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Hepatoprotective Potentials of Aqueous extracts of *Ficus asperifolia* MIQ leaves on CCl_4 induced liver damage in Wistar rats

Opotu, R.O., Uwakwe, A.A., Chuku, L.C and Olua, V.*
Department of Biochemistry Faculty of Science University of Port Harcourt
Augustine.uwakwe@uniport.edu.ng; Lawrence.chuku@uniport.edu.ng;
*corresponding Author: *vyckol@yahoo.com*

Abstract

The hepatoprotective effect of aqueous leaf extracts of *Ficus asperifolia* was investigated in rat model of liver injury induced by carbon tetrachloride ($CC1_4$). An aliquot of 50mg/kg, 100mg/kg, 200mg/kg and 300mg/kg body weight of extracts of *f. asperfolia* was used in the treatment of separate rat groups for 4 weeks after liver damage was established. The hepatic status was monitored using lipid per oxidation with malondialdehyde (MDA) as a marker, Liver marker enzymes (Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Alkaline phosphatase (ALP)) as well as other biochemistry tests (albumin, total protein, globulin and bilirubin level) measured weekly. A significant rise in the various markers were observed after liver damage was induced. Treatment with various concoction of the plants extract showed significant reversal of these parameters when compared with the negative control group ($p<0.05$). However rats with CCl_4 induced liver damage showed dose dependent improved liver conditions while being treated with aqueous leaf extract of *F. asperifolia*. The group treated with 300mg/kg extract however showed best results. Histopathological investigations supported obtained results. *F.asperifolia* may therefore be biochemically significant in reversing hepatotoxicity caused by CCl_4 and may thus be useful in the treatment of liver damage.

Keywords: *Ficus asperifolia;* Hepatotoxicity; Hepatoprotective; Carbon Tetrachloride

Introduction

It is noted that usage of herbs by locals have been implemented with the masses ignorant of the therapeutic properties in these herbs. This days more is known about the brain behind therapeutic potentials of herbs, thereby paving way for the commercialization of their usage in ameliorating disease conditions. Plants have basic nutritional importance by their content of protein, carbohydrates, fats and oils, minerals. In addition to vitamins and provitamins in fruits and vegetables, bioactive plant components often called phytochemicals are present. It has been discovered that regular consumption of fruits, vegetables, herbs and spices has always been associated with health benefits. These plants contain a wide variety of biologically active, non nutritive compounds known as phytochemicals (Sheetal and Jamuna, 2009).Researches have come up with the fact that some of these plant chemicals which biologically function as antinutrients or antioxidants have potentials in helping to reduce the risk of several deadly diseases in man (Ibrahim and Fagbohun, 2012). Plants are used for the treatment of diseases due to the presence of phytochemicals such as flavonoid, alkaloids, phytosterol, tannins and so many others.

The genius, *Ficus* with about several species and of the family of *Moraceae* are Collectively known as figs or fig trees found throughout the tropics. The leaves, stems, roots and fruits of fig trees have proven

useful in ameliorating certain disease conditions. In Africa *F. asperifolia,* grows comfortably along streams and have been proven to have antibacterial and reproductive effects (Annan and Houghton, 2008). Studies shows that *Ficus asperifolia* is rich in phytochemicals such as alkaloids , phytates, saponins, tannin and oxalate etc with the nine essential amino acids not left behind, (Kafimiya *et al.*, 2010).

Classification

Kingdom	Plantae
Phylum	Angiospermae
Class	Embryophyta
Order	Rosales
Family	Moraceae
Genius	Ficus
Subgenius	Sycidium
Species	asperifolia

Studies have also revealed that aqueous extracts of *Ficus asperifolia* exhibits detoxification potentials and therapeutic effects against CCl_4 induced kidney injuries and oxidative damage in rats which could be attributed to its antioxidant constituents (Oluwafemi *et al.,* 2016), thereby confirming the an earlier work earlier by Ojo and Akintayo, 2014, who revealed antioxidant potentials and phenolic content of the extract which could be responsible for its popular and wide traditional use.

Materials and Methods

Collection and identification of Plant material

Ficus asperifolia fresh leave samples were obtained from Alakahia in Rivers state, Nigeri. They were identified at the Department of Plant Sciences and Biotechnology, University of Port Harcourt, Nigeria.

Preparation of crude extracts of *Ficus asperrifolia* MIQ. Leaves

Sampled fresh leaves of *Ficus asperifolia miq* were washed and air dried. The dried samples were crushed and extracted with distilled water at 1:4 ratio leaf (g) to water (ml) after 25 hours at room temperature. Whatman number 1 filter paper was used to filter the extract. The filtrate was placed in a rotary evaporator using water bath at $46^{o}C$ - $60^{0}C$ in order to obtain the crude extract.

Experimental animals

About ninety six wistar albino rats weighing 175-250g breed in the animal house of the Department of Biochemistry, University of Port Harcourt were used. They were randomly selected and kept in eight (8) groups of twelve (12) rats each.

Each group was kept in a separate cage, all the animals were fed with commercially produced fed and water. The feed was purchase from livestock feed shops in Choba, a division of the livestock Feeds Nigeria Ltd, Lagos. Water was obtained from tap connected from the water treatment plant of the university. The animals were subjected to 12hours of light cycle in a properly ventilated room. The animals were allowed to acclimatize for a period of 7 days before the experiment commenced.

Collection of blood and liver samples for analysis

Each animal to be sacrificed was withdrawn from its cage 24hours after administration of extract and anesthetized in chloroform saturated chamber.
The jugular vein was cut and fresh blood was collected into appropriately labeled heparin and EDTA sample bottles for chemical pathology tests. The heparinized blood was centrifuged at 500rpm using an MSE centrifuge for 10minutes to obtain plasma.

After collection of blood samples, the livers of the rats were harvested and preserved in sample bottles containing 10% formaldehyde. The liver samples were later analysed at the histopathology laboratory of the Department of Anatomy, University of Port Harcourt.

Methods for histological examination.

