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Effect Of Aqueous Leaf Extract Of Nauclea Latifolia On Antioxidant And Lipid Biomarkers Of Potassium Bromate - Induced Albino Rats

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Abstract
The current study evaluated the effect of aqueous leaf extract of Nauclea latifolia on antioxidant and lipid biomarkers of potassium bromate induced albino rats. A total of twenty-five (25) apparently assumed healthy male Albino rats were procured, they were divided into five (5) groups and five (5) animals in each cage according to their weight. on the Effect of aqueous leaf extract of Nauclea latifolia on Lipid Biomarkers of Potassium bromate induced Albino rats from the result TC and HDL had a non-significant increase ($P>0.05$) in the extract treated groups when compared to the negative control group while TG decreased significantly ($P<0.05$) at the extract treated groups when compared to the negative control group. On the Effect of aqueous leaf extract of Nauclea latifolia on Serum Antioxidant of Potassium bromate induced Albino rats from the result GSH increased non significantly ($p>0.05$) as the treatment progressed while SOD had a significant increase ($p<0.05$) in groups 4 and 5 that received the extract when compared to the group that was not treated. Catalase also had a significant increase ($P<0.05$) in the treatment groups when compared to the negative control group. Thus, the leaf extract Nauclea latifolia showed a mild restoration in the lipid biomarkers of the animals induced with potassium bromated ($KBrO_3$) at the tested doses.

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I. Introduction

Medicinal Plants have been in use of man for prevention, treatment, and management of diseases for a very long time without even the knowledge of the component and toxicity of the plant (Shafaei *et al.,* 2011). Currently, phytotherapies represent an approximately $14 billion/year industry, which is about 5% of the current $280 billion/year market. Here, it is pertinent to mention that significant regional differences exist between developed and developing countries, where herbal products represent 25% and 80% of medications, respectively (Scott *et al.,* 1998). Among the 56% of currently prescribed synthetic drugs, 24% are derivatives from plant species, 9% are synthetic products modeled from natural products, 6% are extracted directly from the plant species, and 5% are of animal origin (Kushiro *et al.,* 2003). However, the vast repertoire of natural products remains to be tapped. The estimated total number of existing species is between 350,000 and 550,000, of which less than 20% have been investigated for medicinal potential (Wink *et al.,* 2000). Brazil, for example, has around 10% of the world's flora, where less than 1% of its plant species have been investigated for chemical and/or pharmacological properties (Palavan-Unsal*et al.,* 2009). The growing interest in herbal medicine demands information on the various plant preparations used in the treatment of diseases (Sarwar *et al.,* 2011). Scientific evaluation of medicinal plant is important to the discovery of novel drugs and also helps to assess toxicity risks associated with the use of herbal preparations and other conventional drugs of plant origin, One such plant leaf extract that is widely claimed in Traditional Medicine of Nigeria to be used in the management of several ailments such as malaria and enteric fever, diabetes mellitus, poison , gonorrhea and diarrhoea (Igoli*et al.,* 2002) without recourse to its safety or toxicity risks associated with the use of herbal preparations and other conventional drugs is *Nauclea latifolia.*

