Hepatoprotective Potential of Ethanol Leaf Extract of *Launaea taraxacifolia* Willd. (Asteraceae) on Carbon Tetrachloride-induced Liver Toxicity in Sprague-Dawley Rats

1,2James AB, 3Olasore HSA, 2Babalola OM, 3Ikujuni AP, 2Magbagbeola OA
1Department of Biochemistry and Nutrition, Nigerian Institute of Medical Research, Yaba, Lagos, Nigeria.
2Department of Biochemistry, College of Medicine, University of Lagos, Lagos, Nigeria.

Corresponding Author
OA Magbagbeola
Department of Biochemistry, College of Medicine, University of Lagos, Lagos, Nigeria.
E-mail: omagbagbeola@unilag.edu.ng; Tel.: +2348034378974

ABSTRACT

Background: *Launaea taraxacifolia* Willd (Asteraceae), commonly called wild lettuce, is a vegetable used as food and for various folkloric medicinal purposes and has been traditionally claimed to have protective effects against toxicants.

Objective: This study assessed the potentials of *Launaea taraxacifolia* ethanol leaf (LTEL) extract pre-treatment in protecting the liver against carbon tetrachloride (CCl$_4$)-induced toxic damage.

Method: Adult Sprague-Dawley rats were pre-treated orally with LTEL extract (100 and 500 mg/kg) and silymarin at 50 mg/kg body weight for 7 days. A single dose of CCl$_4$ (3 ml/kg body weight) was administered to the rats orally. After the treatment period, serum levels of hepatic enzymes and antioxidants were assayed. Histopathological examination of liver sections was done.

Results: Pre-treatment of rats with LTEL extract significantly (p<0.05) attenuated CCl$_4$-induced acute increase in serum aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) levels compared to the group that received CCl$_4$ only. Reduced glutathione (GSH) was significantly (p<0.05) increased in the pre-treatment groups while malondialdehyde (MDA) was significantly (p<0.05) lower in these groups. No significant (p>0.05) change in catalase (CAT) and superoxide dismutase (SOD) was observed in all the groups. Histologic examination revealed the protective effect of silymarin and the extract on the hepatocyte architecture.

Conclusion: This study suggests that *Launaea taraxacifolia* ethanol leaf extract possesses hepatoprotective property against CCl$_4$-induced liver injury through attenuation of oxidative stress.

Keywords: *Launaea taraxacifolia*, wild lettuce, carbon tetrachloride, hepatotoxicity, silymarin, antioxidants.

INTRODUCTION

Hepatic diseases such as fatty liver, alcoholic and non-alcoholic steatosis, hepatitis viral infections and hepatocellular carcinoma are trending as principal causes of morbidity and mortality worldwide (1–4). The liver is involved in a variety of important defence and metabolic functions such as detoxification, gluconeogenesis and clearance of xenobiotics, as well as secretion of pro-inflammatory cytokines (5,6). These functions predispose it to xenobiotic-induced injury. Liver injury is associated with cell death, membrane damage, decreased level of reduced glutathione (GSH) and elevated levels of serum markers of liver damage such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) (7).

Carbon tetrachloride (CCl$_4$)-induced experimental intoxication is a commonly used model for liver injury in laboratory animals (8,9). Carbon tetrachloride is a simple molecule which when administered to a variety of species, causes hepatic necrosis and fatty liver (9, 10). Although as a lipid-soluble compound, it is well distributed throughout the body, its major toxic effect is on the liver, irrespective of the route of administration. Chronic administration or exposure causes liver tumours, liver cirrhosis and also kidney damage (11). Carbon tetrachloride induced hepatotoxicity is characterized by hepatocellular membrane damage as a result of increased reactive oxygen species leading to lipid peroxidation and elevated plasma levels of hepatic enzymes and fatty degeneration (12).