Liver tissue Were fixed in formalin and the methods proposed by Baker and Silverton (1985) was adopted. Following complete dewaxing, the slides were stained for morphological change using the standard Heamotoxylin (which stains cell nuclei blue to black) and Eosin (which stains the cytoplasm of the cell pink) routine method as described by Baker and Silverton (1985).

Serum enzyme assay

Blood serum was obtained by collecting blood and allowing it to coagulate so as to separate the cells, leaving a clear colored liquid substance called serum.

ALT Activity

About 0.1ml of sample was pipette into a test tube labeled sample while 0.1ml distilled water was pipette into a test tube labeled reagent blank. 0.5ml of buffer solution was added to both the samples and the reagent tubes. The solution in the tubes were mixed and incubated at 37^0C for 30 minutes. An aliquot of 0.5ml 2,4-dinitrophenylhydrazine was then added to both tubes to enhance color development for colorimetric measurement. The tubes were shaken and allowed to stand at 25^0C for 20 minutes. About 5.0ml NaOH was added to both tubes, mixed well and allowed to stand for five minutes in order to terminate previous reactions.

The absorbance of the sample was read against that of the reagent blank at a wavelength of 540nm. The activity of the enzyme (ALT) in plasma samples was read by matching absorbance reading against different ALT concentration in U/L.

Aspartate amino transferase activity methods of estimation

Exactly 0.1ml of serum was pipette into the sample tube while 0.1ml distilled water was pipette into the reagent blank tube, 0.5ml of buffer solution was added to both the sample and reagent blank tubes, mixed and incubated at 37^0C for 30 minutes, 0.5ml 2,4-dinitrophenylhydrazine was then added to both tubes to enhance colour development for colorimetric measurement. The tubes were shaken and allowed to stand at *25°C* for 20 minutes. 5.0ml NaOH was then added to both tubes, mixed well and allowed to stand for 5minutes in order to terminate previous reactions. The absorbance of the sample was read against that of the reagent blank at a wavelength of 540nm. The activity of the enzyme (AST) in plasma samples was obtained by matching absorbance reading against different AST concentrations in U/L.

Alkaline phosphataseactivity method of estimation

A working reagent that is composed of I vial of substrate (p-nitrophenyl phosphate) reconstituted with appropriate amount of buffer was prepared. 1ml of working reagent was added to the test tube. 0.2ml sample (serum) was then added, mixed and initial absorbance was read at *450nm.* A timer was started simultaneously and absorbance was read again after 1, 2 and 3minutes. The semi-micro method was employed and the ALP activity in U/L was calculated thus:

U/L = 2760 x ΔA Where ΔA = change in absorbance.

Lipid peroxidation assay

Lipid peroxidation assay was carried out through the estimation of malondialdehyde (MDA).

Liver MDA Concentration determination

An aliquot of 1g of liver tissue was homogenized in 10 ml of ice-cold Tris-HCI buffer and the homogenate was centrifuged at 3000xg for 10 minutes. 0.2 ml of the supernatant was added to 1 ml of conc. glacial acetic acid in a centrifuge tube. I ml of 1% TBA dissolved in 0.2% NaOH was added to the mixture, put in boiling water for 15 minutes, allowed to cool and centrifuged at 2,500xg for 10 minutes. Absorbance of the clear supernatant was read at 532nm against the reagent blank. MDA concentration was determined from the absorbance by using a molar extinction coefficient of 1.56 x $10^5M^{-1}cm^{-1}$ according to the Adam-Vizi and Seregi (1982);

MDA (units/mg tissue) = (ΔOD x V x 1) ÷ (molar absorbance x Vs x mg tissue)
Where:

ΔOD = Difference between absorbance of blank and test at 532nm
V =Total volume of the reaction mixture
Vs = Volume of sample (microsomal fraction in ml)
Molar absorbance index for MDA = 1.56 x $10^5M^{-1}cm^{-1}$

Biochemical analyses

Bilirubin concentration determination

About 0.2ml of reagent 1 (29mmol sulphanilic acid + 0. 1 7N HCl), 1.0ml of reagent 3 (0.26mmol/L caffeine + 0.52rno1/L sodium benzoate) and 0.2ml sample were pipetted into a test tube and served as sample blank. 0.2ml of reagent 1, 0.005ml of reagent 2 (25mmol/L sodium nitrite), 0.lml of reagent 3 and 0.2m1 of sample were pipetted into another test tube. This served as the test sample. It was properly mixed and allowed to stand for l0minutes at 20°C - 25°C. 1ml of reagent 4 (1.9N NaOH + 0.93mol/L tartrate) was added to each test tube, mixed and allowed to stand for *5-* 30minutes at *25°C.* the absorbance A_{TB} was taken at 578nm.

Total bilirubin was thus calculated as: Total Bilirubin (mmol/L) = 1 85 x A_{TB}

Determination of total protein

Three test tubes were labelled 1 (reagent blank), 2 (standard) and 3 (test sample). Into test tube 1, 0.2ml distilled water and reagent (100mmol/L sodium hydroxide + 16mmol/L Na-k-tartrate) were added. Into test tube 2, 0.02ml sodium azide was pipetted and 1ml Of reagent 1 was added. 0.02m1 of serum and 1ml of reagent 1 was pipetted into test tube 3. The solutions were mixed and incubated at *25°C* for 30minutes. Absorbance was measured of sample A_{SAMPLE} and standard $A_{STANDARD}$ against the reagent blank at 546nm.

Total protein concentration was calculated thus:

Total Protein conc. = A_{SAMPLE} / $A_{STANDARD}$ X standard conc.

Determination of albumin concentration

To cuvette C_1 (blank) was added 0.01mI of distilled water and 3.00ml of BCG reagent *(*75mmol*/L* succinate buffer of pH 4.2 + 0.l5mmol/L BCG + brij 35 preservative).

To cuvette C_2 (standard), was added 0.01 ml of standard composed of human serum albumin *(*4.5g*/*dl*)*, l00mmol Tris buffer of pH 7.3 and 3m1 of BCG reagent.

To cuvette C_3 (test sample) was added 0.01rnl of plasma and 3.00rnl of BCG reagent. The preparations were incubated for 5minutes at 25°*C*. The absorbance of the samples A_{SAMPLE} and that of the standard $A_{STANDARD}$ against the reagent blank were read. The concentration of albumin was thus calculated:

Albumin conc. (g/dl) = A_{SAMPLE} / $A_{STANDARD)}$ X conc. Of standard

Determination of globulin concentration.

