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Ethanoic Extract of Citrullus Lanatus Seeds Modulates Lipid Variables of Diabetes Induced Rats

Brown Holy[1], Alozie, Innocent Chinonyerem and Ben-Chioma, AdlineErima
[1]*Dept. of Medical Laboratory Science Rivers State University, Npkolu, Port Harcourt, Nigeria*
Corresponding Author:Brown Holy

Abstract:*Approaches to the management of diabetes mellitus is multifaceted. The role of some phytonutrients in the reduction of blood glucose levels is being recognized. Present study investigated the effect of ethanoic extract of Citrullus lanatus seeds on biochemical variables and some haematological indices. Seventy-seven (77) male wistar rats divided into eleven (11) groups of seven (7) rats each were used in this study. Diabetes mellitus was induced in the test animals in groups V – XI by streptozotocin (40mg/kg) after 2 weeks feeding on a High-fat diet (HFD). Group I was normal control rats. Groups II, III and IV were non-diabetic control rats given the vehicles 1% ethanol, corn oiland, High-fat diet (HFD) for 2 weeks respectively. Groups V was diabetic control rats. Groups VI and VII were diabetic control rats given the vehicles 1% ethanol and corn oil, respectively. Groups VIII – X were diabetic rats given 200 mg/kg, 400mg/kg and 600mg/kg, respectively, of ethanoic extract of Citrullus lanatus seeds (EECL) in corn oil. Group XI were diabetic rats given 0.5mg/kg Glibenclamide. Treatments were given by gavage daily for 28 days. The body weight of the rats and fasting plasma glucose levels were measured and recorded every week, At the end of the treatment period and following overnight fast, blood samples were collected via cardiac puncture for biochemical and haematological analyses. The mean body weights of groups IV – XI were significantly higher than those of groups I – III after two (2) weeks feeding on the HFD ($p<0.05$). Beginning from the 8^{th} day of EECL treatment to the end of the experiment, the mean body weights among the diabetic groups V – X were significantly lower compared to the non-diabetic groups I – IV. There was no statistical difference in the mean body weight of the glibenclamide-treated group (XI) compared to the non-diabetic groups ($P>0.05$). The mean plasma glucose levels taken at baseline were not significantly different ($p>0.05$) in all the groups. After 28 days of treatment with EECL, there were significant differences in the glucose levels of the 200mg/kg, 400mg/kg and 600mg/kg EECL-treated groups compared to the non-diabetic controls ($p<0.05$), whereas there was no significant difference in the plasma glucose levels of the glibenclamide-treated rats compared to the non-diabetic controls ($p>0.05$). Mean total cholesterol levels of the diabetic groups V – X were insignificantly higher compared to the non-diabetic control groups ($p>0.05$). The mean triglycerides of the diabetic groups V – X were significantly higher compared to the non-diabetic controls ($p<0.05$). The mean HDL-C levels of the diabetic groups V – VIII were significantly lower compared to the non-diabetic control groups ($p<0.05$). The HDL-C of the 400mg/kg and 600mg/kg EECL-treated groups were insignificantly lower compared to the non-diabetic control groups. The mean LDL-C levels of the diabetic groups V – X were significantly higher compared to the non-diabetic control groups ($p<0.05$). Consequently, serum triglycerides level was significantly elevated in the EECL-treated animals and did not improve the lipid variables of the study animals.*

Keywords:*Streptozotocin, diabetes, Citrullus lanatus, lipids, high- fat- diet*

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I. Introduction

Approaches to the management of diabetes mellitus include diet and lifestyle modifications, use of oral hypoglycaemic agents and insulin therapy to control blood glucose levels (Crook, 2012). The role of some phytonutrients in the reduction of blood glucose levels is being recognized. Muhammad et al, (2015) demonstrated significant reduction in blood glucose and cholesterol levels of diabetic rats treated with 200mg of ethanoic extract of Citrullus lanatus (watermelon) seed. Similarly, Deshmukh& Jain, (2015), reported both the hypoglycaemic and anti-lipidaemic effects at varying concentrations of ethanoic extract of Citrullus lanatusseed".

Current drug regimens in the treatment of diabetes mellitus places high financial burden on the subjects and they have to be taken throughout life, with some undesirable side effects This burden is even heavier in resource-poor countries. There is therefore need to search for safer, affordable and effective treatment options for diabetes mellitus".

"Several works have reported the anti-diabetic and anti-lipidaemic effects of Citrullus lanatus seed extracts in experimental rat models (Omigie&Agoreyo, 2014; Muhammad et al,2015; Deshmukh& Jain, 2015).