*Launaea taraxacifolia* Willd. (Asteraceae) is widely used in western Nigeria and northern and eastern parts of Africa as vegetable and also for its medicinal properties (13, 14). It can be used as salad or freshly eaten or cooked in soups or sauces. Wild lettuce is listed among the underutilized and neglected vegetables in the western parts of Nigeria (15). Studies by Arawande *et al.* (16) in Nigeria and Adinortey *et al.* (17) in Ghana on the nutritional value and phytochemical characterization of *L. taraxacifolia* revealed that the extracts of its leaves are rich in potassium, calcium, magnesium, ascorbic...
acid, tannins, and flavonoids. Obi (18) and Arawande et al. (16) reported that apart from use as food, L. taraxacifolia leaves are widely used in the form of infusion for the treatment of several diseases. They reported properties such as antiviral effects, decreased cholesterol levels, regulation of dyslipidaemia and regulation of blood pressure.

Based on the folkloric reports of L. taraxacifolia, we sought to investigate its hepatoprotective potentials against carbon tetrachloride-induced hepatic damage.

MATERIALS AND METHODS

Chemicals

Protease inhibitor cocktail was purchased from MBL International (USA); olive oil (Goya Foods Inc., NJ, USA); liver function assay kits (Randox Laboratories, UK); silymarin, CCl₄ and other analytical reagents were purchased from Sigma Aldrich/Merck (Merck, Germany).

Plant Materials

Fresh green leaves and stems of L. taraxacifolia were obtained from Awolowo Market in Mushin area of Lagos, Nigeria. The plant was taken to the Department of Botany, University of Lagos, Nigeria for botanical identification and a voucher specimen (LUH 7055) was deposited in the herbarium of the Department.

Preparation of the Plant Extract

The leaves of the plant were air dried and milled into coarse powder using Gx160 grinding machine (Honda Domino, Japan). Dried plant powder (2.5 kg) was macerated in 70% ethanol for 72 h. The solution was filtered and the filtrate was concentrated by evaporation at 40°C using a rotary evaporator (Rotavapor, Buchi, Switzerland). The percentage yield of the extract was calculated to be 31.8%.

Experimental Animals

A total of thirty male Sprague-Dawley rats (Rattus norvegicus) weighing between 90-120 g were obtained from the Nigerian Institute of Medical Research, Yaba, Lagos, Nigeria. The animals were acclimatized in the Animal House of the Nigerian Institute of Medical Research, Yaba, Lagos, Nigeria. The animals were divided into groups as presented in Table 1. They were kept in standard clean cages with free access to rat feed (Ladokun Feeds Ltd., Ibadan, Nigeria) and drinking water. All animal experiments were conducted in compliance with the National Academy of Sciences Guidelines for Care and Use of Laboratory Animals (19). The study was conducted in the Department of Biochemistry, College of Medicine, University of Lagos, Nigeria.

Experimental Design

This study was designed based on an iteration of Mohammad et al. (20) to pre-treat the animals with LTEL prior to the administration of CCl₄. Animals were divided into groups as presented in Table 1.

The above pre-treatment lasted for seven days. On the seventh day, each animal in Groups II - V received 3 ml/kg b.w. of a fresh mixture of 30% CCl₄ dissolved in olive oil (vehicle), an hour after the administration of the last dose of the pre-treatment drug or extract. Rats in Group I were administered 3 ml/kg olive oil (orally) and served as the vehicle control. All animals irrespective of the group had access to standard diet all through the study.

Blood and Liver Collection

After 24 h, blood samples were collected into plain sterile tubes by puncturing the retro-orbital plexus and allowed to coagulate to obtain serum while the animals were subsequently sacrificed by cervical dislocation. Sera were separated by centrifugation at 3,000 rpm for 10 min. Rat livers were quickly excised and perfused with chilled 1.15 % (w/v) potassium chloride (KCl) solution in order to remove all traces of haemoglobin. The livers were blotted dry, weighed and small sections were preserved in 10% formal-saline for histopathological evaluation, while the remaining sections were used for chemical assays.

Preparation of Liver Homogenates

Excised and perfused liver samples of an approximate weight of 9.5 g in 0.25 M sucrose solution containing protease inhibitor cocktail (0.5 mM 4-(2-Aminoethyl)benzenesulfonyl fluoride hydrochloride (AEBSF), 0.3 μM aprotinin, 10 μM bestatin, 10 μM E-64, 10 μM leupeptin, and 50 μM Ethylenediaminetetraacetic acid (EDTA)) were homogenized with a dounce homogenizer on ice. Liver homogenates were transferred into 2 ml eppendorf tubes and centrifuged for 20 min at 700 x g for 20 min and the supernatants were further centrifuged at 10,000 x g for 20 min and the supernatants were stored at –80°C (21,22).

