



## HEPATO-CURATIVE EFFECTS OF CRUDE METHANOL AND ETHANOL ROOT EXTRACTS OF *Calotropis procera* (SODOM OF APPLE) ON CCl<sub>4</sub> INDUCED HEPATO-TOXICITY IN ALBINO RATS

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### Abstract:

*The effects of methanol, ethanol root extract of Calotropis procera (MRECP, ERECP) and livolin on liver function indices of CCl<sub>4</sub> induced hepatotoxicity rats model was evaluated. Fifty (50) albino rats were grouped into Five (I, II, III, IV and V) of 10 rats each, 120mg/kg body weight CCl<sub>4</sub> diluted with olive oil in the ratio 1:1 was administered to rats in groups II, III, IV and V intramuscularly followed by oral administration of 10mg/kg livolin, 10mg/kg methanol and ethanol root extract of C. Procera to group III, IV and V respectively. Groups I and II serves as positive and test control respectively. Analysis of variance (ANOVA) for multiple comparison test were used to compare the indices of the liver and kidney functions for the test and control group at 10 days interval for 20 days. The hepatic biochemical markers Alanine Amino Transferase (ALT), Aspartate Amino Transferase (AST), Alkaline Phosphatases (ALP) of group Gp II were significantly higher (P<0.001) compared to others except GpI, while group III (treated with livolin) was statistically decreased (P<0.05) when compared with control (Gp I), this confirms the toxicity and treatment with livolin respectively. Oral administrations of the MRECP lowered all the liver function markers and increased the concentration of urea and albumin after 20 days of exposure. This indicates that MRECP may reverse the chemically induced tissue damage; in contrast, ERECP produced toxicity at both exposures as evidenced from the histopathology of the liver hepatocytes. The histopathological analysis of MRECP indicates improved fine architecture of the liver and kidney cells which are comparable to livolin treated group. In conclusion, the overall results suggest that ethanol root extracts of C. Procera may have moderate hepatorenal curative effects when compared to methanol extracts.*

**Keywords;** *Calotropis Procera, Hepatocurative, antihepatotoxic, hepatotoxicity*

### INTRODUCTION

The use of Traditional medicine in developed as well as developing countries as basis for the treatment of many ailments has been in existence for thousands of years and there is no doubt that their importance has been widely acknowledged. Medicinal plants have continued to play vital roles in the Nigerian healthcare sector, although traditional medical practitioners have not been fully recognized (Emmanuel *et al.*, 2015).

The search for hepatocurative agents that may cure and manage the conditions with high potency dates back to millennia. Various substances of animal and plant origin have been used in folk medicine of different cultures as hepatocurative remedies, some of which have been identified pharmacologically to exert their effects either on the hepatocytes or renal tissues or both (Heinrich *et al.*, 2004) Furthermore, ancient literature alluded to the use of numerous plants/preparations including *C. Procera* root to treat many diseases including liver and kidney damages without any scientific evidence. To understand the scientific reasons behind these folk claims, this work investigated the effects of organic solvent (methanol and ethanol) extract of *C. Procera* root in this study.

*Calotropis procera* belongs to the family Asclepiaceae (milkweed family) of the Genus *Calotropis* R. Br. (*Calotropis*). *Calotropis procera* or Giant milkweed is also known as sodom apple, calotrope, French cotton,

small crown flower (English), Tumfafiya (Hausa), Epuko (Nupe), Common names; auricular tree, dead sea apple, swallow-wort, apple-of-sodom, giant-milk weed, madarmudar, ruberbush, small crownflower, sodom's milkweed algodón de seda, bomba (Spanish), cotton-france, arbre de soie, and bois canon (French), Latin name. *Calotropis procera* (Ait) Ait (Aliyu, 2006). Liver is the very important part of our body responsible for the maximum metabolic and secretory activities and therefore appears to be a sensitive target site for substances modulating biotransformation. Liver is also associated in detoxification from the exogenous and endogenous challenges like xenobiotics, drugs, viral infections and chronic alcoholism. The period and intensity of the pharmacological response to drugs is influenced by their metabolic rate and hence substances capable to modify drug metabolism would be able to change the result of drug therapy. During all such exposures to the above mentioned challenges, if the usual defensive mechanisms of the liver are overpowered, the effect is liver damage. Liver injury or liver dysfunction is a major health problem that challenges not only medical professionals but also the pharmaceutical company and drug regulatory authorities.