However, there is scarcity of data on the effect of ethanoic extract of Citrullus lanatus seeds in obesity-related diabetes mellitus. Metabolic disorders, increased production of inflammatory cytokines, induction of insulin resistance, obesity and arterial hypertension are promoted by long-term administration of diets containing 40% - 60% lipids (Barbosa-da-Silva et al, 2014). High-fat diets promote hyperglycemia and whole-body insulin resistance. It is generally accepted that high-fat diets can be used to generate a valid rodent model for the metabolic syndrome with insulin resistance and compromised β-cell function (Lingohr, et al, 2002; Buethner, et al, 2006). The concurrence of obesity, insulin resistance, hypertension and dyslipidemia is commonly referred to as the metabolic syndrome. Approximately 20–40% of the industrialized world are affected and its prevalence is expected to rise further (Buethner, et al, 2006)".

Present study considers the effect of ethanolic extract of Citrullus lanatusseeds on lipid indices of streptozotocin-induced diabetic rats fed on High-Fat Diet (HFD)".

II. Materials And Methods

2.1 Plant selection and authentication

Fresh watermelon fruits were obtained from Songai Farms, Rivers State. The plant species was authenticated by a certified botanist and kept in the herbarium (UPH/V/1214). The fruits were cut open and the seeds removed. The seeds were washed, air-dried and crushed to obtain the coarse powder.

2.2 Procurement of Animals

Seventy-seven (77) male wistar rats of five weeks old, weighing 90 – 108g were used for this study. They were housed in a well-ventilated stainless steel cage with soft wood shavings as bedding in the animal house. The rats were allowed to acclimatize for seven days with free access to water and laboratory chowed libitum. All the animals used in this work were treated in compliance with the guidelines of the National Institute of Health.

2.3 Preparation of extract

The coarse powder obtained from the crushed seeds of Citrullus lanatus were ground into powder. The plant chemicals were extracted with 80% ethanol for 48 hours in the dark, at 25^{o}C with intermittent shaking. The mixture was filtered first using a muslin cloth, then through Whatman No.1 filter paper. The filtrate was concentrated in a rotary evaporator for two days.

The concentrated extract was preserved in a desiccator for three days and later stored at 4^{o} C for studies. The concentrated extract was administered to the rats using corn oil as the vehicle (Brown, et al. 2000; Geng, et al. 2012). The volume of administration was calculated as follows:

Volume of administration = [weight of animal (in kg) × reference dose (mg/kg)] ÷ stock concentration (mg/ml).

2.4 Preparation of High Fat Diet (HFD)

The High Fat Diet was prepared using standard animal feed (73%), coconut oil (25%) and dietary cholesterol (egg yolk) (2%) (Gandhi, et al., 2013). After careful homogenization, the diet was fed to the test animals (except the normal control group) for two weeks.

2.5 Experimental Design

2.1 Experimental Induction of Diabetes Mellitus

Diabetes mellitus was induced in the test animals (groups V - XI) after two weeks of feeding with the high fat diet. The rats were weighed and fasting blood glucose samples (baseline measurement) were obtained from the retro-orbital sinus. A single dose intra-peritoneal injection of 40mg/kg of streptozotocin (STZ) (Gandhi, et al. 2013) was administered to the test animals. 5% glucose solution was given to the test animals orally for 24 hours in order to reduce mortality arising from the initial hypoglycaemia induced by streptozotocin (Desmukh& Jain, 2015). The rats were observed for 72 hours to allow for development of hyperglycaemia. The non-diabetic control rats (groups I - IV) received the vehicle (0.1M citrate buffer, pH 4.4) in a dose volume of 1ml/kg, intraperitoneally (Srinivasan, et al. 2005). Fasting blood glucose levels were measured using standard spectrophotometric glucose oxidase/peroxidase method before commencement of treatment. Rats with fasting blood glucose levels > 11.1 mmol/L were considered diabetic and included in the study. Weekly fasting blood glucose measurements were carried out until the end of the study.