The concentration of globulin in plasma or serum is calculated from the concentrations obtained for total protein and albumin. Globulin is thus calculated as:

Globulin conc. = Total protein conc. (g/l) — Albumin conc.(g/l)

Statistical analysis

Data collated were analyzed using analysis of variance (ANOVA). Test significance was accepted at 95% confidence limit ($p<0.05$).

Results

Table 1. Effect of crude extract of *F. asperifolia* on TOTAL PROTEIN (g/l) of Wistar albino rats.

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	67.00±2.65	67.00±2.65	67.00±2.65	67.00±2.65
CCl_4	0.5ml/kg	61.00±2.00	51.00±7.55	44.00±0.00	66.00±1.00
F. asperifolia	200mg/kg	85.00±1.00	80.00±2.00	83.00±1.00	84.00±0.00
	300mg/kg	82.33±4.50	80.00±4.00	85.00±1.00	85.33±1.15
CCl_4 +	50mg/kg	65.67±2.52	64.00±1.00	64.00±1.00	67.00±1.73
F. asperifolia	100mg/kg	71.00±1.00	66.67±1.52	66.67±1.53	71.33±1.15
	200mg/kg	77.00±1.00	71.00±3.00[b]	69.67±4.51	75.33±1.15
	300mg/kg	77.00±3.00	76.00±2.00	76.00±2.00	78.00±2.00

Table 2. Effect of crude extract of *F. asperifolia* on ALBUMIN(g/dl) level of Wistar albino rats.

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	4.20±0.20	4.07 ± 0.31	4.07 ± 0.31	4.07 ± 0.31
CCl_4	0.5ml/kg	2.90±0.10	2.70±0.10	2.50±0.10	2.30±0.10
F. asperifolia	200mg/kg	4.70±0.40	4.57±0.58	4.57±0.58	4.33±0.2
	300mg/kg	4.17±0.15	4.40±0.10	5.00±0.10	4.77±0.15
CCl_4 +	50mg/kg	4.00±0.80	3.90±0.00	3.67±0.15	3.47±0.11
F. asperifolia	100mg/kg	3.90±0.10	3.77±0.58	3.77±0.58	3.97±0.58
	200mg/kg	4.27±0.15	4.20±0.20	4.27±0.15	4.30±0.10
	300mg/kg	4.60±0.10	4.17±0.45	4.60±0.10	4.70±0.00

Table 3. Effect of crude extract of *F. asperifolia* on GLOBULIN (g/l) level of Wistar albino rats .

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	25.50±0.50	25.50±0.50	25.50±0.50	25.50±0.50
CCl_4	0.5ml/kg	32.00±1.00	24.00±6.56	19.00±1.00	38.00±7.00
F. asperifolia	200mg/kg	38.00±3.00	34.33±1.53	39.00±2.00	38.67±0.58
	300mg/kg	41.00±3.00	36.00±3.00	35.00±0.00	37.67±0.58
CCl_4 +	50mg/kg	28.00±1.0	25.00±1.00	22.67±1.52	33.00±1.00
F. asperifolia	100mg/kg	32.00±2.00	29.33±2.08	27.00±3.00	32.33±0.58
	200mg/kg	34.33±0.58	29.00±1.00	30.00±0.00	32.00±1.00
	300mg/kg	31.00±4.00	34.33±2.52	30.00±1.00	32.00±1.00

Table 4. Effect of crude extract of *F. asperifolia* on BILIRUBIN (mmol/l) level of Wistar albino rats.

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	4.67±0.95	4.67±0.95	4.67±0.95	4.67±0.95
CCl_4	0.5ml/kg	13.90±0.90	17.40±0.60	19.00±0.00	15.33±1.10
F. asperifolia	200mg/kg	5.60±0.00	5.67±0.58	4.80±0.8	5.50±0.10
	300mg/kg	3.90±0.10	3.97±0.15	3.77±0.05	4.07±0.11
CCl_4 +	50mg/kg	13.93±0.90	14.93±0.12	3.93±0.90	11.33±0.58
F. asperifolia	100mg/kg	10.67±1.35	12.00±0.00	13.50±0.50	10.67±0.58
	200mg/kg	10.17±0.85	11.93±0.12	12.03±0.95	8.00±0.00
	300mg/kg	6.50±0.90	7.93±0.12	11.17±1.85	7.90±0.10

Table 5. Effect of crude extract of *F. asperifolia* on AST (u/l) level of Wistar albino rats.

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	21.00±2.00	21.00±2.00	21.00±2.00	21.00±2.00
CCl_4	0.5ml/kg	63.00±4.00	82.00±6.56	88.67±0.58	59.00±0.00
F. asperifolia	200mg/kg	25.00±2.00	23.00±4.00	27.00±4.00	16.00±0.00
	300mg/kg	21.00±00	17.33±1.53	20.00±3.00	11.00±1.00
CCl_4 +	50mg/kg	35.00±1.00	71.33±4.50	76.00±0.00	35.00±1.00
F. asperifolia	100mg/kg	33.67±2.52	63.00±4.00	58.00±1.0	31.00±0.00
	200mg/kg	32.33±3.06	49.33±2.52	47.00±0.00	28.00±3.00
	300mg/kg	30.33±2.52	44.00±3.00	41.33±0.57	23.00±2.00

Table 6. Effect of crude extract of *F. asperifolia* on ALT (u/l) level of Wistar albino rats.

