a,b,c}	127.25±2.30^{d,e}	5.24±0.05^{e}	99.75±0.83^{a,e}	15.50±0.50^{c}
SRC2	18.53±0.97^{b}	52.04±2.72^{b}	2.03±0.04^{c}	134.50±1.66^{b}	6.00±0.10^{f}	98.50±0.71^{e}	19.50±0.50^{b}
SRC3	23.43±0.77^{f}	65.82±2.16^{f}	1.93±0.06^{a,b}	134.00±2.00^{b}	5.65±0.10^{b}	88.25±0.83^{f}	15.50±0.50^{c}
SR1	19.48±1.01^{a,b,e}	54.71±2.83^{a,b,e}	1.92±0.04^{a,d}	130.75±0.83^{a}	5.22±0.04^{a,e}	101.75±0.83^{b}	19.04±0.15^{a,b}
SR2	19.00±0.60^{b}	53.37±1.70^{b}	1.93±0.07^{a,d}	128.33±1.49^{e,f}	5.16±0.05^{a}	100.67±0.62^{b,g}	19.00±0.35^{a,b,d}
SR3	24.70±0.78^{f}	69.38±2.19^{f}	1.90±0.05^{a,d}	130.33±1.11^{a,e}	5.19±0.05^{a,e}	100.00±0.71^{a,g}	17.67±0.32^{d}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same column with different superscripts are significantly different at $p<0.05$

as well as the plasma levels of total and conjugated bilirubin and total protein, of the test control group was significantly higher ($p<0.05$) than those of the other groups. However, the levels of unconjugated bilirubin and unconjugated/conjugated bilirubin ratio of the test control group was significantly lower ($p<0.05$) than that of the test groups. Table 7 shows the effect of aqueous extract of the rhizomes of *Sansevieria senegambica* on the plasma electrolyte profiles of normal and alloxan-induced diabetic rats. The levels of plasma sodium, urea and blood urea nitrogen of the test control group was significantly higher ($p<0.05$) than those of the other groups. The levels of plasma potassium, chloride and bicarbonate in the test control was significantly lower ($p<0.05$) than those of the other groups while the plasma calcium of the test control was not significantly different from the diabetic treated groups.

The effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on ocular markers of oxidative stress in normal and alloxan-induced diabetic rats is presented in Table 8. The ocular vitamin C content and catalase and superoxide dismutase activities of the test control animals were

Table 8: Effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on ocular markers of oxidative stress in normal and alloxan-induced diabetic rats

Treatment groups	Ascorbic acid content (μmole g^{-1} protein)	Malondialdehyde content (μmol g^{-1} protein)	Catalase activity (Units mg^{-1} protein)	Superoxide dismutase activity (Units mg^{-1} protein)
Normal	11.23±0.47^{a}	0.09±0.00^{a,f}	20.60±0.85^{a}	0.20±0.00^{a}
Test control	8.30±0.01^{b}	0.10±0.00^{c}	18.84±0.19^{b}	0.16±0.00^{b}
Reference	8.72±0.11^{c}	0.07±0.00^{d}	23.43±0.53^{c}	0.18±0.00^{c}
SRC1	11.97±0.20^{d}	0.07±0.00^{e}	22.12±0.85^{d,g}	0.17±0.00^{d}
SRC2	13.79±0.20^{e}	0.09±0.00^{f}	30.10±0.14^{e}	0.26±0.00^{e}
SRC3	9.77±0.30^{f}	0.08±0.00^{g}	25.84±0.40^{f}	0.16±0.00^{b}
SR1	17.71±0.19^{g}	0.09±0.00^{h}	22.15±0.60^{g}	0.25±0.00^{g}
SR2	16.75±0.25^{h}	0.09±0.00^{b}	28.81±0.72^{h}	0.23±0.00^{h}
SR3	12.07±0.06^{d}	0.09±0.00^{a,b}	27.07±0.27^{k}	0.22±0.01^{k}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same column with different superscripts are significantly different at $p<0.05$

Table 9: Effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the haematological indices of normal and alloxan-induced diabetic rats