2.5.2 Animal Groups and Treatment

The rats were assigned to 11 groups of 7 rats each (n=7). Group I (normal control rats: received standard rat feed and water daily); Group II (non-diabetic control rats: received standard rat feed, water and 1% ethanoldaily); Group III (non-diabetic control rats: received standard rat feed, water and corn oil daily); Group IV (non-diabetic control rats: given High-fat diet for 2 weeks, afterwards, standard rat feed and water daily); Group V (diabetic control rats: received standard rat feed and water daily); Group VI (diabetic control rats: receivedstandard rat feed, water and 1% ethanol daily); Group VII (diabetic control rats:received standard rat feed, water and corn oil daily); Group VIII (diabetic rats: received 200 mg/kg of ethanoic extract of Citrullus lanatus seeds, standard rat feed and water daily); Group IX (diabetic rats: received 400 mg/kg of ethanoic extract of Citrullus lanatus seeds, standard rat feed and waterdaily); Group X ((diabetic rats: received 600 mg/kg of ethanoic extract of Citrullus lanatus seeds, standard rat feed and waterdaily); Group XI (Standard Drug Group: received 0.5mg/kg Glibenclamide, standard rat feed and waterdaily). The selected doses were consistent with literature (Deshmukh& Jain, 2015). Treatment was given by gavage daily for 28 days. The body weight of the rats was measured and recorded every week, At the end of the treatment period and following overnight fast, blood samples were collected directlyfrom the heart of each rat for lipidanalysis.

III. Results

3.1 Weight (g) difference in non-diabetic and HFD-STZ induced

The mean body weights of the rats were similar in all the groups after the one (1) week period of acclimatization (table 4.1). The mean body weights of groups IV – XI were significantly higher than those of groups I – III after two (2) weeks feeding on the HFD ($p<0.05$). Beginning from the 8th day of EECL treatment to the end of the experiment, the mean body weights among the diabeticgroups V – X were significantly lower compared to the non-diabetic groups I – IV. There was no statistical difference in the mean body weight of glibenclamide-treated group (XI) compared to the non-diabetic groups ($P>0.05$).

Table 4.1: Weight (g) difference in non-diabetic and HFD-STZ induced diabetic rats**.**

GROUPS	Week 0	Week 1	Week 2 (HFD)	Week 3 (HFD)	Day 1 (Treatment)	Day 8 (Treatment)	Day 15 (Treatment)	Day 22 (Treatment)	Day 29 (Treatment)
Feed + Water	96.3±6.29^{ae}	110±5.35^{ae}	113±6.72^{a,e}	122±1.77^{a,e}	128±1.51^{a,e}	134±2.76^{a,e}	151±5.45^{a,e}	165±5.38^{a,e}	173±4.85^{a,e}
1% Ethanol	96.0±7.00^{bf}	110±3.76^{bf}	114±6.24^{b,f}	120±2.19^{b,f}	128±1.80^{b,f}	135±4.75^{b,f}	151±4.76^{b,f}	162±2.70^{b,f}	174±5.24^{b,f}
Corn Oil	98.6±3.60^{cg}	114±7.87^{cg}	116±6.53^{c,g}	118±2.77^{c,g}	129±1.90^{c,g}	137±3.21^{c,g}	152±3.91^{c,g}	166±2.41^{c,g}	173±2.93^{c,g}
HFD + Feed + Water	96.7±8.64^{dh}	112±6.18^{dh}	125±2.81^{a,bc}	133±3.80^{a,bc}	134±1.53^{a,b,c}	138±3.42^{d,h}	154±9.21^{d,h}	163±6.63^{d,h}	173±5.94^{d,h}
HFD + STZ	95.3±4.42	113±5.96	123±2.48^{a,bc}	133±2.14^{a,bc}	123±2.04^{e,f,g,h}	121±3.67^{e,f,g,h}	113±6.19^{e,f,g,h}	114±4.07^{e,f,g,h}	116±3.26^{e,f,g,h}
HFD + STZ + 1% Ethanol	98.1±4.41	114±3.99	123±2.08^{a,bc}	132±1.90^{a,bc}	123±3.02^{e,f,g,h}	119±2.51^{e,f,g,h}	120±6.65^{e,f,g,h}	119±3.16^{e,f,g,h}	123±2.44^{e,f,g,h}
HFD + STZ + Corn Oil	98.7±4.46	111±5.84	124±3.35^{a,bc}	132±3.90^{a,bc}	124±2.87^{e,f,g,h}	121±4.72^{e,f,g,h}	123±4.39^{e,f,g,h}	115±5.53^{e,f,g,h}	123±1.95^{e,f,g,h}
HFD + STZ + 200mg/kg EECL	97.9±3.80	113±4.31	124±1.38^{a,bc}	134±1.51^{a,bc}	123±2.97^{e,f,g,h}	119±4.15^{e,f,g,h}	116±4.47^{e,f,g,h}	112±3.55^{e,f,g,h}	120±3.10^{e,f,g,h}
HFD + STZ + 400mg/kg EECL	99.0±2.24	111±5.69	123±1.62^{a,bc}	133±2.31^{a,bc}	123±1.98^{e,f,g,h}	118±1.95^{e,f,g,h}	116±4.86^{e,f,g,h}	119±2.75^{e,f,g,h}	120±2.48^{e,f,g,h}
HFD + STZ + 600mg/kg EECL	98.9±3.39	113±3.86	126±1.11^{a,bc}	136±1.35^{a,bc}	124±2.30^{e,f,g,h}	123±2.18^{e,f,g,h}	123±2.54^{e,f,g,h}	119±2.61^{e,f,g,h}	131±4.54^{e,f,g,h}
HFD + STZ + 0.5mg/kg Glibenclamide	97.3±3.50	112±5.83	126±4.10^{a,bc}	133±6.50^{a,bc}	123±2.82^{e,f,g,h}	134±4.96	147±2.19	161±2.44	170±3.65