Serum Liver Marker Enzymes Assay

To assess the liver function of the rats, the activities of hepatic marker enzymes aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) were determined in the sera of the study animals.

Assay of Aspartate Aminotransferase

Aspartate aminotransferase was determined using the method of Reitman and Frankel (23). Serum (30 μl) was mixed with 100 μl of substrate. AST substrate was prepared using 2 mM α-ketoglutarate and 200 mM aspartate in 0.1 M, pH 7.4

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre-Treatment (7 Days)</th>
<th>CCl₄ Toxicant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>1 ml/kg b.w. distilled water orally</td>
<td>No CCl₄ (3 ml/kg b.w. olive oil vehicle only)</td>
</tr>
<tr>
<td>Group II</td>
<td>1 ml/kg b.w. distilled water orally</td>
<td>3 ml/kg b.w. 30% CCl₄ orally</td>
</tr>
<tr>
<td>Group III</td>
<td>50 mg/kg b.w. silymarin orally</td>
<td>3 ml/kg b.w. 30% CCl₄ orally</td>
</tr>
<tr>
<td>Group IV</td>
<td>100 mg/kg b.w. LTEL orally</td>
<td>3 ml/kg b.w. 30% CCl₄ orally</td>
</tr>
<tr>
<td>Group V</td>
<td>500 mg/kg b.w. LTEL orally</td>
<td>3 ml/kg b.w. 30% CCl₄ orally</td>
</tr>
</tbody>
</table>

Table 1: Experimental Design for Animal Pre-treatment and Dosing with CCl₄
phosphate buffer. The mixture was incubated at 37°C for 60 min. Subsequently, 1 mM 2,4-dinitrophenylhydrazine reagent (100 µl) was added and after 20 min, 1 ml of 0.4 N sodium hydroxide was added and incubated for exactly 30 min. Absorbance was read at 490 nm (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom).

**Assay of Alanine Aminotransferase**

The same assay method described for AST was used with the exception that the AST substrate was replaced with ALT substrate (2 mM α-ketoglutarate and 200 mM alanine in 0.1 M, pH 7.4 phosphate buffer). The mixtures were incubated at 37°C for 30 min. Subsequently, 1 mM 2,4-dinitrophenylhydrazine reagent (100 µl) was added and after 20 min, 1 ml of 0.4 N sodium hydroxide was added and incubated for exactly 30 min. Absorbance was read at 490 nm (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom).

**Assay of Alkaline Phosphatase**

Alkaline phosphatase was assayed using the method described by Bessey et al. (24) as modified by Wright et al. (25) using Randox standard kits supplied by Randox Laboratories (United Kingdom) which is based on measurement of the rate of hydrolysis of phosphate esters. The substrate, p-nitrophenylphosphate (pNPP) is hydrolysed by alkaline phosphatase from the sample in the presence of magnesium ions, to form p-nitrophenol which confers yellowish colour on the reaction mixture. Its intensity was monitored at 405 nm to give a measure of enzyme activity. In a cuvette, 10 µl of serum was mixed with 500 µl of the reagent. The initial absorbance was read at 405nm, and subsequently over 3 min (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom). The mean absorbance per min was used in the calculation: ALP activity (IU/l) = 2742 × ΔA 405 nm/min. Where: 2742 = Extinction coefficient; ΔA 405 nm/min = change in absorbance per min for the liver lysate.

**Assays for Antioxidant Status of Liver Homogenate**

**Determination of Liver Catalase (CAT) Activity**

Hepatic tissue catalase activity was determined according to the method of Chance and Meekly (26) by measuring the decrease in absorbance at 240 nm due to the decomposition of H₂O₂ in UV. The assay was carried out by mixing 2.5 ml of 50 mmol phosphate buffer (pH 5.0), 400 µl of 5.9 mmol H₂O₂ and 100 µl of liver homogenate supernatant. Changes in absorbance of the reaction solution at 240 nm were determined after one minute (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom). One unit of CAT activity was defined as an absorbance change of 0.01 as units/min/mg protein.