Nauclea latifolia, "Egbo egbesi" in Yoruba, "Ubuluinu" in Ibo and "Tabasiya" in Hausa is a Rubiaceae commonly known as pin cushion tree. It is a straggling shrub or small tree of about 10 ft high and is a native of the tropical Africa and Asia. The leaves are broadly elliptic to round ovate. It is found in areas like Abuja, Enugu, Akwa Ibom, Cross River, Kontangora, abia state and some other parts of Nigeria. It has found application in folk medicine for treatment of malaria, hypertension, diarrhea, tuberculosis, dysentery and also as a laxative (Okiemy-Andissa et al., 2004). Phytochemical analysis identifies indole-quinolizidine, alkaloids (glycoalkaloids) and saponins as the major components (Karou et al., 2011). Gidado et al. (2005) reported antidiabetic properties for the root and leaf extract while, Taiwe et al. (2010) reported anti-depressant and anti-anxiety effect for the root extract of the plant. A decoction of the stem in water has been demonstrated to exhibit a high antiparasitic potential (Benoit-Vical et al., 1998). The aqueous extract also showed effectiveness against chloroquine resistance strains of Plasmodium falciparum (Benoit-Vical et al., 1998). Hot aqueous and ethanolic extract was demonstrated to exhibit strong antibacterial property (Okiei et al., 2011). Alkaloid rich extract of *Nauclea latifolia* can react in vitro with mammalian DNA, leading to G2-M cell cycle arrest and heritable DNA-damage. In liver, kidney and blood cells, it induces single-strand breaks (Traore et al., 2000). However, despite the acclaimed and documented uses, there appears to be a paucity of information on the safety of repeated and prolonged use of this plant. This research data provides an insight into the antioxidant, anti-inflammatory, and antidiabetic potential of *Nauclea latifolia* leaf extracts as well as the relationship between these activities. The leaf is a rich source of natural antioxidant, anti-inflammatory, and antidiabetic compounds. It further suggests its relevance in the management of postprandial hyperglycaemia by suppressing the hydrolysis of dietary carbohydrates. The bioactive compounds responsible for this antioxidant, anti-inflammatory, and antidiabetic activities were not identified and thus, could be identified, isolated, and assessed. Also, in vivo confirmation of *Nauclea latifolia* leaf antidiabetic potential and mechanisms should be further evaluated.

Nauclea latifolia is used in the treatment of several diseases (Okwori et al., 2008). The stem bark is used for the management of toothache, dental caries, septic mouth, malaria, diarrhea, and dysentery (Nworgu et al., 2008, Okiei et al., 2011). The leaf decoction is used as a remedy for diabetes mellitus in Northern Nigeria while the bark extract has been reported effective in the treatment of wounds, coughs, and gonorrhoea (Gidado et al., 2005, Madibunyi, 1995). Akubue and Mittal (1982) reported the use of the roots for the treatment of hypertension. N. latifolia extracts demonstrated antimicrobial properties against Klebsiella pneumoniae (Tona et al., 1999) and several other organisms like Bacillus subtilis, Escherichia coli, Salmonella enteritidis, Pseudomonas aeruginosa, (Omer et al., 1998, Hussain and Deeni, 1991). The fruit extract exhibited activity against Human Immunodeficiency Virus (Hussein et al., 1999). Nauclefine and naucletine were reported as alkaloids from the plant (Karou et al., 2011). Nauclefolinine and five known triterpenoids were also reported from the roots of Nauclea latifolia (Ngnokam et al., 2003). The plant extracts are used in the management of infectious and oxidative stress related diseases (Gidado et al., 2005, Okiei et al., 2011). Some of the activities exhibited by Nauclea latifolia extracts suggest the presence of antioxidant and antimicrobial components in the extracts. In other to validate this, we have investigated Nauclea latifolia leaf extracts for its *antioxidant* and antimicrobial secondary metabolites. The inhibiting or preventive effects of herbs or spices against the deleterious consequences of oxidative stress are due to the presence of natural antioxidants in them. Drug

formulations that are antioxidant based are used in the prevention and treatment of complex diseases which include atherosclerosis, stroke, diabetes, Alzheimer's disease, and cancer.