3.2 Fasting Blood Glucose levels (mmol/L) of non-diabetic and HFD-STZ induced diabetic rats.

The mean plasma glucose levels taken at baseline (prior to STZ treatment) were not significantly different ($p>0.05$) in all the groups (table 4.2). On the 1st day of EECL treatment, the mean plasma glucose of all the diabetic groups (V - XI) were significantly higher compared to the non-diabetic control groups ($p<0.05$). This pattern of results was unchanged by the 8th, 15th and 22nddays of EECL treatment with the exception of the glibenclamide-treated group (XI). Mean glucose levels of the glibenclamide-treated group (XI) had started to fall on the 8th day (from 18.29±5.81 mmol/L on day 1 to 10.49±1.56 mmol/L on day 8). By day 15 of EECL treatment the mean plasma glucose (8.33±1.35 mmol/L) of group XI had fallen to statistically insignificant level ($p>0.05$) when compared to the non-diabetic controls.After 28 days of treatment with EECL, the mean plasma glucose levels of the non-diabetic control groups (I - IV) were 5.08±0.25 mmol/L, 4.41±0.49 mmol/L, 5.05±0.66 mmol/L and 5.04±0.81 mmol/L, respectively. The mean plasma glucose levels of the diabetic control groups (V - VII) were 25.46±1.30 mmol/L, 20.04±2.69 mmol/L and 24.79±3.39 mmol/L, respectively. Mean plasma glucose levels of the EECL-treated groups (VIII - X) were 20.00±2.75 mmol/L, 12.71±0.68 mmol/L and 15.10±1.17 mmol/L for the 200mg/kg, 400mg/kg and 600mg/kg doses, respectively. Mean plasma glucose level of the glibenclamide-treated group (XI) was 7.07±0.41 mmol/L.There was no significant difference in the plasma levels of the glibenclamide-treated rats compared to the non-diabetic controls (table 4.3). There were

significant differences in the glucose levels of the EECL-treated groups at all the dose levels used compared to the non-diabetic controls ($p<0.05$).

Table 4.2 Fasting Blood Glucose levels (mmol/L) of non-diabetic and HFD-STZ induced diabetic rats