	Magnitude								
Parameter	Normal	Test control	Reference	SRC1	SRC2	SRC3	SR1	SR2	SR3
Packed cell volume (%)	34.4±0.9^{a}	31.0±1.0^{c}	38.8±0.8^{d}	30.5±1.0^{c}	33.3±0.4^{e}	38.5±0.5^{f,d}	40.3±0.5^{g}	42.7±0.7^{b}	35.0±1.0^{a}
Haemoglobin concentration (g dL^{-1})	12.0±0.1^{a}	11.4±0.2^{c}	12.3±0.4^{a,d}	10.8±0.3^{b}	11.1±0.1^{b,c}	12.7±0.2^{d}	13.1±0.2^{e}	13.4±0.2^{f}	11.9±0.1^{a}
Red cell count ($\times10^{9}$ cells L^{-1})	5.7±0.3^{a,c}	5.5±0.0^{c}	6.7±0.3^{d}	5.7±0.3^{a,c}	6.0±0.2^{e}	6.9±0.3^{b,d}	6.7±0.2^{b,d}	6.6±0.5^{b,d}	5.8±0.2^{a}
Total white cell count ($\times10^{9}$ cells L^{-1})	9.7±0.4^{a}	11.95±0.3^{b}	10.2±0.2^{c}	8.7±0.8^{d}	6.8±0.3^{e}	8.5±0.5^{d}	11.2±0.5^{f}	6.5±0.5^{e}	14.1±0.3^{g}
Neutrophils count (%)	5.3±0.4^{a,d,e,f}	3.7±0.5^{c}	5.5±0.5^{d}	9.3±0.4^{e}	5.5±0.5^{d}	3.5±0.5^{a,c}	2.7±0.3^{b,f}	8.7±0.6^{e}	2.7±0.3^{a,b}
Lymphocytes count (%)	81.7±0.5^{a}	83.3±0.5^{b}	77.5±0.53^{c}	81.0±0.6^{d}	86.8±0.8^{e}	90.5±0.5^{f}	89.0±0.7^{g}	90.0±0.7^{f}	93.7±0.5^{h}
Monocytes count (%)	12.8±0.5^{a}	12.8±0.8^{a}	15.6±1.1^{c}	9.8±0.6^{d}	8.3±0.4^{e}	6.3±0.4^{f}	8.3±0.5^{e}	4.7±0.6^{b}	4.0±0.7^{b}
Eosinophils count (%)	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}
Basophils count (%)	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}	0.0±0.0^{a}
Mean cell volume (fL)	58.7±1.6^{a,c}	57.5±0.8^{c}	56.5±1.1^{c,d}	53.5±0.6^{e}	54.7±1.2^{d,e}	57.0±0.5^{c}	59.9±0.9^{a}	65.7±1.0^{b}	59.9±0.4^{a}
Mean cell haemoglobin concentration (g dL^{-1})	35.5±0.6^{a}	34.1±0.5^{c}	36.3±0.5^{a}	36.2±0.6^{a}	32.1±0.5^{d}	34.4±0.5^{c}	27.0±1.0^{b}	31.7±0.4^{d}	35.7±0.4^{a}
Mean cell haemoglobin (pg $cell^{-1}$)	20.8±0.7^{a}	19.8±0.6^{a,c,g}	20.5±0.5^{a,d}	19.4±0.8^{c,e,g}	18.9±0.5^{f,g}	19.6±0.4^{c,d}	19.9±0.7^{a,e,f}	20.9±0.7^{a,b,e}	21.5±0.6^{b}
Platelet count ($\times10^{4}$cells mm^{-3})	44.8±0.3^{a}	34.1±0.1^{b}	34.3±0.1^{c}	38.9±0.1^{d}	45.6±0.2^{e}	59.4±0.1^{f}	47.2±0.1^{g}	56.6±0.1^{h}	54.4±0.1^{k}

Values are Mean±SD, n = 5, per group. $^{a, b, c}$Values in the same row with different superscripts are significantly different at $p<0.05$

significantly lower ($p<0.05$) than those of the other groups. The test control group had significantly higher malondialdehyde content than the other groups.