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	23.00±2.00	23.0±02.0	23.00±2.00	23.00±2.00
CCl_4	0.5ml/kg	52.00±0.00	59.33±7.51	9.33±7.51	47.66±4.59
F. asperifolia	200mg/kg	14.50±2.50	27.67±6.02	10.67±2.31	9.33±2.30
	300mg/kg	12.67±3.06	30.67±1.53	10.00±2.00	8.00±2.00
CCl_4 +	50mg/kg	25.00±00	40.72±2.08	38.67±1.53	27.33±2.52
F. asperifolia	100mg/kg	30.67±5.86	40.33±2.31	30.00±2.00	26.33±1.53
	200mg/kg	29.00±4.00	10.00±2.00	27.67±2.52	25.33±1.53
	300mg/kg	25.33±2.52	10.00±2.00	23.33±2.08	23.00±2.65

Table 7. Effect of crude extract of *F. asperifolia* on ALP (u/l) level of Wistar albino rats

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	34.00±0.00	34.00±0.00	34.00±0.00	34.00±0.00
CCl_4	0.5ml/kg	67.00±0.00	76.00±1.73	73.00±2.00	59.00±4.00
F. asperifolia	200mg/kg	41.00±2.00	40.00±3.00	38.00±1.00	37.67±0.58
	300mg/kg	38.00±1.00	34.67±2.52	34.67±2.52	36.00±1.00
CCl_4 +	50mg/kg	54.00±1.00	61.00±2.00	63.00±0.00	54.00±1.00
F. asperifolia	100mg/kg	51.67±2.08	44.00±1.00	46.67±1.53	39.00±1.00
	200mg/kg	45.33±2.52	38.00±1.00	44.33±2.52	37.00±1.00
	300mg/kg	32.33±2.29	35.33±1.53	39.00±1.00	29.00±1.00

Table 8. Effect of crude extract of *F. asperifolia* on MDA level of Wistar albino rats

TREATMENT	DOSE	DAY 7	DAY 14	DAY 21	DAY 28
CONTROL	-	0.29±0.01	0.29±0.01	0.29±0.10	0.29±0.01
CCl_4	0.5ml/kg	0.71±0.03	0.89±0.09	1.10±0.21	0.70±0.10
F. asperifolia	200mg/kg	0.29±0.01	0.24±0.04	0.19±0.01	0.20±0.10
	300mg/kg	0.24±0.04	0.25±0.05	0.16±0.02	0.20±0.00
CCl_4 +	50mg/kg	0.50±0.01	0.65±0.05	0.75±0.05	0.50±0.10
F. asperifolia	100mg/kg	0.41±0.02	0.50±0.00	0.70±0.10	0.40±0.00
	200mg/kg	0.40±0.00	0.50±0.10	0.55±0.05	0.37±0.06
	300mg/kg	0.35±0.05	0.44±0.05	0.50±0.00	0.20±0.02

Discussion

The results of the hepatoprotective potentials of aqueous extracts of *Ficus asperifolia* MIQ leaves on CCL_4 induced liver damage in Wistar rats is as shown in Tables 1 to 8 above, the parameter studied include levels of total protein, albumin and bilirubin concentrations, ALT, AST, ALP, and MDA activities. These were used to measure both CCl_4 induced chronic hepatotoxicity and subsequent antioxidant effect of *Ficus asperifolia* against the damage caused.

In studying the hepatoprotective effect of aqueous extracts of the leaves of *f. aspersoria*, the well described models of Abdel-moemin *et al.*,2009 and Jayasekhar *et al.*, 1997 were employed.

With respect to reports on previous works done by other researchers (Abdel-moemin *et al.*, 2009; Obi *et al.*, 1998 and Jayasekhar *et al.*, 1997), the result revealed that CCl_4 caused an elevation in serum ALP,ALT AST, total bilirubin ad albumin concentration, there was also a increase in liver MDA concentration, which progressed with progressive administration of CCl_4, indicating liver injury. Hence, serum or plasma enzymes have been used as indices monitoring chemically induced tissue damage. It has also been shown that CCL_4 has the capability of damaging other body organs such as kidney when exposure is prolonged. Though injected intraperitioneally for the purpose of this experiment, CCl_4 when inhaled orally can raise serum hepatic enzymes, indicating liver damage (Barnes and Jones,

1967). However, CCl_4 is highly toxic thus, mineral oil was proposed for use as an innocuous vehicle administering it to experimental animals.

The level of enzyme leakage from the liver and other organs serves as a monitor for the extent of the damage caused by CCL_4 (Brattin *et al.*, 1985),. The pattern of enzyme activity in the serum reflects the condition of the organs and tissue.

This study revealed the hepatoprotective and antihepatotoxicity potentials of aqueous extracts of the leaves of *Ficus asperifolia* and also its antioxidant effect which by extension, could stall membrane damage.

The results obtained from the liver markers and liver MDA activities indicated a dose dependent remediation of the effect of CCl_4 administered.

Group2, which was treated with only CCl_4 at a dose of 0.5mg/kg body weight of animal twice a week for three weeks indicated a marked increase in serum ALT, AST, ALP and liver MDA activities, these increase was seen to be directly proportional to administered doses, indicating marked liver damage due to lipid peroxidation. The treatment groups 5, 6, 7 and 8, which received 50mg/kg, 100mg.kg, 200mg/kg, and 300mg/kg body weight aqueous extracts of the plant showed a significant dose dependent reduction in the these parameters throughout the treatment period hen compared to group 2. ALT was the most reduced serum enzyme activity at $p<0.05$. the reversal of the serum enzyme activities and liver MDA concentration by the extract may be due to the prevention of leakage of intracellular enzyme by its membrane stabilizing activity. This is in agreement with the healing of hepatic parenchyma cells and the regeneration of hepatocytes as reported by Sipes *et al.*, 1977.

Biochemical parameters such as albumin and bilirubin serve as indicators for liver damage. Albumin is used as an indicator of liver impairment, reduced absorption or protein loss (Sacher and McPherson, 2000).

An observable significant reduction in albumin level was noted in group 4 treated with only CCL_4, at $p<0.05$, indicating possible chronic liver disease (Gaul *et al.*, 1984). The extract of the leaves of *Ficus asperifolia*, however was able to restore values to normalcy based on the dose administered. Bilirubin on the other hand was markedly increased in the CCL_4 group. This was however noticeably reduced at $p<0.05$ when the varying concentrations of the extract was administered.

It is worthy of note that on final week, when CCl_4 was not administered to any of the groups, there was a significant reduction in serum enzyme levels of group 2 (CCl_4) group when compared with the values of the previous week, attesting to the detoxifying and regenerative potentials of the liver as shown in literatures. This was probably not noticed in the previous weeks as the exposure to CCl_4 was chronic.