Diabetes mellitus (DM) is a noncommunicable, chronic ailment that is not only affecting a high proportion of the world's populace but also affecting more of the developing countries of the world compared to the developed nations [Ogbera A. O.and C. Ekpebegh et al., 2014,]. A worldwide survey by International Diabetes Foundation (IDF) showed a diagnosis of 415 million people with diabetes, with a projected increase to over 600 million people by 2040 [Boles, R. Kandimalla and P. H. Reddy et al., 2017,]. Epidemiological statistics show that Nigeria is responsible for a fifth of all reported cases of diabetes in the sub-Saharan Africa, with a steep increase in the prevalence of this disease from the rural area to members of the high socioeconomic population [O. A. Fasanmade et al., 2015,]. Nauclea latifolia is an evergreen tree with multiple stems and adapts very well in both the tropical rainforest zone and the savanna woodlands situated in the west and central Africa [Z. A. M. Nworgu.,2008,]. This tree is known to have various medical uses, particularly by folk medicine men [A.Gidado et al., 2008,]. It can also serve as a chewing stick to treat stomachache and tuberculosis at its initial stage [A. Gidado, D. A. Ameh et al., 2005,]. Reports show that *Nauclea latifolia* formulation and decoction preparations are used in ethnomedicine to treat hyperglycaemia and diabetes by different ethnic groups in Nigeria [U.F. EzuruikeandJ. M.Prieto et al., 2014,] activity of *Nauclea latifolia* leaf extract on α-glucosidase and αamylase, and the mechanisms are yet to be experimentally documented. The association between the inhibition of these enzymes and its reported antioxidant activities has also not been examined. These observations prompted this study to assess the antidiabetic mechanisms of *Nauclea latifolia* leaf extracts on enzymes linked to diabetes mellitus. The aim of this study was to screen for the antioxidant and lipid biomarkers of aqueous leaf extract of Nauclea latifolia on potassium bromated ($KBrO_3$) induced rats.

II. Materials And Methods

Plantcollection

The leaves of ***Nauclea latifolia*** was harvested from a compound bush in Abia State University, Uturu, Abia State, Nigeria. The plant was authenticated at the department of Plant Science and Biotechnology, Abia State University, Uturu, by a taxonomist, and voucher samples deposited in the departmental herbarium.

Plant preparation and extraction

The extract was prepared using the method described by Daniel *et al*., (2012) with slight modifications. The dried leaves were milled into a fine powder and stored in a cellophane bag until use. The leaves were air-dried for four weeks into a constant weight.

Preparation of stock (plant extract) solution

One hundred grams (100g) of powdered leaves of ***Nauclea latifolia*** was soaked in 1000ml of water for 24 hours with stirring for proper mixing and drained using a muslin cloth. It was filtered using Whatman no. 1 filter paper in other to get a clear filtrate.

Animal Model

In this study, a total of fifteen (25) apparently assumed healthy male Albino rats (9-10weeks old) weighing between (120 - 140 g) was procured from the animal house, Department of Physiology, College of Medicine, University of Nigeria Nsukka. The rats were kept in the animal house at the Department of Biochemistry, Abia State University, Uturu. The animals were allowed to acclimatize for 14 days under standard laboratory condition prior to the commencement of this investigation with free access to commercial feed (Vita feed Nig. Ltd.) and clean drinking water *ad libitum*. Ethical principles of World Health Organization of good laboratory practice regulations of 1998 and United States guidelines for experimental animals (Care Animal Use Committee, 1998) was strictly adhered to throughout the study.

Sacrifice of Animals.

The animals were sacrificed 24 hours after extract was induced on the fourteenth day. All animals were weighed carefully and correctly before sacrifice. Cotton wool was soaked in chloroform and placed in a Bel-ArtTM SP Scienceware^{TM} Lab Companion Vacuum Desiccator (AS23Jencon Hemel, Eng). Each rat was transferred from the cage to the desiccator in turns. After the rat had been properly anaesthetized, the heart was punctured and blood samples from each animal were collected into heparinized bottled.

Biochemical Analysis

HDL, VLDL, TC and TG were assayed according to manufacturer's instruction using Randox Diagnostic kits (Randox Laboratories Ltd., Crumlin, UK) as described by Reitman and Frankel (1957) and Tietz et al., (1983).

Statistical analysis

Results were expressed as mean ± SD (standard deviation). Statistical analysis was performed by One-way analysis of variance (ANOVA) with the ANALYSTAT^{TM} Statistics software package, version 1.6.50. One-way ANOVA with a Turkey test post-hoc was used to identify statistical differences among groups. A *p*-value of <0.05 was considered statistically significant.