Table 9 shows the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the haematological indices of normal and alloxan-induced diabetic rats while Fig. 3 shows the frequency distribution of the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the red cell morphology of normal and alloxan-induced diabetic rats. 100% of the cells of the animals on 200 and 300 mg kg^{-1} treatment were normochromic while both treatments produced about 75 and 33% normocytic cells, respectively. The 100 mg kg^{-1} treatment and the

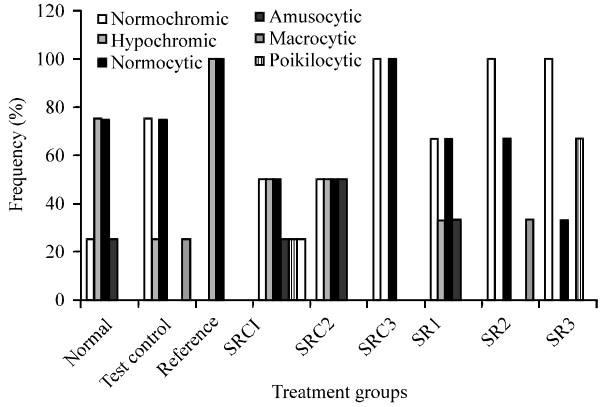

Fig. 3: Frequency distribution of the effects of an aqueous extract of the rhizomes of *Sansevieria senegambica* on the red cell morphology of normal and alloxan-induced diabetic rats

treatment controls produced mixed effects on cell morphology. This shows that the extract had no deleterious effect on red cell morphology. The packed cell volume, haemoglobin concentration, red cell count and platelet count of the test control were significantly lower ($p<0.05$) than those of the diabetic treated groups. The total white cell count of the test control group was significantly higher ($p<0.05$) than the other groups, except SR3 which had significantly higher ($p<0.05$) values. The neutrophil count of the test control group was significantly lower ($p<0.05$) than the other groups, except SR1 and SR2 which had significantly lower ($p<0.05$) values. The lymphocyte count of the test control group was significantly lower ($p<0.05$) than the other groups, except control, reference and SRC1 which had significantly higher ($p<0.05$) values. The monocyte count of the test control group was significantly higher ($p<0.05$) than the other groups, except reference which had a significantly higher ($p<0.05$) value and control which was not significantly lower. The mean cell volume of the test control group was significantly lower ($p<0.05$) than SR1, SR2 and SR3; significantly higher ($p<0.05$) than SRC1 and SRC2 but not significantly different from control, reference and SRC3. The mean cell haemoglobin concentration of the test control group was significantly lower ($p<0.05$) than control, reference, SRC1 and SR3; significantly higher ($p<0.05$) than SRC2, SR1 and SR2 but not significantly different from SRC3. The mean cell haemoglobin of the test control group was not significantly different from those of the other groups, except SR3 which had significantly higher ($p<0.05$) values. There were no significant differences in the eosinophil and basophil counts of all the groups.

DISCUSSION

The extract was rich in a number of bioactive flavonoids. All of these compounds have established antineoplasmic and anticarcinogenic properties (Dillard and German, 2000; Evans, 2005). Some have immunomodulatory, some have hypocholesterolemic while some others have hypoglycemic properties.

Alloxan induced diabetes mellitus is often characterized by decreased insulin level, hyperglycemia, elevated triglycerides and total cholesterol and decreased high density lipoprotein (Hemalatha, 2008). The high percentage reduction in plasma glucose levels, produced by the extract in this study, supports the use of the plant in the management of diabetes mellitus. The hypoglycemic effect of the extract may have been produced by flavonoids (Table 2) present in the

aqueous extract of the rhizomes. The flavonoids with established hypoglycemic activity include apigenin, quercetin, epicatechin, kaempferol, naringin, genistinin and myricetin (Chakravarthy *et al.*, 1982; Ong and Khoo, 1996; Vessal *et al.*, 2003; De Sousa *et al.*, 2004; Jung *et al.*, 2004; Lee, 2006; Panda and Kar, 2007; Tapas *et al.*, 2008). The extract may exert its anti-hyperglycemic activity by enhancing glucose uptake, stimulating insulin secretion from pancreatic α cells and insulin like activity, or by converting pro-insulin to insulin, or alternatively, by inhibiting hepatic gluconeogenesis. Thus, anyone or a combination of some or all of the above mentioned components could have been responsible for the hypoglycemic effect of the extract, observed in this study.

Elevated levels of plasma triglyceride, is both an independent and synergistic risk factor for cardiovascular diseases (Dobiaova, 2004; Martirosyan *et al.*, 2007; McBride, 2007). It is often associated with hypertension (Lopes *et al.*, 1997; Zicha *et al.*, 1999), abnormal lipoprotein metabolism, obesity, insulin resistance and diabetes mellitus (Franz *et al.*, 2002; McBride, 2007; Shen, 2007). The treatments significantly reduced plasma levels of triglycerides (Table 4). This effect may have been mediated by the flavonoid (Table 2) content of the extract. According to Middleton *et al.* (2000), flavonoids decreases plasma of triglycerides levels.