Liver cell injury caused by various toxic chemicals like certain antibiotics, chemotherapeutic agents, carbon tetrachloride, thioacetamide, excessive alcohol consumption and microbes. Herbal medicines have been applied for the treatment of liver disorder for a lengthy period. Many herbal preparations are available in the market and therefore present review is aimed to compile the data on promising phytochemicals from medicinal plants that have been tested in hepatotoxicity models using modern scientific system (Amitet *et al.*, 2014). Although aqueous root extract of *C. Procera* has been used for the treatment of different ailments, over the years little or no information on the use of the root extract for prevention or treatment of hepatotoxicity is available. The aim of this study therefore, is to assess the hepatocurative effects of methanol and ethanol root extract of *Calotropis procera* on CCl<sub>4</sub> induced hepatotoxicity in albino rats.

## MATERIAL AND METHODS

### Plant materials

Root of *C. procera* was collected from Kanya Babba, Babura local government, of Jigawa State. Specimens of the leaves and bark were removed. The root was dug using hoe and a shovel. The root of *Calotropis procera* was allowed to dry under the shade, it was then ground using mortar and pestle. The extract of the plant root was prepared by weighing 200 g and soaking of the root powder in methanol and ethanol solvents separately (BDH) for 2 weeks.

### Acute toxicity test in albino rats:

Acute toxicity tests of methanol and ethanol extract of *C. procera* roots were performed separately in male and female rats according to OECD guideline for chemicals tests (OECD, 2001). The limit test at dose level of 2000 mg/kg body weight was administered orally (gavage) to six fasted males and females rats per extract. The females were nulliparous and non-pregnant. The animals of different groups were individually observed for 120 min post-treatment and at least once daily for 14 days for mortality and signs of toxicity such as changes in skin and fur, eyes, mucus membranes, convulsion, salivation, diarrhea, lethargy, sleep and coma.

### Experimental animals

Based on Lethal Dose 50 (LD50) values obtained from acute toxicity studies, the selection of dose for sub-chronic toxicity was carried out. The dose selected in this study is 10 mg/kg body weight. This dose corresponded at 1/100 of LD50 obtained in the acute toxicity tests. Fifty (50) male and female albino rats obtained from the physiology Department, faculty of Basic Medical Science, Bayero University, Kano, were kept in the departments of Biological science, Bayero University, Kano for two weeks acclimatization. The animals were grouped into five (I, II, III, IV and V) of 10 animals each. Group II, III, IV and V were administered with 120mg/kg CCl<sub>4</sub>, 10mg/kg livolin (a standard antihepatotoxic drug) and 10mg/kg methanol and ethanol extract of *C. procera* roots respectively; while group I and II serve as normal and negative control respectively. Gps III – V were managed as in the design protocol below; Carbontetra

Chloride (CCl<sub>4</sub>) was dissolved in olive oil and 120mg/kg body weight was injected intramuscularly.

### Protocols for evaluating hepatocurative activity of *C. procera* root prepared in subsection 2.1

**Group I:** Normal control received neither CCl<sub>4</sub> nor extract.

**Group II:** Negative control, induced with 120mg/Kg body weight (CCl<sub>4</sub>), no extract.

**Group III:** Hepato-induced toxicity rats Administered with 10mg/kg Livolin.

**Group IV:** Hepato-induced toxicity rats Administered with 10mg/kg methanol extract.

**Group V:** Hepato-induced toxicity rats administered with 10mg/kg ethanol extract.

### Biochemical assay

The following liver function indices were assayed by standard methods as specified. Aspartate amino transferase (AST) and Alanine amino transferase (ALT) was determined by the method of (Reitman and Frankel, 1957) and the method is based on transamination reaction. Alkaline phosphatase (ALP) was assayed by (Rec, 1972). Serum urea by Diacetylmonoxime method of Nessler's as describe by (Evans, 1968), serum bilirubin by (Jendrassik and Grof, 1938) and Serum Creatinine by (Chalwa, 1999). Serum Bicarbonate (HCO<sub>3</sub><sup>-</sup>) was determined by (Van Slyke, 1927), Serum Chloride ion (Cl<sup>-</sup>) by (Schales and Schales 1994), Sodium and Potassium was determined by (Henry, 1974).