Experimental Design

Randomized complete block experimental design recommended by Ogbeibu, (2005) was used in the study. The experimental animals were divided randomly into five (5) groups (replicates) of six (6) animals each: ***group***I: normal control, ***group II:*** hepatotoxic group (negative control), g***roup III:*** hepatotoxic group treated orally with 25mg/ml Vitamin C: a standard hepatoprotective drug, g***roup IV:*** Hepatotoxic group treated orally with 400mg/kg body weight of aqueous leaf extract of ***Nauclea latifolia and group V:*** Hepatotoxic group treated orally with 800mg/kg body weight of aqueous leaf extract of ***Nauclea latifolia.***

Composition of rat feed

The commercial rat feed purchased from Okigwe in Imo State is made up of the following constituents as shown in the table below.

Composition of rat feed

Parameter	Value
Crude protein	16.00%
Calcium	1.0%
Crude fibre	7.0%
Fat/oil	5.0%
Phosphorus	0.4%
Lysine	0.75%
Methionine	0.36%
Salt (min)	0.30%
Energy	2450Kcalkg

Chemicals and reagents

Chemicals used in this work were of analytical grade and products of Sigma Chemical Company (Mayer and Baker Ltd, Fluker and BDH). Some of the chemicals used were manufactured by Randox and TECO Diagnostics, USA. Reagents include: Wagners solution, ferric chloride, sodium hydroxide (NaOH), ethanolic acetic acid, ammonium hydroxide, potassium ferrocyanide (K_2Fe (CN) $_6$), hydrogen tetraoxosulphate (vi) acid (H_2SO_4), methanol, ethanol, diethylether, butanol, sodium chloride (NaCl) and tannic acid.

Preparation of drugs

Vitamin C 100mg solution was prepared by crushing the tablet in a glass mortar and was dissolved in 40ml of distilled water to give 25mg/ml stock solution. Vitamin C was orally administered to the animals.

Determination of superoxide dismutase activity (SOD)

The method of Sun and Sigma as described by Ogbunugafor*et al*., (2010) was adopted. The reaction mixture (3ml) contained 2.95ml sodium carbonate buffer (0.05M, ph. 10.2), 0.02ml of serum and 0.03ml of epinephrine in 0.005N HCL used to initiate the reaction. The reference cuvette contained 2.95ml buffer, 0.03ml of substrate (epinephrine) and 0.02ml of water. An extinction coefficient for epinephrine at 480nm of 4020 $m^{-1}cm^{-1}$ was used in calculating activity.

Determination of catalase activity

The catalase activity was determined according to the method of Sinha (1972). This method is based on the fact that dichromate in solution with acetic acid is reduced to chromic acetate when heated in the presence of hydrogen peroxide via the formation of per chromic acid which is an unstable intermediate. Chromic acetate produced is measured calorimetrically at 610 to 750nm. The presence of dichromate in the mixture does not affect the colorimetric determination of chromic acetate, because dichromate does not absorb in the 570 to 610nm region. The catalase preparation is allowed to split hydrogen peroxide for different periods of time. The reaction is stopped at different time intervals by the addition of dichromate/acetic acid mixture; and the remaining hydrogen peroxide (H_2O_2) is determined by measuring chromic acetate calorimetrically.

Reagents

5% potassium bromate ($KBrO_3$): $KBrO_3$ (5g) was dissolved in distilled water and made up to 100ml.

0.2M H_2O_2: This was prepared by adding 22.68ml of 30% H_2O_2 (8.82M) to distilled water and made up to 100ml.

Dichromate/acetic acid: This was prepared by mixing 5% solution of potassium dichromate with glacial acetic acid in a 1:3 volume ratio.

0.1M phosphate buffer, pH 7.0: Disodium hydrogen tetraoxosulphate (v) dehydrate ($Na_2HPO_4.2H_2O$) (3.58g) and 1.19g of sodium dihydrogentetraoxosulphate (v) dehydrate was dissolved in distilled water and made up to 100ml with distilled water. The pH was adjusted to 7.0.