GROUPS	Week 3 (Baseline)	Day 1 (Treatment)	Day 8 (Treatment)	Day 15 (Treatment)	Day 22 (Treatment)	Day 29 (Treatment)
Feed + Water	4.92±0.37[a]	4.39±0.55[a]	4.27±0.76[a]	5.37±0.45[a]	4.40±0.79[a]	5.08±0.25[a]
1% Ethanol	4.99±0.13[b]	4.63±0.62[b]	4.18±0.74[b]	4.73±0.38[b]	4.15±0.53[b]	4.41±0.49[b]
Corn Oil	4.83±0.76[c]	5.09±0.35[c]	4.53±0.50[c]	5.31±0.46[c]	3.97±0.70[c]	5.05±0.66[c]
HFD + Feed + Water	5.17±0.47[d]	5.10±0.59[d]	4.83±0.81[d]	4.98±0.83[d]	4.84±0.79[d]	5.04±0.80[d]
HFD + STZ	4.95±0.61	14.43±2.02[a,b,c,d]	17.55±3.35[a,b,c,d]	16.39±2.12[a,b,c,d]	22.85±4.64[a,b,c,d]	25.46±1.30[a,b,c,d]
HFD + STZ + 1% Ethanol	5.11±0.33	17.71±2.90[a,b,c,d]	16.25±3.66[a,b,c,d]	19.34±2.33[a,b,c,d]	18.49±1.39[a,b,c,d]	20.04±2.69[a,b,c,d]
HFD + STZ + Corn Oil	5.43±0.44	18.26±2.73[a,b,c,d]	18.02±4.99[a,b,c,d]	21.47±3.62[a,b,c,d]	20.80±3.50[a,b,c,d]	24.79±3.39[a,b,c,d]
HFD + STZ + 200mg/kg EECL	5.44±0.25	18.19±4.43[a,b,c,d]	18.94±1.96[a,b,c,d]	20.61±2.72[a,b,c,d]	19.31±2.56[a,b,c,d]	20.00±2.75[a,b,c,d]
HFD + STZ + 400mg/kg EECL	4.80±0.64	21.19±5.75[a,b,c,d]	18.55±2.40[a,b,c,d]	20.07±1.82[a,b,c,d]	21.77±3.47[a,b,c,d]	12.71±0.68[a,b,c,d]
HFD + STZ + 600mg/kg EECL	5.26±0.34	21.08±4.83[a,b,c,d]	19.12±4.04[a,b,c,d]	18.67±1.99[a,b,c,d]	20.42±3.12[a,b,c,d]	15.10±1.17[a,b,c,d]
HFD + STZ + 0.5mg/kg Glibenclamide	5.29±0.29	18.29±5.81[a,b,c,d]	10.49±1.56	8.33±1.35	7.80±1.06	7.07±0.41

3.3 Fasting Lipid Profile of non-diabetic and HFD-STZ induced diabetic rats.

Mean total cholesterol levels of the diabetic groups V – X were insignificantly higher compared to the non-diabetic control groups ($p>0.05$). The mean total cholesterol levels of the 200mg/kg, 400mg/kg and 600mg/kg EECL-treated groups were similar to those of the diabetic controls. The 0.5mg/kg glibenclamide-treated group had total cholesterol levels similar to those of the non-diabetic control groups. Mean triglycerides of the diabetic groups V – X were significantly higher compared to the non-diabetic controls ($p<0.05$), while that of group XI was insignificantly higher ($p>0.05$).

The mean HDL-C levels of the diabetic groups V – VIII were significantly lower compared to the non-diabetic control groups ($p<0.05$). The HDL-C of the 400mg/kg and 600mg/kg EECL-treated groups were insignificantly lower when compared to the non-diabetic control groups.

The mean LDL-C levels of the diabetic groups V – X were significantly higher compared to the non-diabetic control groups ($p<0.05$). In addition, the mean LDL-C levels of the 400mg/kg and 600mg/kg EECL-treated groups, plus the 0.5mg/kg glibenclamide-treated group were significantly lower compared to the diabetic control groups.

Table 4.4: Fasting Lipid Profile of non-diabetic and HFD-STZ induced diabetic rats.

GROUPS	Total Cholesterol (mmol/L)	Triglycerides (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)
Feed + Water	1.67±0.18[a,e]	1.31±0.24[a,e]	0.56±0.18[a,e]	0.95±0.11[a,e]
1% Ethanol	1.72±0.04[b,f]	1.32±0.09[b,f]	0.53±0.26[b,f]	1.00±0.09[b,f]
Corn Oil	1.70±0.16[c,g]	1.28±0.20[c,g]	0.56±0.32[c,g]	1.01±0.09[c,g]
HFD + Feed + Water	1.70±0.05[d,h]	1.31±0.24[d,h]	0.54±0.32[d,h]	0.99±0.11[d,h]
HFD + STZ	1.79±0.04	2.27±0.10[a,b,c,d]	0.06±0.03[e,f,g,h]	1.41±0.12[a,b,c,d]
HFD + STZ + 1% Ethanol	1.80±0.03	1.91±0.14[a,b,c,d]	0.18±0.04[e,f,g,h]	1.39±0.04[a,b,c,d]
HFD + STZ + Corn Oil	1.82±0.04	1.95±0.55[a,b,c,d]	0.16±0.07[e,f,g,h]	1.42±0.07[a,b,c,d]
HFD + STZ + 200mg/kg EECL	1.80±0.05	1.83±0.13[a,b,c,d]	0.20±0.04[e,f,g,h]	1.31±0.06[a,b,c,d]
HFD + STZ + 400mg/kg EECL	1.77±0.04	1.92±0.47[a,b,c,d]	0.26±0.02	1.21±0.08[a,b,c,d]
HFD + STZ + 600mg/kg EECL	1.78±0.08	1.86±0.33[a,b,c,d]	0.27±0.03	1.20±0.06[a,b,c,d]
HFD + STZ + 0.5mg/kg Glibenclamide	1.69±0.14	1.57±0.11	0.42±0.16	1.06±0.05

Data represented as Mean ± SD; [a,b,c,d] = significantly higher compared to the non-diabetic control groups; [e,f,g,h] = significantly lower compared to the non-diabetic control groups.