### Histopathology

The biopsies of the liver were fixed with 10% formal saline, dehydrated with ascending grade of alcohol, cleared with toluene, infiltrated with molten paraffin wax. Section of the liver was stained with haematoxylin and Eosin method (Ovwioro, 2002).

### Statistical Analysis

Data were subjected to one-way analysis of variance (ANOVA) and treatment mean were compared to positive and negative control by using Tukey-Kramer Multiple Comparisons Test, a component of GraphPad InStat3 Software (2000) version 3.05 by GraphPad In

## RESULTS

### Acute toxicity study of the plant extract.

In acute toxicity study carried out in albino rats, the limit test at dose level of 2000 mg/kg body weight in single oral administration of methanol and ethanol extract did not cause any death after 72 h post-treatment in males and females rats. Also any behavioral changes including changes in skin and fur, eyes, mucus convulsion, salivation, diarrhea and lethargy did not observed in treated groups 14 days post-treatment.

### Sub-chronic toxicity study.

Table 1 and 2 shows the Serum liver enzyme activities of (ALT, AST, and ALP) and concentrations of albumin (ALB), total bilirubin (T. BIL), and direct bilirubin (D. BIL) for groups of rats orally administered with methanol and ethanol root extract of *C. procera* and livolin at 10 and 20 days respectively, while Serum levels of kidney function indices of CCl<sub>4</sub> hepatotoxic rats treated with the extract for 10 and 20 days are represented in table 3 and 4 respectively.

**Table 1: Serum activities of ALT, AST and ALP, and concentration of ALB, T. BIL and D.BIL for groups of CCl<sub>4</sub> induced Hepatotoxicity rats orally administered with methanol and ethanol root extract of *C. procera* and livolin for 10 days.**

GROUP	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	ALB (mg/dl)	T.BIL (mg/dl)	D.BIL (mg/dl)
I(positive control)	32 ± 4.5	44.6±5.08	92. ± 6.44	4.26 ± 0.24	1.37±0.17	4.0±0.3
II(negative control)	40 ± 4.1 <sup>a</sup>	64.7± 8.6 <sup>b</sup>	281 ± 22.5 <sup>a</sup>	1.78 ± 0.25 <sup>a</sup>	1.8 ± 0.09 <sup>b</sup>	8.0 ±0.27 <sup>b</sup>
III (livolin)	35 ± 2.5	44.6±6.77	99.8±2.168	2.9 ± 0.122 <sup>a</sup>	1.39 ± 0.25	2.1±0.2
V (methanol)	39 ± 1.00 <sup>b</sup>	51 ± 9.62	110 ± 10.0	3.34 ± 0.51 <sup>a</sup>	1.28 ± 0.2	6.1 ± 1.4
VI(ethanol)	34 ± 1.00	48.4±6.54	75.4±3.286	3.0 ± 0.200 <sup>a</sup>	1.17 ± 0.05	5.8 ± 1.7

Values in the same column with (a) and (b) are significant at P< 0.001 and P< 0.01 respectively when compared with the control.

**Table 2: Serum activities of ALT, AST and ALP, and concentrations of ALB, T. BIL and D.BIL for groups of CCl<sub>4</sub> induced hepatotoxicity rats orally administered with methanol and ethanol root extract of *C. procera* and livolin for 20 days.**

GROUP	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	ALB (mg/dl)	T.BIL (mg/dl)	D.BIL (mg/dl)
I(positive control)	23.8±9.58	43.6±3.28	89.4±4.53	3.5± 0.08	0.9± 0.20	0.85±0.10
II(negative control)	45.6±4.67 <sup>a</sup>	59.4±9.43 <sup>b</sup>	270±21.33 <sup>a</sup>	1.3 ± 0.20 <sup>a</sup>	1.43±0.05 <sup>b</sup>	2.2± 0.40 <sup>a</sup>
III (livolin)	20.8±5.89	40.6±5.59	95.6±3.13	2.2 ± 0.50	1.118±0.08	1.03±0.20
IV(methanol)	28.8±5.93	37.6±2.51	86.6±3.36	3.8 ±0.78	1.1 ± 0.07	0.8±0.08
V (ethanol)	27.6±2.51 <sup>c</sup>	42.8±5.630	70.4±4.27 <sup>b</sup>	2.3 ± 0.02	1.6 ± 0.52 <sup>a</sup>	0.8±0.20