The efficacy of any drug in countering hepatotoxicity is dependent mainly on its ability to either reduce the harmful effect or restore the normal hepatic physiology disrupted by the toxin. Histopathlogical studies as shown in the appendix shows the change in CCl_4 induced fatty degeneration, effacement of lobular architecture, infiltration with chronic inflammatory cells and ballooning degeneration of cells with the administration of extracts of *Ficus asperifolia* at the afore stated doses. The observation supports the results for biochemical tests carried out. Though the biochemical mechanism for the antihepatotoxic effect of the leaf extract was not examined, in this study, it is possible that the plant may have acted directly or indirectly in alleviating liver damage

Conclusion

This study however shows that exposure of experimental rats to CCl_4 could induce chronic liver damage as shown by the abnormal increase in serum activities of ALT, AST, ALP, bilirubin, increased liver MDA and reduced PCV and albumin.

However, the administration of aqueous extracts of *Ficus asperifolia* leaves at varying doses (50mg, 100mg, 200mg, and 300mg) had a significant antihepatotoxic effect as shown in the reduction and reversal of the above parameters when compared with the CCl_4 group.

Conflict of interest: The authors declares no conflict of interest.

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APENDIX 1

PLATE 1. NORMAL CONTROL GROUP 1

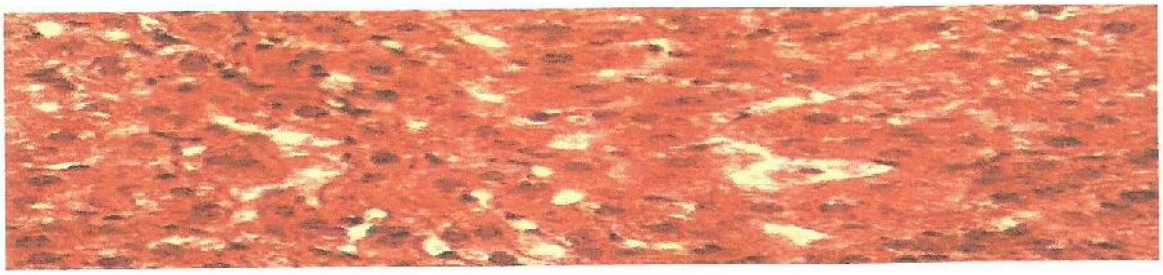

PLATE 2. DRUG CONTROL GROUP 3

PLATE 3 GROUP 4 CCl_4 TREATMENT AFTER 7 DAYS

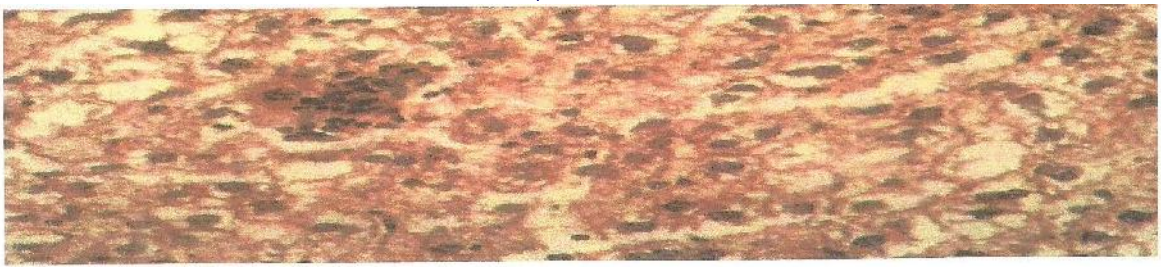