**Determination of Liver Superoxide Dismutase (SOD) Activity**

Superoxide dismutase activity of liver tissue was estimated by the method of Kakkar et al. (27, 28). The assay was done by mixing 100 µl of phazemine methosulphate (186 µmol), 1.2 ml of sodium pyrophosphate buffer (0.052 mmol; pH 7.0), 300 µl of supernatant after centrifugation (1500 × g for 10 min followed by 10000 × g for 15 min) of liver homogenate was added to the reaction mixture. Enzyme reaction was initiated by adding 200 µl of nicotinamide adenine dinucleotide (NADH; 780 µmol) and stopped after 1 min by adding 1 ml of glacial acetic acid. Amount of chromogen formed was measured by reading the colour intensity at 560 nm (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom). Results were expressed in units/ min/mg protein.

**Determination of Lipid Peroxidation**

Malondialdehyde was determined in the liver lysates using the method of Buege and Aust (29). MDA reacts with thiobarbituric acid reactive substance (TBARS) to produce a red coloured complex which has peak absorbance at 532 nm (30). The reaction mixture in a total volume of 1.0 ml contained 0.58 ml phosphate buffer (0.1 mol; pH 7.4), 0.2 ml homogenate supernatant, 0.2 ml ascorbic acid (100 mmol), and 0.02 ml ferric chloride (100 mmol). The reaction mixture was incubated at 37°C in a shaking water bath for 1 h. The reaction was stopped by addition of 1.0 ml 10% trichloroacetic acid. Following addition of 1.0 ml 0.67% thiobarbituric acid, all the tubes were placed in boiling water bath for 20 min and then shifted to crushed ice-bath before centrifuging at 2500 × g for 10 min. The amount of TBARS formed in each of the samples was assessed by measuring optical density of the supernatant at 535 nm using spectrophotometer against a reagent blank (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom). The results were expressed as µmol TBARS/ml of liver lysate.

**Determination of Reduced Glutathione Activity**

The assay is based on the method described by Rahman et al. (31) in which reduced glutathione (GSH) reacts with 5,5–Dithiobis-(2-nitrobenzoic acid) (DTNB/Ellman’s reagent) to produce TNB chromophore. The product has a maximal absorbance at 412 nm due to oxidized glutathione-TNB adduct (GS-TNB). The rate of formation of TNB, measured at 412 nm, is proportional to the concentration of GSH in the liver lysate. Briefly, liver clear lysate (0.5 ml) was precipitated with 0.5 ml of 4% sulfosalicyclic acid on ice for 1 h. Precipitates were sedimented at 1200 × g for 20 min at 4°C and 0.1 ml supernatant was added to a reaction mix containing 2.7 ml phosphate buffer (0.1 mol; pH 7.4) and 0.2 ml DTNB (100 mmol). The yellow colour developed was read immediately at 412 nm (T70 UV/VIS Spectrophotometer, PG Instruments, United Kingdom). It was expressed as µmol GSH/ml liver lysate.

**Histology**

The liver histology was processed according to the method described by Adeneye et al. (32). The animals were sacrificed on final day of CCl₄ administration. Liver was harvested and subsequently washed, fixed, sectioned and stained with haematoxylin and eosin dye for examination under light microscope (Leica DMLB2, Leica Microsystems, Germany).

**Statistical Analysis**

The experimental results are presented as mean ± S.E.M. (standard error of mean) and median interquartile ranges. One-way Analysis of Variance (ANOVA) for intergroup comparison
(group effect) was used and p<0.05 considered as significant using a non-parametric tool (Kruskal-Wallis) for post-hoc analysis. All analyses were carried out using R-programming with the Hmisc and ggpubr packages for analysis and the creation of boxplots (33–35).

RESULTS

Biomarkers of Liver and Kidney Function

The effect of pre-treatment with LTEL extract on serum AST, ALT and ALP activities in CCl$_4$-intoxicated rats are shown in Figures 1–3. Intoxication of rats with CCl$_4$ caused hepatocellular damage as shown by significant (p<0.05) elevation in the activities of serum AST, ALT and ALP in the untreated group compared to control. However, there was a significant (p<0.05) attenuation of the levels of AST and ALT only in rats pre-treated with 500 mg/kg b.w. extract compared with the CCl$_4$-only group. A significant (p<0.05) dose-dependent decrease in the level of ALP in rats treated with the extract was observed.

