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## Anti-Hyperglycemic and Anti-Hyperlipidemic Effects of Aqueous Stem Bark Extract of *Acacia albida* Delile. in Alloxan-Induced Diabetic Rats

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### ABSTRACT

This study investigated anti-hyperglycemic and anti-hyperlipidemic effects of aqueous stem bark extract (ASE) of *Acacia albida* Delile. in alloxan induced diabetic rats. Animals were randomly divided into seven groups of five animals each: Normal Control (NC), normal treated with 500 mg kg<sup>-1</sup> b.w. ASE, Diabetic Control (DC), diabetics treated with 125 mg kg<sup>-1</sup> b.w. ASE (DLD), 250 mg kg<sup>-1</sup> b.w. ASE (DMD), 500 mg kg<sup>-1</sup> b.w. ASE (DHD) and 0.08 mg kg<sup>-1</sup> b.w. glibenclimide (DGL). Results showed that, Fasting Blood Glucose (FBG) of DC significantly ( $p < 0.05$ ) increased by 15.35%. Treatment with extract or glibenclimide significantly ( $p < 0.05$ ) caused a reduction on FBG compared in dose-dependent pattern. DHD recorded highest decrease (57.55%) compared to DGL (60.33%). DC also showed significant ( $p < 0.05$ ) decrease in serum HDL cholesterol and increase in total cholesterol, triglycerides and LDL cholesterol in the rats. Treatment with ASE of *Acacia albida* was able to prevent these effects significantly ( $p < 0.05$ ) compared to DC. Other diabetic-induced abnormalities ameliorated were serum markers of liver damage, food and fluid intakes, body weight changes and Packed Cell Volume (PCV). Conclusively, ASE of *Acacia albida* possessed anti-hyperglycemic and anti-hyperlipidemic effects in alloxan induced diabetic rats.

**Key words:** *Acacia albida*, anti-hyperglycemic, anti-hyperlipidemic, diabetes mellitus

### INTRODUCTION

Diabetes Mellitus (DM) is a condition that causes hyperglycemia due to either decreased insulin secretion or decreased insulin sensitivity of target tissues (Panini, 2013). Recent data indicates that more than 371 million people have DM and 4.8 million people died due to DM and these figures are likely to double by 2035 (IDF, 2013). Moreover, inspite of great therapeutic innovations in DM, the existing pharmacological approaches (sulphonylureas, glucosidase inhibitors and biguanide) do not adequately improve the consequences of DM. In addition, they caused characteristic profiles of serious side effects which include hypoglycemia, weight gain, gastrointestinal discomfort, nausea, liver and heart failure and diarrhea (Kane *et al.*, 2005). Therefore, search for novel molecules has been extended to natural plant based remedies, since less or no adverse effects were reported from plant products.

*Acacia albida* Delile. (*Faidherbia albida* Delile A. Chev.) belongs to Mimosaceae family. It is commonly known as Apple-ring acacia or Ana tree and is a native to Africa and some parts of Middle East and Asia. It grows up to 30 m tall, leaves compound and bipinnate with leaflets borne along the pinnae. Flowers are borne in dense axillary panicles 3.5-16 cm long with a peduncle

2-4 cm long. It possessed grey to whitish bark, smooth when young, more fissured and flaky and more cork-like in older specimens (Wood, 1992). Various parts are claimed traditionally to be effective in treatment of various diseases in Nigerian folkloric medicine. Bark decoction is employed in treatment of diarrhea, leprosy, pneumonia cough and in difficulty during delivery to mention a few (Orwa *et al.*, 2009). Stem bark infusion is used to treat chest pain and trypanosomiasis in northern part of Nigeria (Tijani *et al.*, 2008). *Acacia albida* is among the plants almost neglected by research scientist despite its extensive usage and vast popularity in traditional medicines.