Values in the same column with (a), (b) and (c) are significant at P< 0.001, P< 0.01 and P<0.05 respectively when compared with the control.

**Table 3: Concentration of urea, creatinine, bicarbonate, chloride, potassium and sodium for group of CCl<sub>4</sub> induced hepatotoxicity rats orally administered with methanol and ethanol root extracts of *C. Procera* and livolin for 10 days.**

GROUP	UREA (mg/dl)	CREAT (mg/dl)	HCO <sub>3</sub> <sup>-</sup> (mmol/l)	Cl <sup>-</sup> (mmol/l)	K <sup>+</sup> (mEq/l)	Na <sup>+</sup> (mmol/l)
I (positive control)	0.77 ± 0.10	24.10 ± 4.90	31.20 ± 0.90	276.00±43.00	5.10 ± 1.10	315.60 ± 41.20
II(negative control)	0.58 ± 0.04	14.30 ± 3.40	66.90 ± 5.10 <sup>a</sup>	209.00±37.90 <sup>b</sup>	10.10 ± 0.70 <sup>a</sup>	278.00 ± 41.80
III (livolin)	0.90 ± 0.30 <sup>a</sup>	30.40 ± 0.20	33.40 ± 0.90	173.00±3.60 <sup>a</sup>	5.80 ± 0.20	200.70 ± 3.40 <sup>a</sup>
V (methanol)	0.70± 0.10 <sup>c</sup>	33.40± 2.50 <sup>c</sup>	43.00 ± 6.20	177.80±6.50 <sup>a</sup>	4.70 ± 1.80	214.70±6.70 <sup>a</sup>
VI (ethanol)	0.60 ± 0.20	30.00 ±3.80	41.30 ± 4.50	167.70±0.16 <sup>a</sup>	6.90 ± 2.20 <sup>c</sup>	202.10±4.40 <sup>a</sup>

Values in the same column with (a), (b) and (c) are significant at P< 0.001, P< 0.01 and P<0.05 respectively when compared with the control.

**Table 4: Concentration of urea, creatinine, bicarbonate, chloride, and potassium and sodium for group of CCl<sub>4</sub> induced hepatotoxicity rats orally administered with methanol and ethanol root extracts of *C. procera* and livolin for 20 days.**

GROUP	UREA (mg/dl)	CREAT (mg/dl)	HCO <sub>3</sub> <sup>-</sup> (mmol/l)	Cl <sup>-</sup> (mmol/l)	K <sup>+</sup> (mEq/L)	Na <sup>+</sup> (mmol/l)
I (control)	1.7 ± 0.08	17.97 ± 1.8	18.9 ± 1.3	144.2 ± 13.7	2.40 ± 0.57	164.3 ± 12.7
II (negative control)	1.86 ± 0.2	25.6 ± 1.99 <sup>a</sup>	30.3 ± 0.68 <sup>a</sup>	162.2 ± 6.7	5.30 ± 0.60 <sup>c</sup>	188.8 ± 6.93
III (livolin)	0.87 ± 0.2 <sup>a</sup>	32.6 ± 3.14 <sup>a</sup>	29.1 ± 1.08 <sup>a</sup>	106.7 ± 3.2	4.40 ± 0.58	132.6 ± 5.01
V (methanol)	1.33 ± 0.12 <sup>c</sup>	24.0 ± 1.8	31.6 ± 1.3 <sup>a</sup>	90.2 ± 5.8	3.00 ± 1.13	116.7 ± 7.8
VI (ethanol)	1.5 ± 0.24	33.6 ± 2.6 <sup>a</sup>	26.5 ± 1.1 <sup>a</sup>	152. ± 33.8	36.1 ± 1.33	184.0 ± 32.8

Values in the same column with (a), (b) and (c) are significant at P < 0.001, P < 0.01 and P < 0.05 respectively when compared with the control.