The administration of the extract produced significantly higher plasma HDL cholesterol levels. This portends reduction of cardiovascular risk. According to clinical data, increases in plasma HDL cholesterol concentration decrease cardiovascular risk (Assmann and Gotto, 2004; Rang *et al.*, 2005).

Elevated levels of plasma VLDL cholesterol is a risk factor for cardiovascular disease (Ademuyiwa *et al.*, 2005; Lichtenstein *et al.*, 2006) and often found in the diabetic (Rang *et al.*, 2005; Shen, 2007; Brunzell *et al.*, 2008; Krauss *et al.*, 2006) individuals. In this study, a significantly lower plasma VLDL cholesterol levels was observed in the treated animals compared to test control.

High plasma level of LDL cholesterol is a risk factor for cardiovascular disease (Ademuyiwa *et al.*, 2005; Lichtenstein *et al.*, 2006) and often accompanies hypertension (Shepherd, 1998; Zicha *et al.*, 1999). Conversely, reductions in plasma LDL cholesterol have been considered to reduce risk of coronary heart disease (Rang *et al.*, 2005; Shen, 2007). In this study, a significantly lower plasma LDL cholesterol levels were observed in the animals given the extract, indicating the likely cardio-protective effect of the extracts at that dose.

Many studies have shown that non-HDL cholesterol is a better predictor of cardiovascular disease risk than is LDL cholesterol (Liu *et al.*, 2005; Brunzell *et al.*, 2008). Therefore, the significantly lower plasma non HDL cholesterol observed in the treated diabetic groups indicates the ability of the extract to reduce cardiovascular risk.

This cholesterol lowering effect of the extract may be due to its content of flavonoids, e.g., apigenin, catechin, naringenin and kaempferol (Table 2) which are known to have cholesterol lowering and atheroprotective activity (Lee *et al.*, 1999; Dillard and German, 2000; Chu *et al.*, 2004; Basu *et al.*, 2007; Panda and Kar, 2007; Lau, 2008). Again, anyone or a combination of some or all of the above mentioned components could have been responsible for the hypocholesterolemic effect of the extract, observed in this study.

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 (Brehm *et al.*, 2004; Dobiaova, 2004; Usoro *et al.*, 2006; Martirosyan *et al.*, 2007). Low atherogenic indices are protective against coronary heart disease (Usoro *et al.*, 2006). This again high lights the ability of the extract to protect against cardiovascular complications in the diabetic.

These results show that the extract had a dose dependent positive effect on the integrity and function of the liver and kidney of the diabetic rats. Decreased erythrocyte and plasma potassium concentrations have been associated with glucose intolerance. Potassium depletion causes glucose intolerance which is associated with impaired insulin secretion (Rowe *et al.*, 1980). Therefore, the reversal of the diabetes induced reduction in plasma potassium levels portends ability of the extract to improve insulin activity. The treatment of also reversed the diabetes induced metabolic acidosis in the test animals.

Alloxan induced diabetes in rats is usually accompanied by increases in thiobarbituric acid reactive substances, an indirect evidence of intensified free-radical production (Maritim *et al.*, 2003). Raised levels of these thiobarbituric acid reactants are consistently observed in diabetes (Wollf, 1993; Hartnett *et al.*, 2000; Atalay and Laaksonen, 2002; Rajdl *et al.*, 2005; Samuel *et al.*, 2010). Elevations in blood and tissue levels of thiobarbituric acid-reactive substances, mainly malondialdehyde, are very reliable indices of oxidative stress and lipid peroxidation (Imai *et al.*, 1991; Atalay and Laaksonen, 2002). Therefore, the profiles of malondialdehyde, observed in the test rats clearly indicate that the extract protects against oxidative stress in ocular tissues. This antioxidant protective effect may be due to the presence of flavonoids in the extract (Table 2); a family of compounds that are very strong antioxidants (Dillard and German, 2000; Middleton *et al.*, 2000; Soetan, 2008; Tapas *et al.*, 2008). Prominent among these is apigenin, the highest component of the flavonoid portion of the extract (Table 2) which had earlier been reported to exert a similar effect on the liver of alloxan-induced diabetic mice (Panda and Kar, 2007).