Previous studies indicated that *A. albida* stem bark possessed anti-malarial activity (Oluwakanyinsola *et al.*, 2010), antimicrobial (Kubmarawa *et al.*, 2007; Usman *et al.*, 2013), antipyretic, anti-inflammatory and anti-diarrheal effects (Tijani *et al.*, 2008). It was also shown to be relatively safe acutely (Salawu *et al.*, 2010; Lawal *et al.*, 2012). Furthermore, Salisu *et al.* (2009) reported that root bark methanolic extract possessed antidiabetic effect. Similarly, Gaber *et al.* (2013) have shown that seed aqueous extract possessed mild hypoglycemic and hypolipidemic effects in type 2 diabetic patients. More recently, in our study, *A. albida* Aqueous Stem Bark (ASE) demonstrated potent anti-trypansomal activity against *Trypanosoma brucei* (Alhaji *et al.*, 2014). Similarly, both qualitative and quantitative phytochemistry indicated saponins, flavonoids, phenolics and alkaloids to be the major phytochemicals present (Alhaji *et al.*, 2014). To our knowledge, no study is available that investigated anti-hyperglycemic as well as anti-hyperlipidemic effects of *A. albida* ASE. Thus, the present study is aimed to investigate the anti-hyperglycemic and anti-hyperlipidemic effects of the ASE of *A. albida* in alloxan-induced diabetic rats as a way of exploring its potentials in management of DM.

## MATERIALS AND METHODS

**Plant collection:** The ASE of *A. albida* was harvested from gardens around Samaru, Zaria, Nigeria in the month of March, 2012. Identification was done at the Herbarium unit, Department of Biological Sciences, Ahmadu Bello University, Zaria, Nigeria and voucher specimen was deposited.

**Aqueous extract preparation:** One hundred gram (100 g) powdered (pulverized) material was soaked in 1 L of distilled water in a tightly closed round bottom flask at room temperature for a period of 24 h and filtered first through muslin cloth and then Whatman's No. 1 filter paper. The whole process was repeated three times and further heated at 45°C on water bath for 2 h to ensure maximum yield of aqueous soluble compounds. The extract was reconstituted in distilled water when required.

**Experimental animals:** Adult male albino rats of Wistar strain weighing 100-160 g obtained from the Department of Pharmacology, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, Zaria were used in this study. They were housed in well ventilated animal house, maintained under standard condition of temperature 25±5°C, relative humidity 35-60% and 12 h light/dark cycles. All experimental animals were allowed to acclimatize laboratory ambiance for a period of two weeks, fed with standard pellet diet (Vital feeds, Jos) and tap water *ad libitum*, prior to the experiment.

**Induction of diabetes:** DM was induced in the rats by single intraperitoneal injection of 150 mg kg<sup>-1</sup> b.w. of freshly prepared alloxan monohydrate in normal saline. In order to prevent fatal hypoglycemia due to massive pancreatic insulin release, rats were treated with 5%

glucose solution bottles in their cages for a period of 24 h. After three days, rats with FBG level  $>150 \text{ mg dL}^{-1}$  were considered diabetic and used for the study. All protocols were conducted under strict compliance of animal ethics guidelines for laboratory animal use and care of Ahmadu Bello University, Zaria.

**Experimental design:** The study involved thirty five male Wistar strain albino rats. The animals were randomly divided into the following seven experimental groups with five animals in each: group I and II were Normal Control (NC) treated with vehicle and  $500 \text{ mg kg}^{-1} \text{ b.w. ASE}$  (NAA) respectively; group III was diabetic (DC) treated with vehicle, groups IV, V and VI were diabetics administrated orally with  $125 \text{ mg kg}^{-1} \text{ b.w. ASE}$  (DLD),  $250 \text{ mg kg}^{-1} \text{ b.w. ASE}$  (DMD) and  $500 \text{ mg kg}^{-1} \text{ b.w. ASE}$  (DHD); group VII was diabetic treated orally with glibenclimide ( $0.08 \text{ mg kg}^{-1} \text{ b.w.}$ ). The ASE prepared in distilled water was administered to treated animals orally for three weeks. Blood samples from the tail vein were collected for the measurement of FBG. Weekly FBG (using Accu-chek Advantage Glucometer; Roche Diagnostics, USA), body weight (b.w.) and PCV (using Microhaematocrit method) changes were monitored in addition to daily fluid and food intakes. At the end of three weeks experimental period, animals were humanely sacrificed in the fasted state. The blood samples were centrifuged at 3500 rpm for 20 min to separate blood serum.

**Determination of biochemical parameters:** Total cholesterol, HDL-cholesterol, triglycerides, ALT, AST as well as total protein and albumin were assayed in the serum using commercial reagent kits (Randox Laboratories, UK). LDL-cholesterol was calculated by Friedewald *et al.* (1972) as follows:

$$\text{LDL} = \text{TC} - \text{HDL} - \frac{\text{TG}}{5.0}$$

**Statistical analysis:** Data was presented as means $\pm$ SD and analyzed using statistical software package (SPSS for Windows, version 18, IBM Corporation, NY, USA) using Tukey's-HSD multiple range *post-hoc* test. Values were considered significantly different at  $p < 0.05$ .