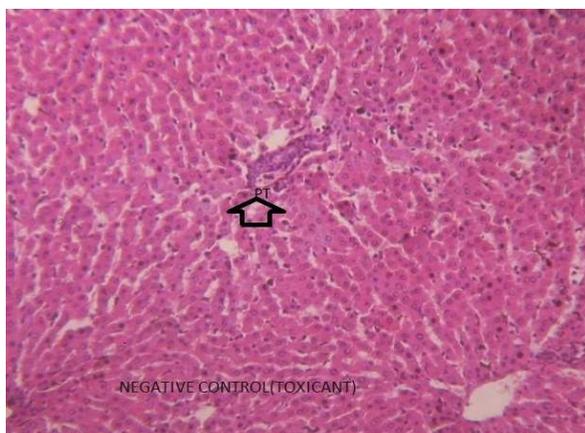


Plate 2: Histopathology lesions. Photomicrograph of transverse section of albino rats toxicant of group B. showing severe liver damage with perlobular liver necrosis of the portal area. H & E stain x 10

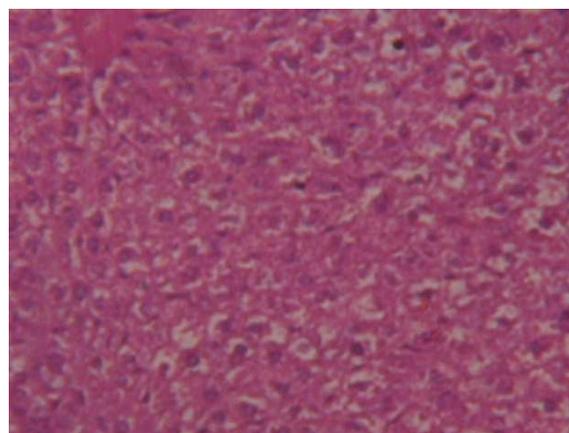


Plate 6: Histopathology lesions; Photomicrograph of transverse section of albino rats test group F treated with ethanolic extract of *C. procera* root, showing severe cytolysis and kryolysis. H & E stain x 10.

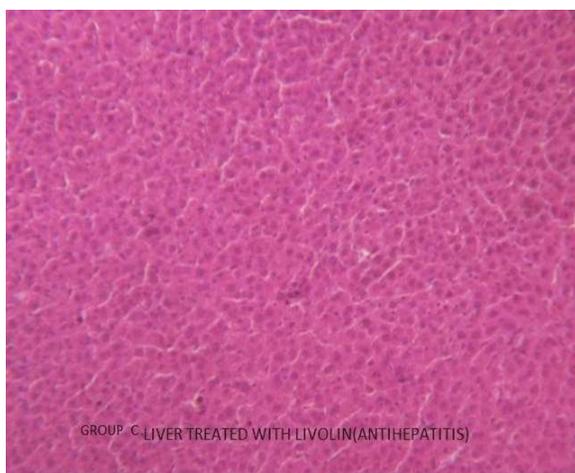


Plate 3: histopathology lesions, Photomicrograph of transverse section of albino rats liver treated with livolin; shows normal liver architecture with the hepatocytes arranged as thin plates separated by fine vascular sinusoid. H & E stain x 10.

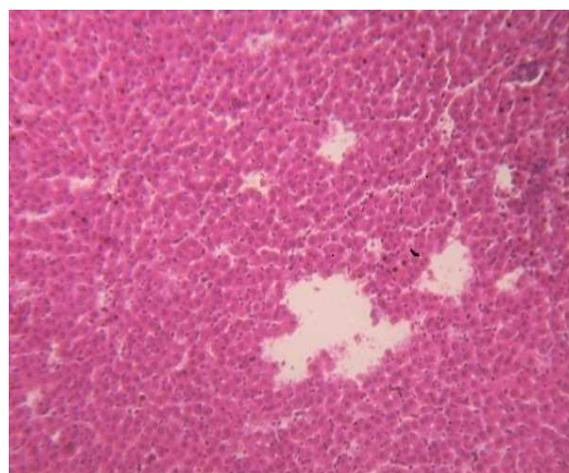


Plate 5; Histopathology lesions, photomicrograph of transverse section of albino rats group E liver treated with methanolic extract of *C. procera* root, showing a severe liver damage with mild perlobular toxic necrosis. H & E stain x 10