PLATE 4. GROUP 4 CCL_4 TREATMENT AFTER 14 DAYS

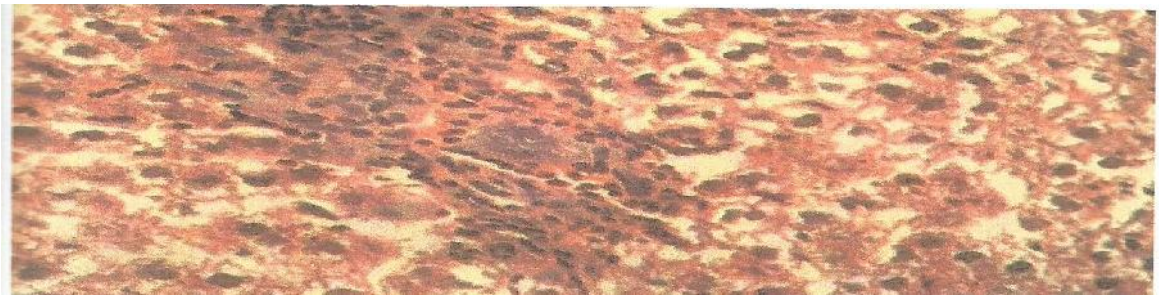

PLATE 5. GROUP 4 CCL_4 TREATMENT AFTER 21 DAYS

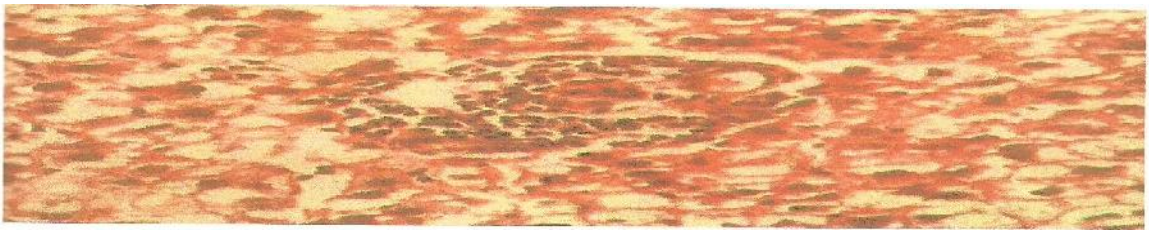

PLATE 6. GROUP 5, 50mg/kg TREATMENT AFTER 7 DAYS

PLATE 7. GROUP 5, 50mg/kg TREATMENT AFTER 14 DAYS

PLATE 8. GROUP 5, 50mg/kg TREATMENT AFTER 21 DAYS

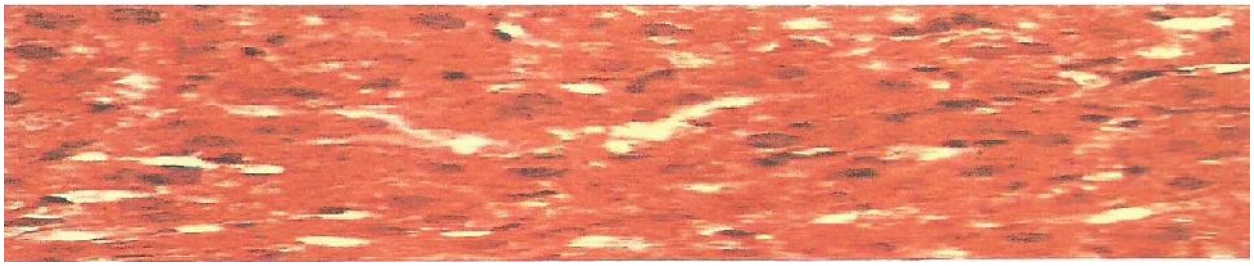

PLATE 9. GROUP 5, 50mg/kg TREATMENT AFTER 28 DAYS

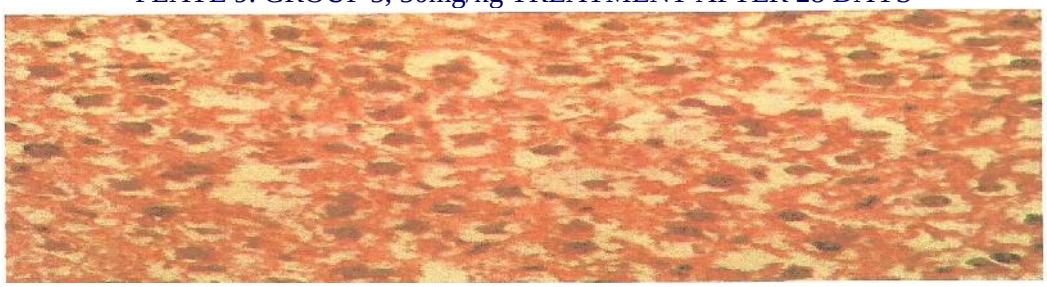

PLATE 10. GROUP 6, 100mg/kg TREATMENT AFTER 7 DAYS

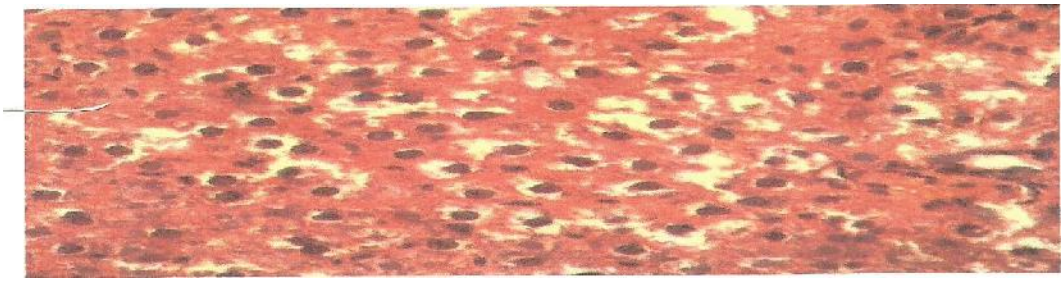

PLATE 11. GROUP 6, 100mg/kg TREATMENT AFTER 14 DAYS

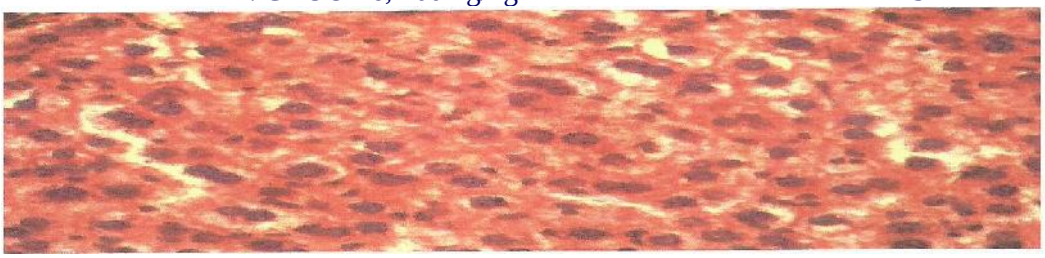

PLATE 12. GROUP 6, 100mg/kg TREATMENT AFTER 21 DAYS

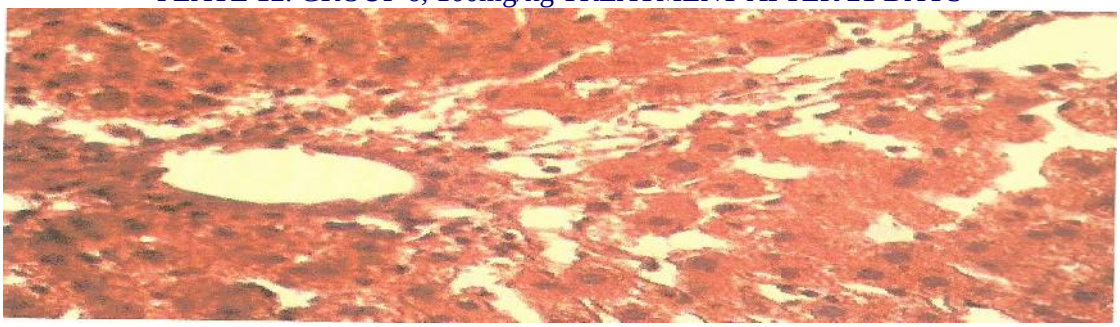

PLATE 13. GROUP 6, 100mg/kg TREATMENT AFTER 28 DAYS