## RESULTS

The result on weekly FBG is presented in Fig. 1. At the first week of the study, alloxan induction significantly ( $p < 0.05$ ) increased FBG above normal in DC compared to NC and was maintained throughout the experimental period. This increase was significantly ( $p < 0.05$ ) prevented by extract treated animals in dose-dependent manner. DHD recorded the highest decrease of 57.55% which is comparable to DGL (60.33%) while, DLD have lowest decrease of 25.75% (Table 1). Administration of the extract to NAA group did not affect FBG.

Daily fluid and food intakes significantly ( $p < 0.05$ ) increased in DC compared to NC (Fig. 2). Treatment with ASE or glibenclimide to diabetic animals significantly ( $p < 0.05$ ) maintained fluid and food intakes close to that observed in NC. Similarly, NAA demonstrated no effect on fluid and food intakes. Moreover, DM caused significant ( $p < 0.05$ ) reduction in b.w. (24.12%) compared to NC which increases by 1.56% (Table 2). This decrease was completely prevented in all the extract treated groups. The improvement in b.w. did not differ significantly ( $p < 0.05$ ) within the diabetic treated groups. NAA showed significant ( $p < 0.05$ ) increase in b.w. compared to NC group.

Alloxan induction caused increase in serum total-cholesterol, triglycerides, LDL-cholesterol and decrease in serum HDL-cholesterol in diabetic animals (Fig. 3). However, treatment of the extract

Table 1: Effect of aqueous stem bark extracts of *A. albida* on FBG of normal and alloxan-induced diabetic rats

Post-treatment period	Fasting blood glucose (mg dL <sup>-1</sup> )						
	NC	NAA	DC	DLD	DMD	DHD	DGL
Week 1	98.45±4.11	95.55±3.11	363.25±4.13	382.55±7.25	377.11±8.34	384.45±8.34	391.56±6.24
Week 3	101.25±3.26	97.52±5.22	412.22±8.65	270.28±11.32	218.27±8.76	171.15±6.24	159.45±6.11
*Change in FBG (%)	2.42±1.05 <sup>a</sup>	2.55±2.77 <sup>a</sup>	15.35±4.25 <sup>c</sup>	-25.75±8.33 <sup>d</sup>	-44.15±10.25 <sup>e</sup>	-57.55±7.55 <sup>f</sup>	-60.33±10.33 <sup>g</sup>

All values are means±SD of five replicates. Values with different superscripts along a row are statistically different ( $p < 0.05$ ). \*Values represent differences between initial and terminal FBG, negative signs indicate decreases, ( $n = 5$ ), NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclamide

Table 2: Effect of aqueous stem bark extracts of *A. albida* on change in body weight of normal and alloxan-induced diabetic rats

Post-treatment period	Body weight (g)						
	NC	NAA	DC	DLD	DMD	DHD	DGL
Week 1	132.34±2.47	116.31±6.37	139.33±3.55	148.35±3.56	132.13±5.87	129.31±4.59	134.25±8.13
Week 3	134.41±3.89	141.33±4.96	103.10±7.09	152.33±3.85	137.33±9.19	134.11±4.88	156.25±6.66
*Change in body weight (%)	+1.56±0.61 <sup>a</sup>	+20.42±5.31 <sup>b</sup>	-24.12±3.20 <sup>c</sup>	+3.10±1.12 <sup>d</sup>	+2.88±1.33 <sup>d</sup>	+2.71±1.82 <sup>d</sup>	+15.45±2.09 <sup>e</sup>

All values are means±SD of five replicates. Values with different superscripts along a row are statistically different ( $p < 0.05$ ). \*Values represent differences between initial and terminal body weight, negative signs indicate decreases, ( $n = 5$ ), NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclamide

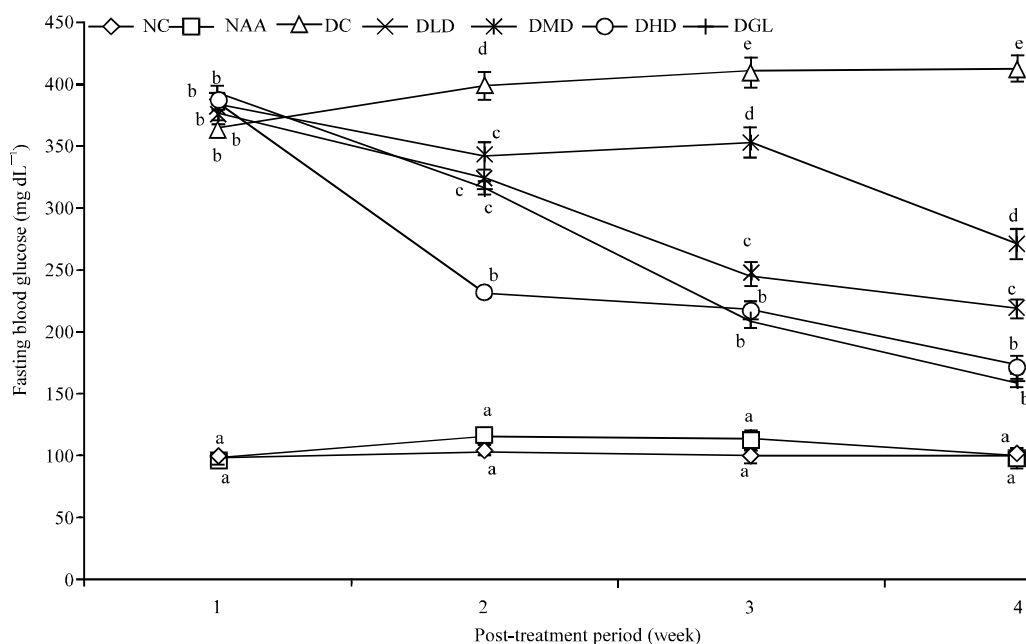


Fig. 1: Weekly FBG levels of different animal groups. Data are presented as the mean±SD of five animals. Values with different letters (a-e) for a given week are significantly different from each other (Tukey's-HSD multiple range *post hoc* test,  $p < 0.05$ ). NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclamide

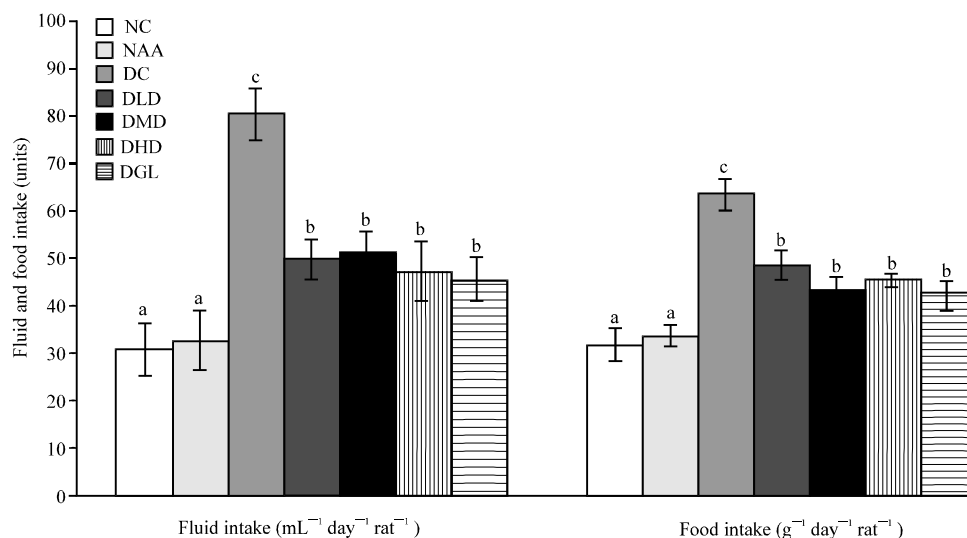


Fig. 2: Food and fluid intakes of the different groups during the experimental period. Data are presented as the mean $\pm$ SD of five animals. Values with different letters (a-e) for a given week are significantly different from each other (Tukey's-HSD multiple range *post hoc* test,  $p < 0.05$ ). NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclamide

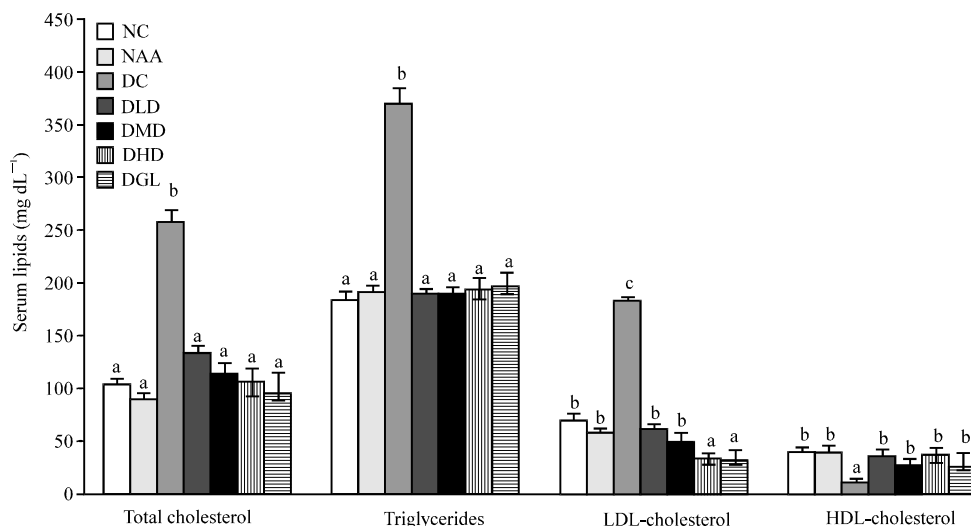


Fig. 3: Serum lipid profile in different animal groups at the end of the experimental period. Data are presented as the mean $\pm$ SD of eight animals. Values with different letters (a-e) for a given parameter are significantly different from each other (Tukey's-HSD multiple range *post hoc* test,  $p < 0.05$ ). NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclamide

attenuated these alterations in DLD, DMD and DHD which were comparable to that observed in DGL (Fig. 3). The levels of lipids profiles in DLD, DMD and DHD groups did not differ significantly ( $p < 0.05$ ) compared to NC (Fig. 3).

Table 3: Effect of aqueous stem bark extracts of *A. albida* on Packed Cell Volume (PCV) of normal and alloxan-induced diabetic rats

Post-treatment period	Packed cell volume (%)						
	NC	NAA	DC	DLD	DMD	DHD	DGL
Week 1	43.75±2.32	46.00±6.33	47.75±2.25	42.20±1.53	46.25±4.09	48.00±2.52	45.35±0.33
Week 3	62.25±4.94	44.00±2.51	29.50±3.50	30.10±1.32	38.35±1.55	39.30±4.91	51.30±0.88
*Change in PCV (%)	46.53±8.54 <sup>a</sup>	-5.83±4.43 <sup>b</sup>	-39.53±3.54 <sup>c</sup>	-21.70±4.99 <sup>d</sup>	-18.51±6.00 <sup>e</sup>	-16.01±8.65 <sup>f</sup>	12.41±1.36 <sup>g</sup>

All values are means±SD of five replicates. Values with different superscripts along a row are statistically different (p<0.05).

\*Values represent differences between initial and terminal PCV, negative signs indicate decreases, (n = 5), NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclimide

Table 4: Effect of aqueous stem bark extracts of *A. albida* on serum AST, ALT, total protein and albumin of normal and alloxan-induced diabetic rats

Parameters	NC	NAA	DC	DLD	DMD	DHD	DGL
AST (U L <sup>-1</sup> )	77.00±3.74 <sup>a</sup>	62.67±1.19 <sup>b</sup>	79.00±1.40 <sup>a</sup>	61.33±0.93 <sup>b</sup>	71.00±3.00 <sup>c</sup>	69.25±2.56 <sup>d</sup>	84.75±2.00 <sup>e</sup>
ALT (U L <sup>-1</sup> )	35.00±2.00 <sup>a</sup>	27.67±4.48 <sup>b</sup>	36.00±1.50 <sup>a</sup>	95.00±2.52 <sup>c</sup>	32.00±3.00 <sup>d</sup>	97.25±0.85 <sup>e</sup>	30.05±1.41 <sup>f</sup>
T. Proteins (g dL <sup>-1</sup> )	6.38±0.80 <sup>a</sup>	6.36±0.46 <sup>a</sup>	1.70±0.10 <sup>b</sup>	1.27±0.12 <sup>c</sup>	6.09±0.32 <sup>d</sup>	6.18±0.40 <sup>e</sup>	4.06±1.03 <sup>f</sup>
Albumin (g dL <sup>-1</sup> )	2.77±0.40 <sup>a</sup>	1.92±0.66 <sup>b</sup>	1.11±0.50 <sup>c</sup>	1.49±0.67 <sup>d</sup>	2.03±0.01 <sup>e</sup>	1.26±0.57 <sup>d</sup>	2.25±4.99 <sup>a</sup>