Procedure

Hydrogen peroxide solution (4ml of 0.2M) was added to 5ml of phosphate buffer (0.01M of pH 7.4). The serum (1ml) was added to the hydrogen peroxide or buffer mixture and the mixture was gently mixed at room temperature. 1ml portion of the reaction mixture was withdrawn and dispensed into 2ml dichromate or acetic acid reagent at 1minute interval and the steady absorbance reading taken at 570nm.

Calculation

The monomolecular velocity constant K for the decomposition of hydrogen peroxide by catalase was determined by using the equation for the first-order reaction $K= (1/t) (\log S_o/S)$. Where S_o is the initial H_2O_2 concentration and S is the concentration of hydrogen peroxide at a particular time interval given as t (minutes). The values of K are plotted against t and the velocity constant of catalase K[o] at 0minute determined by extrapolation (that is the interval on the vertical axis). The catalase contents of the samples were expressed in terms of: Kat.f. =K[o]/mg protein (per ml).

Determination of Malondialdehyde (MDA) level

Lipid peroxidation was ascertained by formation of Malondialdehyde (MDA) and measured by thiobarbituric reactive (TBARS) method previously described by Onkawa*et al*., (1979). Reaction mixture containing serum (0.5ml), TCA (0.5ml) and TBA (0.5ml) was incubated in boiling water for 15minutes. The pink color of chromogen formed was extracted in butanol solution (2.0ml). The mixture was centrifuged at 3000 rpm for 10minutes, and the supernatant was read at 532nm.

Determination of Glutathione (GSH)

The GSH level was determined using the method described by Ellman (1959) with slight modifications. Serum (0.5ml) was added to 2ml of 5% TCA and centrifuged at 3000 rpm for 10minutes. The supernatant (1ml) was added to 0.5ml of DTNB (10nm) in the presence of 3ml phosphate buffer (0.1M, pH 7.4). Absorbance was read at 420nm.

Determination of Serum Total Cholesterol

Serum cholesterol was estimated by the cholesterol oxidase peroxidase method of Schehler and Nussel (1975). The reagent kit was manufactured by Agape Diagnostics, Switzerland. Cholesterol is measured enzymatically in serum or plasma in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol. One of the reaction byproducts, H_2O_2 is measured quantitatively in a peroxidase catalyzed reaction that produces a color. Absorbance is measured at 500 nm. The color intensity is proportional to cholesterol concentration.

Clean test tubes were placed in a rack and labelled blank, standard, test and control respectively. One milliliter (1 *ml*) of cholesterol working reagent was dispensed into each tube. Ten microlitres (10ul) each of standard, test and control reagent was dispensed into the respective tubes. The contents were mixed and incubated for 5minutes at 37˚C. The absorbance of each tube was read at 540nm against a reagent blank.

Calculation

$$\text{Cholesterol conc. (mg/dl)} = \frac{\text{Absorbance of test or control}}{\text{Absorbance of standard}} \times 200$$

Determination of Triglyceride

Serum triglyceride was estimated by the glycerol -3- phosphate oxidase-TOPS method of Schehler and Nussel (1975). The reagent kit was made by Agape Diagnostics, Switzerland.

Clean test tubes were placed in a rack and labelled blank, standard, test and control respectively. One milliliter (1*ml*) of triacylglycerol reagent was dispensed into each tube. Ten microliters (10ul) of standard, test and control reagents were dispensed into each tube mixed and incubated for 5 minutes at 37^0C. Change in absorbance was measured at 546nm against a reagent blank for each sample.

Calculation

Triacylglycerol conc. (mg/dl) = $\frac{\text{Absorbance of test}}{} \times 200$

HDL-Cholesterol

Serum HDL–Cholesterol was estimated by the precipitation method according to Assmann (1979). The reagent kit was made by Agape Diagnostics, Switzerland.