Biomarkers of Oxidative Damage

Table 2 shows the effect of pre-treatment with LTEL extract on liver antioxidant enzyme system, GSH level and lipid peroxidation of CCl$_4$-intoxicated rats. Carbon tetrachloride administration resulted in significant (p<0.05) decrease in the activities of CAT and SOD as compared to control rats. There were no significant (p>0.05) changes in the activities of SOD and CAT enzymes in rats treated with the extract when compared with the CCl$_4$-treated group. Carbon tetrachloride intoxication resulted in a significant (p<0.05) decrease in the levels of GSH. Pre-treatment with 500 mg/kg b.w. of the extract resulted in a significantly (p<0.05) higher level of GSH compared to the CCl$_4$-only treated group. Also, there was significant (p<0.05) increase in the level of MDA in the group treated exclusively with CCl$_4$. Pre-treatment with the extract resulted in significantly lower levels of MDA at both doses compared with the CCl$_4$-treated group.

Table 2: Effect of LTEL on GSH and MDA levels, and SOD and CAT Activities in All Experimental Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>GSH (µmol/mL)</th>
<th>SOD (µmol/min/mg pro)</th>
<th>CAT (µmol/min/mg pro)</th>
<th>MDA (µmol/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative control</td>
<td>33.37±1.19</td>
<td>7.26±0.86</td>
<td>15.52±1.33</td>
<td>2.24±0.53</td>
</tr>
<tr>
<td>CCl$_4$</td>
<td>25.53±1.09</td>
<td>1.86±0.49</td>
<td>13.58±0.85</td>
<td>4.69±0.35</td>
</tr>
<tr>
<td>Silymarin (50 mg/kg) pre-treatment + CCl$_4$</td>
<td>31.11±1.09*</td>
<td>2.49±0.25</td>
<td>15.37±0.97</td>
<td>2.18±0.58***</td>
</tr>
<tr>
<td>LTEL (100 mg/kg) pre-treatment + CCl$_4$</td>
<td>27.24±1.06</td>
<td>2.13±0.22</td>
<td>16.89±1.20</td>
<td>1.88±0.48***</td>
</tr>
<tr>
<td>LTEL (500 mg/kg) pre-treatment + CCl$_4$</td>
<td>30.84±1.17*</td>
<td>4.07±0.14</td>
<td>18.52±1.74</td>
<td>1.54±0.55***</td>
</tr>
</tbody>
</table>

Values are presented as mean ± S.E.M. (n=5). *p<0.05, **p<0.01, ***p<0.001 as compared to CCl$_4$ toxicant only group.
Histopathological Assessment of the Liver

Plate 1(a-h) shows the photomicrographs of the hepatocytes. The protective effects of silymarin (50 mg/kg) and the LTEL extract (100 and 500 mg/kg) on the hepatocytes are evident. The CCl₄ administration caused a necrotic effect on the hepatocytes with the loss of cellular architecture as evident with the presence of ghost hepatocytes (a and b). The pre-treatment with silymarin (c and d) and the extract (e to h) prevented hepatocyte damage as evident in the normal hepatocyte cellular architecture. However, there were some lipid droplets (steatosis) and lymphocytic infiltration indicating mild hepatocellular injury in the extract administered groups.

Plate 1: Haematoxylin and eosin staining photomicrographs of liver sections of study animals (V- central vein; H- hepatocytes; S- steatotic hepatocytes). (a) and (b) show extensive necrosis and ghost outlines; (c) and (d) show no evidence of hepatic injury despite CCl₄ intoxication in the sylimarin pre-treated group; (e) and (f) show the presence of moderate steatosis (S) and lymphocytic infiltration (L) in the 100 mg/kg LTEL pre-treated group; (g) and (h) show the presence of steatosis (S) and mild hepatic injury in the 500 mg/kg LTEL pre-treated group.