IV. Discussion

After a one-week period of acclimatization, the mean body weight of the rats in all the groups were similar, However, this changed as the animals in groups IV–XI were placed on a High-Fat Diet (HFD) for two weeks. This significant increase ($p<0.05$) in the mean body weight of rats placed on HFD is consistent with literature (Levinet al. 1997; Farleyet al. 2003). On the other hand, following the administration of STZ (40 mg/kg) the mean body weight of rats in groups V – XI reduced significantly ($p<0.05$) when compared to the

non-diabetic control groups (I – IV), an effect that has been documented (Skovsø, 2014). The restoration of mean body weight in the glibenclamide-treated group followed the amelioration of hyperglycaemia. However, treatment with EECL at the dose levels used in this work did not bring about significant weight gain in the treated diabetic rats. This is probably due to the slow progress by the EECL in reversing hyperglycaemia in the treated animal groups.

Significant reduction in the mean plasma glucose levels of the EECL-treated rats at the three dose levels used was not demonstrated after 28 days of treatment. Meanwhile, mean glucose levels of the glibenclamide-treated group (XI) had started to fall on the 8^{th} day (from 18.29 ± 5.81 mmol/L on day 1 to 10.49 ± 1.56 mmol/L on day 8), returning to non-significant levels ($p>0.05$) after 28 days' treatment when compared to the non-diabetic controls. These findings are similar to those of Omigie&Agoreyo (2014) at the 200mg/kg dose level where they reported a mild reduction in blood glucose levels after 28 days' administration of ethanoic extract of C. lanatus seeds, but differ from the report of other workers (Deshmukh& Jain, 2015; Muhammad et al. 2015). However, these workers did not introduce High-fat diets before the administration of STZ. The non-significant reversal of hyperglycaemia in the EECL-treated groups appears to be related to the introduction of a High-fat diet prior to STZ treatment in this work, possibly because the effects of the HFD such as induction of insulin resistance may not have been easily overcome.

The mean total cholesterol levels among the diabetic control groups V – VII and were observed to be non-significantly higher compared to those of the non-diabetic control groups, EECL-treated groups and glibenclamide-treated group ($p>0.05$). This is related to the poor glycaemic control in these groups of animals which inevitably triggered lipolysis in other to generate metabolic energy. But the 400mg/kg, 600mg/kg EECL-treated groups and the 0.5mg/kg glibenclamide-treated group had total cholesterol levels similar to those of the non-diabetic control groups is possibly due to improved glucose utilization achieved in these groups of animals.
Mean triglyceride levels of the glibenclamide-treated group were insignificantly higher compared to the non-diabetic controls, while the diabetic control groups and the EECL-treated groups had significantly higher results. The 200mg/kg, 400mg/kg and 600mg/kg EECL dose levels did no produce significant reversal of hypertriglyceridaemia. This could be explained by the observed poor glycaemic control in these groups. But in spite of this, an elevation of HDL-C levels was produced by the 400mg/kg and 600mg/kg EECL dose levels compared to the diabetic controls. However, Deshmukh& Jain, 2015 and Muhammad et al. 2015 reported significant lowering of triglycerides and elevation of HDL-C at the 200mg/kg dose level. As earlier posited, the HFD given to the animals before STZ induction of diabetes could explain this deviation from their reports.
The mean LDL-C levels observed in diabetic groups V – X were significantly higher compared to those of the non-diabetic controls. These levels correspond to the higher mean triglyceride levels observed in this work. This finding is associated with the poor control of plasma glucose levels observed in these animal groups following HFD/STZ treatment. The lipid profile pattern observed in this work appears to be a reflection of the toxic events elicited by the HFD/STZ treatment of the animals resulting in hyperglycaemia and weight loss and the failure of the EECL treatment to effectively bring about normoglycaemia.

V. Conclusion

Contrary to previous submissions, this study did not observe the ethanoic extract of Citrullus lanatus (watermelon) seed improve lipid profile of rats fed with high fat diet and in streptozocin induced diabetics.

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