Principle

The chylomicrons, very-low-density lipoproteins (VLDL)of serum is precipitated by phosphotungstic acid and magnesium ions. After centrifugation, high-density lipoproteins (HDL) are in the supernatant. The HDL content of supernatant is measured by an enzymatic method.

Procedure

Precipitation

Clean test tube was placed in a rack and 30uL of HDL reagent and 30uL of test sample added to the tube. The content was mixed, well, allowed to stand for 10 minutes at room temperature, mixed again and centrifuged for 10 minutes at 4000 rpm. After centrifugation, the clear supernatant was separated from the precipitate within one hour using a Pasteur pipette. HDL cholesterol concentration was determined using cholesterol reagent.

HDL cholesterol determination

Three clean test tubes were placed in a rack and labelled blank, standard, test and control respectively. One thousand microliters (1000uL) of cholesterol reagent was dispensed into each tube. Fifty mircoliters (50uL) of HDL standard and the HDL supernatant were dispensed into standard and test tubes respectively. The contents were mixed and incubated for 5 minutes at 37^{o}C. The absorbances of the standard and test samples were measured at 630nm against a reagent blank.

Calculation

HDL cholesterol conc. in (mg/dl) = $\frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times N \times 2$

Where 2 = dilution factor of the sample

N= concentration of standard (50mg/dl)

LDL cholesterol conc. in mg/dl = Total cholesterol - (HDL cholesterol + $\frac{\text{triglycerides}}{5}$)

Determination of VLDL- Cholesterol

Very low density lipoprotein was estimated by dividing the values of triglyceride concentration by 5.

Statistical analysis

Results were expressed as mean ± SD (standard deviation). Statistical analysis was performed by One-way analysis of variance (ANOVA) with the ANALYSTAT^{TM} Statistics software package, version 1.6.50. One-way ANOVA with a Turkey test post-hoc was used to identify statistical differences among groups. A *p*-value of <0.05 was considered statistically significant.

III. Results

Table 1: Effect of aqueous leaf extract *of Nauclea latifolia* on Lipid Biomarkers of Potassium bromate induced Albino rats.

Treatment	TC (mg/dL)	HDL(mg/dL)	TG (mg/dL)
Group 1 (NC)	100.53±2.67^{a}	51.90±1.40a	114.55±4.24^{a}
Group 2 (-ve Control)	94.15±6.90^{a}	42.98±2.74^{b}	133.60±4.86^{b}
Group 3 (Strd. Drug)	100.58±0.85^{a}	52.10±2.44^{c}	126.85±4.20^{c}
Group 4 (Extract at 400mg/kg b.w)	100.10±4.10^{a}	54.10±1.01^{a}	120.75±1.51^{a}
Group 5 (Extract at 800mg/kg b.w)	97.00±2.77^{a}	55.88±3.14^{a}	122.88±2.64^{d}

Values are mean ± SD for N=5 (number of animals per group). Values down the column bearing the same letter of alphabets are not significantly different (P>0.05) while values with different subscript are significant at P<0.05.TC: Total Cholesterol HDL: High density lipoproteins TG: Triglyceride B.W: Body weight.

From the table above TC and HDL had a non-significant increase (P>0.05) in the extract treated groups when compared to the negative control group while TG decreased significantly (P<0.05) at the extract treated groups when compared to the negative control group.

Table 2: Table 1: Effect of aqueous leaf extract *of Nauclea latifolia* on Serum Antioxidant of Potassium bromate induced Albino rats.