DISCUSSION

This study is part of a scientific investigation of a folkloric anti-poison claim of several alternative medical practitioners in the south-western region of Nigeria. In our study, we present the hepatoprotective data of LTEL in rats using CCl₄ as the toxicant. Liver enzymes (ALT, AST and ALP) are biochemical markers of acute hepatocellular damage. Their elevated serum levels reflect the degree of hepatocellular membrane damage and cause their leakage (36). This study shows that a single dose of carbon tetrachloride causes liver injury as indicated by the elevated levels of biochemical parameters such as ALT, AST and ALP in the group exposed to CCl₄ with no pre-treatment. This is an indication of hepatic damage caused by the metabolism of CCl₄. In liver injury, there is disruption in the transport function of hepatocytes, resulting in the leakage of plasma membrane, thereby causing leakage of these enzymes leading to increase in their serum levels (37). However, the results showed that pre-treatment with LTEL extract protected the animals against CCl₄-induced hepatic damage. This is evidenced by the significant decrease in the levels of serum AST and ALT (at 500 mg/kg) and ALP at both doses.

The hepatoprotective effect of L. taraxacifolia is in agreement with the work of Kuatsienu et al. (38) who used cisplatin as the hepatotoxicant and showed that LTEL is not toxic at a dose of 1000 mg/kg. This protective effect of LTEL extract could be a function of its rich phytochemical constituents. Phytochemical analysis of L. taraxacifolia as reported by Adinortey et al. (17) confirmed the presence of several phytochemicals (e.g. flavonoids, tannins and saponins) essentially associated with antioxidant and/or hepatoprotective effects (16). Reports have shown that phenolic compounds possess diverse pharmacological effects (i.e. antioxidant and hepatoprotective) (39, 40).

Several notable pathological conditions associated with
CCl₄-induced hepatotoxicity, namely fatty liver, cirrhosis and necrosis, have been said to result from the formation and effect of reactive intermediates (i.e. trichloromethyl free radicals, CCl₃⁺) metabolized by the mixed function cytochrome P450 in the endoplasmic reticulum. These free radicals covalently bind to the unsaturated fatty acids of membrane lipids and thereby result in the formation of chloroform and lipid radicals which initiate the chain reactions of lipid peroxidation in the hepatic cells, causing liver damage.

Although production of active radicals, including oxygen free radicals and non-oxygen free radicals, is a well known phenomenon in normal metabolic process, excessive free radicals known as reactive oxygen species (ROS) pose a great threat to various biological molecules through lipid peroxidation and DNA damage. Such damage results in various diseases such as cancer, hepatic injury, arteriosclerosis, and reperfusion injury.

Superoxide dismutase and catalase are primary enzymes involved in mopping up of reactive oxygen species such as superoxide radical and hydrogen peroxide (44). The decreased SOD and CAT levels in the serum of rats exclusively treated with CCl₄ is an acute biochemical response to CCl₄-induced oxidative stress. There was a non-significant increase in the activities of these enzymes in rats pre-treated with the extract. Koukoui et al. (40) reported a similar result in their chronic hepatoprotective study on LTEL extract using cisplatin as the hepatotoxicant.

Glutathione is a tripeptide antioxidant found in liver. This compound has been suggested to protect the thiol groups of proteins from oxidation by free radicals. The metabolism of CCl₄ leads to the formation of CCl₃⁺, which easily conjugate with GSH. After a significant depletion of GSH, this reactive metabolite binds to cellular macromolecules covalently. When the hepatic GSH depletion reaches a threshold level, lipid peroxidation develops and severe cellular damage is produced. A marked depletion in the level of GSH observed in the group treated exclusively with CCl₄ is an evidence of hepatocellular injury caused by CCl₄. However, rats pre-treated with 500 mg/kg LTEL extract were protected against the effect of CCl₄. This is evidenced in the significant higher levels of GSH in this group compared with the CCl₄-treated animals revealed a significant hepatic damage of the liver. However, animals pre-treated with L. taraxacifolia ethanol leaf extracts showed protective effects, especially at 500 mg/kg.

CONCLUSION
This study has demonstrated that Launaea taraxacifolia ethanol leaf extract ameliorates carbon tetrachloride-induced hepatic injury, which may be through its antioxidant activities by scavenging free radicals to prevent oxidative stress and inhibit lipid peroxidation.

ACKNOWLEDGEMENTS
The authors wish to acknowledge Mr. A. Magbagbeola for providing the folkloric information on Launaea taraxacifolia and the opportunity to collect the plant specimen from his garden for pilot studies.

CONFLICT OF INTEREST
The authors declare no conflict of interest.

REFERENCES