The diabetic state is often accompanied by lowered levels and altered metabolic turnover of ascorbic acid (Atalay and Laaksonen, 2002; Samuel *et al.*, 2010). The high extracellular glucose concentration in the diabetic impairs the cellular uptake of ascorbic acid and accentuates the problems which are associated with its deficiency (Samuel *et al.*, 2010). Studies show that ascorbic acid protects the lens and other tissues of the eye from light damage (Varma, 1991; Taylor, 1993). Therefore, the high ocular ascorbic acid levels caused by the extract, portends a consolidation of antioxidant status of the eyes and the consequent protection of its tissues from free radical damage. This correlates with the observed reduction in the level of lipid peroxidation.

Reduced activities of the antioxidant enzyme systems, often results from the progressive glycation of the enzymatic proteins, in the diabetic (Hartnett *et al.*, 2000; Atalay and Laaksonen, 2002; Samuel *et al.*, 2010). Numerous reports indicate variations in the levels of antioxidants in the diabetic patients (Hartnett *et al.*, 2000; Samuel *et al.*, 2010). In this study, the treatment improved the ocular activities of catalase and superoxide dismutase of the animals.

The extract had a positive effect on the haemopoietic system of the test rats. It significantly increased the red cell mass, haematocrit, haemoglobin concentration, lymphocyte and platelet counts while decreasing total white cell and monocyte counts. The ability of the extract to inhibit diabetes induced anaemia in the test animals may be due the presence of quercetin in the extract (Table 2). Earlier, Sen *et al.* (2005) had reported the antianaemic activity of quercetin.

According to some experimental and pathological studies, white blood cells play important roles in destabilizing coronary artery plaques at the onset of acute coronary syndrome (Moreno *et al.*, 1994; Van der Wal *et al.*, 1994; Libby, 2001). However, an elevated white blood cell count in peripheral blood is a known risk factor of coronary artery disease (Takeda *et al.*, 2003). Therefore, the lower total white blood cell count, seen in the test rats, implied the ability of the extract to protect against diabetes induced increases in total white cell count. It also implies reduction of the risk of coronary artery disease. The reduced white cell count may have been produced by the

immunomodulatory activity of the flavonoids present in the extract (Table 2). The extract contained apigenin, kaempferol, luteolin, epicatechin, quercetin and (-)-epigallocatechin 3-O-gallate (Table 2), all of which had been reported to dose dependently reduce the proliferation of peripheral blood mononuclear cells (Kuo *et al*., 2004; Yoon *et al*., 2006; Vinardell and Mitjans, 2008; Sternberg *et al*., 2008, 2009; Lin *et al*., 2011). Accordingly, Stadelmann (2007) claimed that the immunomodulatory effect of luteolin may be beneficial in the treatment of neurodegenerative diseases such as multiple sclerosis which has an underlying T-cell mediated autoimmune pathology. Similarly Huang *et al*. (2010), posited that the inhibition of dendritic cells maturation and function by quercetin, suggests that quercetin may be a potent immunosuppressant and may have therapeutic applications in inflammatory diseases, such as periodontitis. Lin *et al*. (2011) reported that kaempferol attenuate dendritic cell function, thus suggesting that kaempferol has potential in the treatment of chronic inflammatory and autoimmune diseases.

The observed increase in platelet count has both favorable and unfavorable implications. It implies increase in clotting and protection against bleeding. It is associated with increased insulin resistance and predisposition to adverse cardiovascular events. Increased platelet count is an independent predictor of insulin resistance among non-obese type 2 diabetes mellitus patients (Taniguchi *et al*., 2003). It increases 2-fold, the risk of adverse cardiovascular events (Kaushansky, 2009).

This study showed that the extract was hypoglycemic, positively affected the haemopoietic system and integrity and function (dose dependently) of the liver and kidney of the diabetic rats; improved the lipid profile and had no deleterious effect on red cell morphology. The profiles of malondialdehyde and antioxidant vitamins in the test rats clearly indicate that the extract protects against oxidative stress in ocular tissues. This study also revealed the presence of pharmacologically active compounds in the rhizomes extract. All of these, highlight the cardio-protective potential of the rhizomes of *Sansevieria senegambica* and support its use in traditional health care practices for the management of diabetes mellitus.

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