All values are means±SD of five replicates. Values with different superscripts along a row are statistically different (p<0.05), (n = 5), NC: Normal control, DC: Diabetic control, NAA: Normal treated *A. albida*, DLD, DMD and DHD: Diabetic treated lower, medium and higher doses, respectively, DGL: Diabetic treated glibenclimide

Administration of ASE to NAA caused a significant (p<0.05) decrease in PCV (5.83%) compared to NC. This effect was significantly (p<0.05) more pronounced in DC animals (39.53%). Treatment with ASE for three weeks significantly (p<0.05) and dose-dependently improved the levels of PCV in alloxanized rats. The effect was completely prevented in DGL group (Table 3). Similarly, serum levels of AST and ALT were not affected by DM. The diabetic treated animals showed a fluctuation in serum AST and ALT levels which were found to be significant compared to DC group. The levels in NAA decreased significantly compared to NC (Table 4). Similarly, serum total protein and albumin in DC were significantly (p<0.05) decreased compared to NC. This decrease was completely prevented by the extract treated groups which is comparable with DGL.

## DISCUSSION

In this study, we investigated for the first time, the anti-hyperglycemic and anti-hyperlipidemic effects of *A. albida* ASE in alloxan-induced diabetic rats. It is evident from the FBG of DC that diabetes was successfully induced and maintained throughout the study period. Administration of *A. albida* ASE or glibenclimide caused reduction on FBG in dose-dependent fashion. This might be attributed to one or a combination of the following; insulin secretory effect of the extract from the remnant  $\beta$ -cells, improve glucose uptake by peripheral tissues or slowed down glucose absorption in the gastrointestinal tract (GIT) or regulated the metabolism of glucose by the liver (Ibeh and Ezeaja, 2011). Our finding was in line with previous studies (Egunyomi *et al.*, 2011; El-Demerdash *et al.*, 2005).

The classical symptoms of DM include polydipsia, polyphagia and weight loss (American Diabetes Association, 2007) which clearly manifested in DC. Treatment of *A. albida* ASE or glibenclimide for three weeks improved both fluid and food intakes. Similarly, NAA maintained fluid and food intakes near to that observed in NC. The decrease in b.w. observed in DC could be attributed to increased physiological alterations caused by DM.

It is an established fact that dyslipidemia is among the major risk factor associated with uncontrolled DM contributing to secondary complications (Arvind *et al.*, 2002). Damage to  $\beta$ -cells by alloxan, apparently increases lipolysis and cholesterol biosynthesis due to reduced insulin levels (Abdel-Sattar *et al.*, 2011) which, in turn, result into hyperlipidemia. Significant decrease in LDL-cholesterol, triglycerides and total cholesterol with concomitant increase in HDL-cholesterol is highly required for a potential anti-diabetic agent. It is clearly evident from the present findings that, *A. albida* ASE at various doses administered improved lipid metabolism and could be regarded as potential anti-diabetic agents. This lipid lowering effect of the extract was comparable with DGL, indicating similar mechanistic pattern with glibenclimide, a known anti-diabetic drug. But this assumption is subjected to further scientific validation. The extract had no effect on lipid profiles of NAA throughout the study period which is in line with the previous study reported on other plants (Parveen and Siddiqui, 2011). The dose-dependent anti-hyperglycemic and anti-hyperlipidemic effects observed in this study could also be attributed to major active principles present in *A. albida* ASE as reported in previous studies (Alhaji *et al.*, 2014).

Furthermore, in consistent with previous study, administration of *A. albida* ASE (both hyperglycemic and normoglycemic animals) caused reduction on PCV levels (Oluwakanyinsola *et al.*, 2010). The effect was more pronounced in diabetic compared to normal treated animals. Treatment of ASE ameliorated this effect in dose-dependent manner. Similarly, it was also observed from the results, serum AST and ALT levels were not affected by diabetes and could possibly be due to short period of study. This was in line with previous study reported by Opajobi *et al.* (2011). So, the observed decrease in serum total protein and albumin in DC may also be linked to the net increase in protein breakdown rather than a decline in protein synthesis (Moller and Nair, 2008). Treatment with ASE attenuated these alterations as evidenced by increase in total protein and albumin levels which is in line with the previous studies (Salahuddin *et al.*, 2010).

Conclusively, the results of this study demonstrated that *A. albida* ASE possessed anti-hyperglycemia and anti-hyperlipidemia in alloxan-induced diabetic rats and hence, improved the carbohydrate, protein and lipid homeostasis. However, bioassay-guided fractionations to isolate active components are currently underway.

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