Treatments	GSH (µ/l)	CAT (µ/l)	SOD (µ/l)	MDA (mmol/l)
Group1 NC	68.68 ± 3.09^{a}	27.9 ± 1.72^{a}	36.58 ± 2.99^{a}	0.29 ± 0.03^{a}
Group 2 -ve Control	49.23 ± 2.50^{b}	21.3 ± 1.12^{b}	31.10 ± 2.02^{b}	1.94 ± 0.08^{a}
Group 3 Std. Drug	56.6 ± 2.35^{a}	25.25 ± 0.67^{c}	32.30 ± 1.82^{c}	1.21 ± 0.21^{a}
Group 4 (extract at 400mg/kg b.w)	57.85 ± 2.08^{a}	26.87 ± 1.02^{d}	36.00 ± 1.60^{d}	1.05 ± 0.08^{a}
Group 5 (extract at 800mg/kg b.w)	58.75 ± 1.35^{a}	27.92 ± 0.49^{e}	34.22 ± 1.88^{e}	1.00 ± 0.07^{a}

Values are mean ± SD for N=5 (number of animals per group). Values down the column bearing the same letter of alphabets are not significantly different (P>0.05) while values with different subscript are significant at P<0.05. GSH: Glutathione SOD: Superoxide dismutase CAT: Catalase MDA: Malondealdehyde NC: Normal control B.W.: Body weight -ve: Negative control Std: Standard drug.

From the table above GSH increased non significantly (p>0.05) as the treatment progressed while SOD had a significant increase (p<0.05) in groups 4 and 5 that received the extract when compared to the group that was not treated. Catalase also had a significant increase (P<0.05) in the treatment groups when compared to the negative control group.

IV. Discussion

The deleterious effects of oxidants and lipid peroxidation can be countered by an organized antioxidant defense mechanism in humans. This mechanism involved several enzymatic and non-enzymatic processes that protect cells against oxidative damage. The major enzymatic antioxidant systems include superoxide dismutase, glutathione Peroxidase and catalase. The non-enzymatic antioxidants include glutathione and dietary constituents such as vitamins, flavonoids and carotenoids, which protect cells from the damaging effect of the radicals and lipid peroxidation (Rim *et al*., 1993). From the table 2 above, GSH increased non significantly (p>0.05) as the treatment progressed while SOD had a significant increase (p<0.05) in groups 4 and 5 that received the extract when compared to the group that was not treated. Catalase also had a significant increase (P<0.05) in the treatment groups when compared to the negative control group. This suggests that catalase plays important role in ameliorating the damaging effects of hydrogen peroxide produced in several cellular processes such as photorespiration, oxidation, DNA synthesis (Haenen, 1989). Malondealdehyde activity in the extract treated groups when compared to group 2 (hepatotoxic group) in this study suggests that the decreased levels of MDA (marker of lipid peroxidation) in hepatotoxic rats clearly showed that hepatotoxic rats were exposed to an increased oxidative stress via lipid peroxidation and had mild restoration in the extract treated groups, (Ayinla *et al*., 2015).

Lipids and lipoproteins over the years have been risk factors for several disorders including cardiovascular disease (CVD). Additionally, it has been demonstrated that high levels of serum total cholesterol (TC), triglycerides (TG), LDL cholesterol, very-low-density lipoprotein (VLDL), low concentration of HDL cholesterol, and increased body mass index (BMI) are significantly associated with cardiovascular diseases (CVD). (Rizvi and Nagra, 2014). From the table above TC and HDL had a non-significant increase (P>0.05) in the extract treated groups when compared to the negative control group while TG decreased significantly (P<0.05) at the extract treated groups when compared to the negative control group. This is supported by a work carried out by Rizvi and Nagara, (2014) that a reduction in these lipid biomarkers helps to reduce CVD.

In conclusion, this study demonstrated the biochemical effect of aqueous leaf extract of *Nauclea latifolia* in counteracting the Potassium Bromate induced toxicity in albino rats. The results suggest that aqueous leaf extract of *Nauclea latifolia* offers positive effect against Potassium Bromate induced toxicity in albino rats. The administration of the extract shows that TC and HDL had a non-significant increase (P>0.05) in the extract treated groups when compared to the negative control group while TG decreased significantly (P<0.05) at the extract treated groups when compared to the negative control group. Thus, aqueous leaf extract of*Nauclea latifolia* can be taken when a toxic substance is taken into the biological system to prevent damage of some organs in the body.