## DISCUSSION

In this study, CCl<sub>4</sub> induced toxicity rats (GpII) was evident by rise in serum levels of the liver function indices (AST, ALT, ALP, Total and Direct Bilirubin as compared to normal control (GpI). The injury or disease of liver can be judged by estimating the serum parameters like ALT, AST, ALP, ALB and BLN. The serum level of the enzymes is increased due to cellular leakage (Molander, 1995). In CCl<sub>4</sub> induced

toxicity, CCl<sub>3</sub><sup>°</sup> is produced as a free radical. It binds to lipoprotein leading to peroxidation of lipid of endoplasmic reticulum. The fact that ALT is raised at both exposure indicates that CCl<sub>4</sub> have induced toxicity in accordance with (Alhassan *et al.*, 2009) who reported that rats treated with high dose of CCl<sub>4</sub> developed profound hepatic damage and oxidative stress as evidenced by increase in the serum activities

of ALT, AST, ALP, Total and Direct Bilirubin that are indicators of cellular leakage and loss of functional integrity of cell membrane in liver. The toxicity increases with the increase of day of exposure (Garba *et al.*, 2013); (Aidemiet *et al.*, 2014).

Daily oral administration of livolin at 10mg/kg for 10 and 20 days exposure brought back the activities of the liver function indices to near normal except for Albumin which reduced after 10 days but become elevated after 20 days an evidence of increased activity with the increase in the day of exposure. The decreased Albumin could be due to decline in total protein content, can be deemed as a useful index of the severity of cellular dysfunction in chronic liver diseases (Venukumar and Latha, 2002). This shows that livolin forte has an ability to cure liver damage induced by CCl<sub>4</sub>; this could be due to the presence of essential phospholipids (polyunsaturated phosphatidylcholine). Phosphatidylcholine has hepatoprotective effect against ethanol, alkyl alcohols, tetrachloride, paracetamol and galactosamine (Parris, 1996). It also appears to be a defense against steatosis and fibrosis of the liver by speeding regeneration and stabilization of membranes, and stopping lipid peroxidation (Olukiran *et al.*, 2014). It has also been reported by (Thabrew *et al.*, 1987) that antihepatotoxic agents decrease the increased level of these enzymes.

Daily oral administration of 10 and 20 doses of 10mg/kg methanol root extract of *Calotropis Procera* (MRECP) when compared with normal control (Grp I) and toxicant group (grp II) showed a significant increase in ALT and significant decrease (<0.05) in ALB indicating non-significant liver improvement at 10 days exposure of the extract. The activities of all the liver function indices were normalized after 20 days exposure to methanol root extract of *Calotropis Procera* (MRECP). The observed increase in ALT activity and decrease in ALB indicates that MRECP may not have anti-hepatitis activity at the adopted dosage. Since ALT is the principal enzyme of the liver and hypoalbuminaemia is associated with impaired albumin synthesis in the liver or liver disease (Doumas *et al.*, 1971). The result of histopathology of MECPR plate 5 after 10 day's shows severe liver damage with mild perilobular toxic necrosis. At 20 days exposure, MECPR produced almost similar curative effect with livolin forte (polyunsaturated phosphatidylcholine), hence the values are comparable and very potent against chemically induced liver disease. Although the possible mechanism for the anti-hepatotoxic properties of MECPR have not been reported yet. It is assumed that the effect of MECPR on liver protection is related to glutathione-mediated detoxification as well as free radical suppressing activity. On the other hand, the kidney parameters show statistically significant decrease in urea, creatinine, chloride and sodium and statistically insignificant increase in bicarbonate and potassium when compared with normal control group (Grp I). After 20 days however, the urea concentration remain low an indication of toxicity in the liver, as increase protein utilization probably induced by the extract and/or impairment in the kidney excretory function. As was reported by other