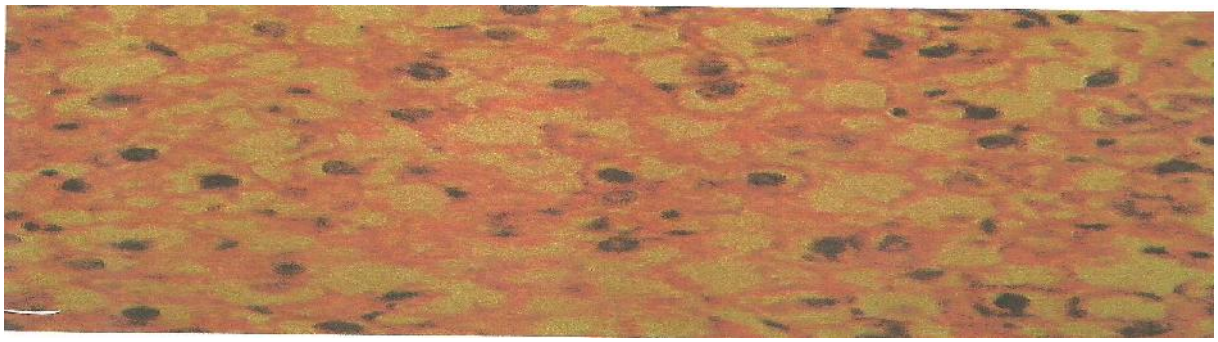

PLATE 14. GROUP 7, 200mg/kg TREATMENT AFTER 7 DAYS

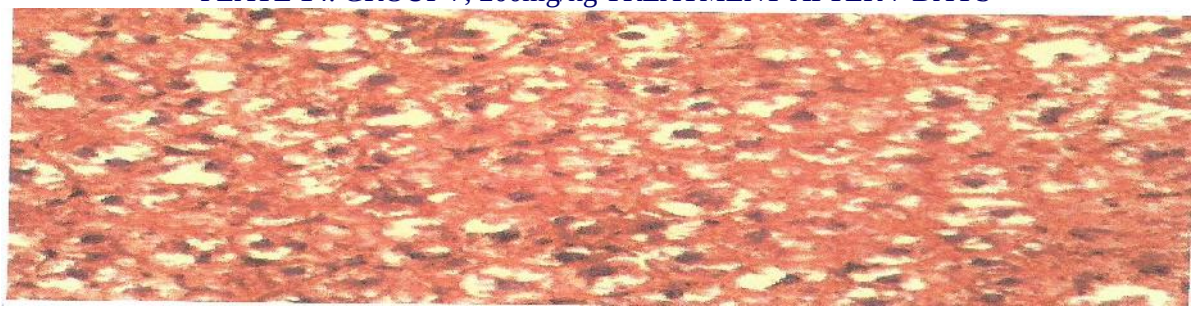

PLATE 15. GROUP 7, 200mg/kg TREATMENT AFTER 14 DAYS

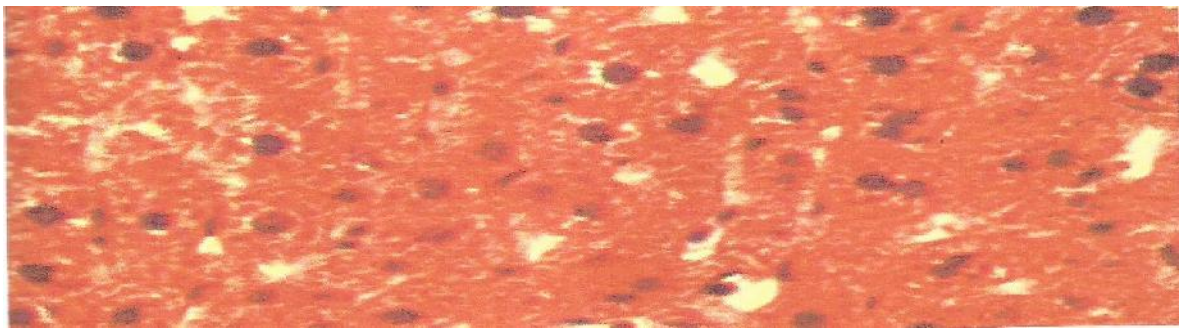

PLATE 16. GROUP 7, 200mg/kg TREATMENT AFTER 21 DAYS

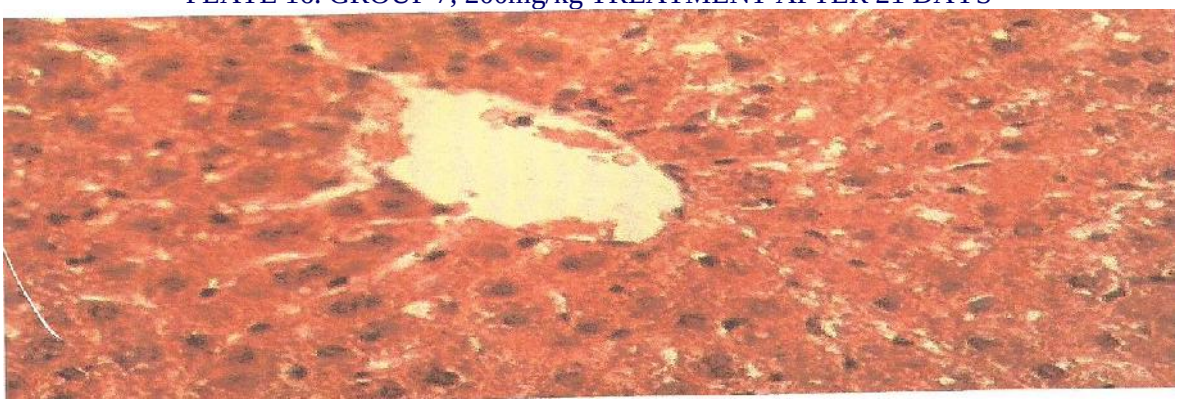

PLATE 17. GROUP 7, 200mg/kg TREATMENT AFTER 28 DAYS

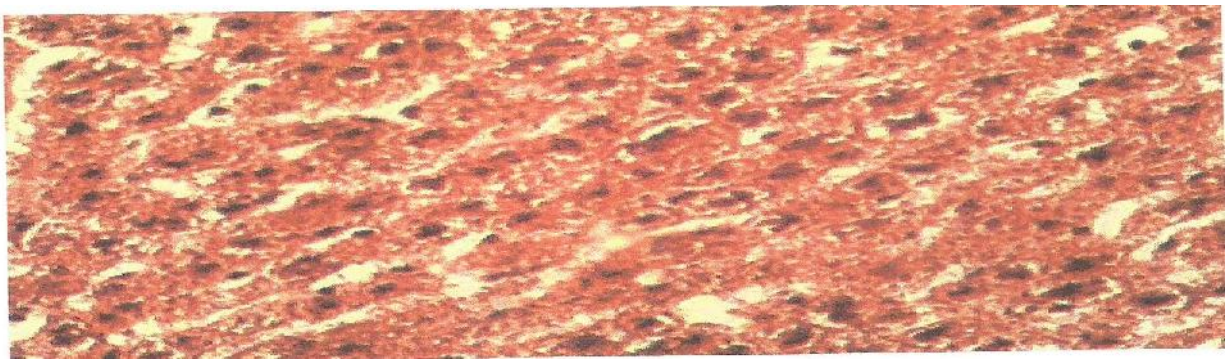

PLATE 18. GROUP 8, 300mg/kg TREATMENT AFTER 7 DAYS

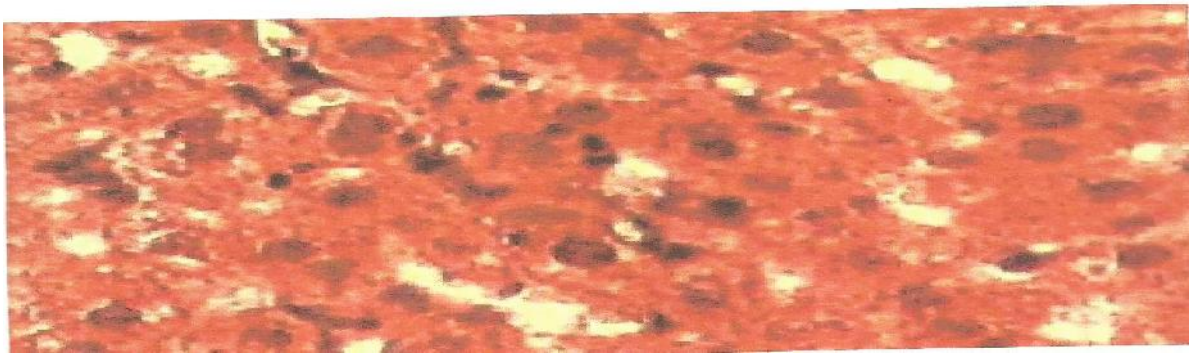

PLATE 19. GROUP 8, 300mg/kg TREATMENT AFTER 14 DAYS

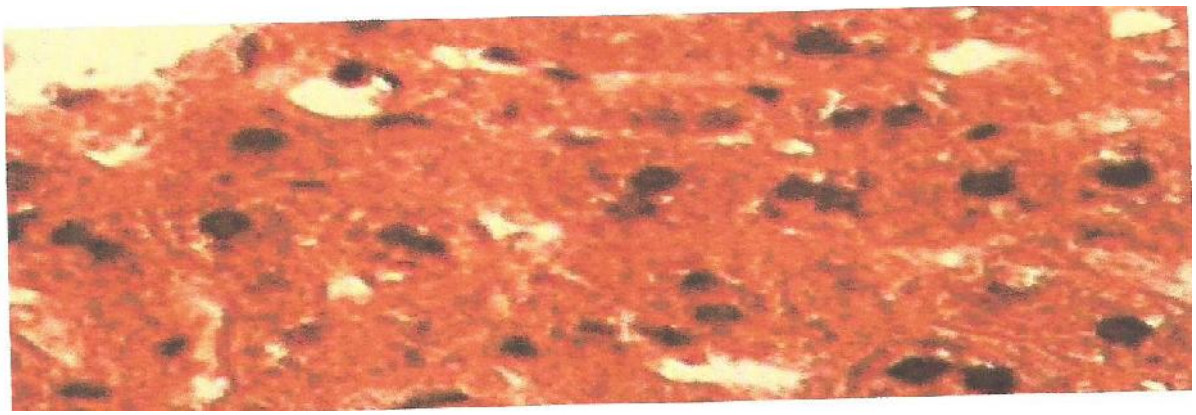

PLATE 20. GROUP 8, 300mg/kg TREATMENT AFTER 21 DAYS

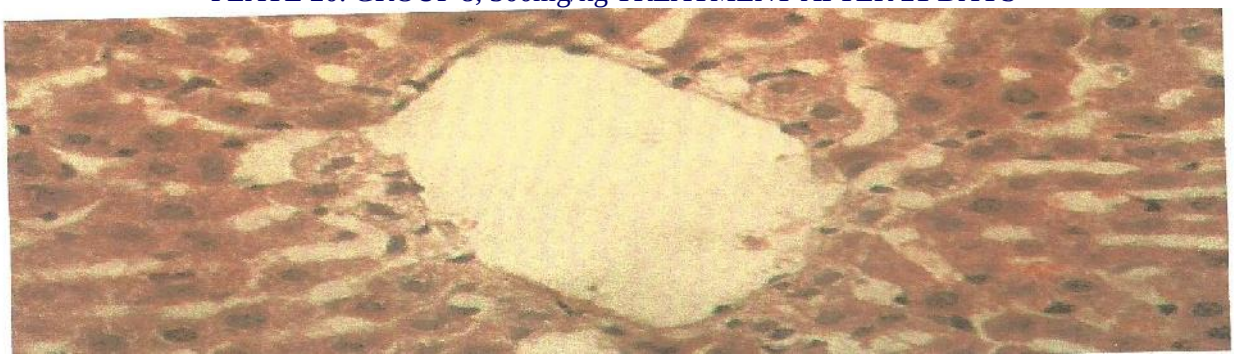

PLATE 21. GROUP 8, 300mg/kg TREATMENT AFTER 28 DAYS

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