workers that hepatic toxicity induced by CCl<sub>4</sub> also causes disorders in kidneys, lungs, and testis as well as in blood by generating free radicals (Ozturk *et al.*, 2003). Findings by (Ogeturk, *et al.*, 2005) suggested that exposure to this solvent (CCl<sub>4</sub>) causes acute and chronic renal injuries. However, the pathogenesis of Carbon tetrachloride (CCl<sub>4</sub>) - induced renal dysfunction is not completely known. It may be due to the functional state of liver, or renal injury may develop independently to hepatic events (Ogeturk, *et al.*, 2005). The kidney condition is improved after 20 days exposure, due to insignificant change in creatinine, chloride, potassium and sodium. Sodium is usually measured with other factors to evaluate problems associated with kidneys, adrenal glands, muscles and nerves. Although other factors can cause body's sodium to go up, these include eating a lot of salt, reduced fluid intake, eating licorice, hyperventilation and kidney disease. Factors such as vomiting or diarrhea, burn, less water, when adrenal glands are not working well, heart or liver disease and vigorous exercise can cause lower body's sodium level (Tom, 2009).

Daily oral administration of 10mg/kg ethanol root extract of *Calotropis procera* (ERECP) for 10 days produced a decrease in ALB, while no statistically significant difference was observed in the other liver biochemical parameters. This may be due to the ability of ERECP to cure liver damage. This agrees with the findings of (Bhumika, 2016) who found that, treatment with hydro-ethanolic extract of *C. procera* flowers (200 mg/kg and 400 mg/kg) has brought back the altered levels of biochemical markers to the near normal levels in a dose dependent manner. Our findings are also similar to the study conducted by Absaret *et al.*, (2007) on ethanolic extract of the flower of *C. procera*. Treatment for 20 days elevate the liver parameters especially ALT a principal enzyme of the liver, indicating toxicity. This could possibly be due to the toxic effect of the extract itself on the liver and kidney as evidenced by elevated levels of markers of hepatonephrotoxicity due to the accumulation of toxic doses of the active principles of the root bark extract of the plant. Findings from this study corroborate the results of Daniel *et al.*, 2013); and Pouokamet *et al.*, (2007)

who reported marked increases in these parameters after administering aqueous extract of *C. procera*. It also supports the results of Buraimoh (2001) that showed that the ethanolic leaf extract of *C. procera* is detrimental to the integrity of the liver tissues as evident in the necrotic nature of the liver. (Mohammed, 2011) also reported the toxicity of the root extract in both female and male rats after long exposure. This study contradicts the reported hepatoprotective activity of the root bark extract (Basuet *et al.*, 1997; Patilet *et al.*, 2011). The histopathological analysis of the liver, plate 6 confirms the toxicity, showing a severe cytolysis and kryolysis of the liver cells (Yemitan and Izegebu, 2006). This is contrary to the study conducted by (Bhandari *et al.*, 2003) who studied the effect of ethanol extract of ginger on country- made liquor (CML) induced liver injury in rats. Their result showed that administration

of ginger ethanolic extract (200mg/kg) orally from day 15 to 21 along with CML produce significant lowering of serum AST, ALT, ALP and tissue lipid peroxide levels.

The result of the kidney parameters indicates statistically significant depletion of urea, creatinine and electrolytes. This might be due to interference with the release of anti-diuretic hormone (ADH) or the ability of the kidney to produce concentrated urine can greatly increase some nutrient losses, resulting in potassium depletion, hypocalcaemia, pyometra, inadequate protein uptake by reducing urea production, and Cushing's syndrome (Michell, 1979). Hyponatremia is primarily associated with renal sodium wasting and water retention due to an inability to excrete ingested water. The latter may be due to the persistent secretion of ADH, although free water excretion can also be limited in some disorders like renal failure and primary polydipsia in which the ADH levels may be appropriately suppressed. Serum potassium, the major cation in the intracellular fluid, is normally maintained within a narrow range through

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- CONCLUSION**
- In conclusion, the methanol root extract of *C. Procera* produce more curative effect against CCl<sub>4</sub> induced liver toxicity in albino rats than ethanol extract of the plant.
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