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Original Research Article

Free Radical Scavenging Effect of Aqueous Extract of *Salacia nitida* Root Bark against Alcohol-Induced Hepatic Stress in Wistar Rats

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ABSTRACT

Alcoholic decoction of *Salacia nitida* root bark is used for the treatment of malaria, and prolonged alcohol dependence causes an imbalance in antioxidants. This study evaluated the free radical scavenging effect of aqueous extract of *Salacia nitida* root bark against ethanol-induced hepatic stress in rats. Thirty-six rats (210-290 g) divided into 6 groups with 6 rats each were used. Rats in groups A were given physiological saline, B given ethanol daily for 18 days without treatment and C–F given ethanol for 18 days then treated with 800, 1000, 1200 mg/kg of the extract, and 25 mg/kg of silymarin daily for further 18 days. Liver homogenate was prepared at the end of the experimental period and used for analyses of malondialdehyde and some antioxidant enzymes. Data were statistically analyzed using ANOVA and results considered significant at 95% confidence level ($p < 0.05$). Results showed that *Salacia nitida* root bark extract was safe and also, showed that treatments of ethanol-induced liver injury in groups C – E rats with the extract significantly ($p < 0.05$) decreased the levels of malondialdehyde, glutamyl S-transferase, and increased activities of catalase, superoxide dismutase, glutathione peroxidase, and glutathione reductase, while the level of glutathione was non-significantly ($p > 0.05$) raised compared to rats in group B. Therefore, aqueous extract of *Salacia nitida* root bark ameliorated ethanol-induced oxidative stress in hepatic tissues of the experimental rats, which may play a significant role in the manufacture of antioxidants that could be used to manage liver problems.

***Keywords*:** Antioxidants, Liver injury, Oxidative stress, Reactive oxygen species, Root bark, *Salacia nitida*.

Introduction

The concentration of alcohol in the body over time determines its effects on the various organs of the body. Prolonged alcohol dependence is toxic to the body and causes metabolic disturbances which include an imbalance in the pro- and antioxidant levels in the body.[1,2] Alcohol is oxidized in the liver to acetaldehyde by alcohol dehydrogenase, cytochrome P450 2E1 (CYP2E1), and catalase. The acetaldehyde induces oxidative stress or contributes to other mechanisms that promote oxidative damage.[3,4] Oxidation of alcohol by the microsomal ethanol oxidizing system brings about the production of more free radical species that cause liver and tissue injuries via peroxidation of cell membrane lipids,[5] and alteration of the antioxidant status.[6,7] Excessive intake of alcohol increases the intestinal permeability to other substances including bacterial endotoxins such as lipopolysaccharides,[8] which initiate the generation of reactive oxygen species (ROS) and liver damage.[9] Medicinal plant parts, including root bark of *Salacia nitida* are used for the treatment of hepatic problems by the low socio-economic class since treatment options are limited.[10] *Salacia nitida* Linn Benth is a member of the Celastraceae family. In Southern Nigeria, alcoholic decoctions of *S. nitida* root bark are orally taken by people as a cure for liver problems and for treatment of malaria. The phytochemical analysis of *S. nitida* root bark revealing the presence of spartein, lunamarine, ribalinidine, tannins, sapogenin, phenol, epicatechin, catechin, rutin, kaempferol, anthocyanin, and phytate has been reported.[11] Therefore, this study evaluated the free radical scavenging effect of aqueous extract of *S. nitida* root bark against alcohol-induced hepatic stress in Wistar albino rats.

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Materials and Methods

Collection of plant materials

Salacia nitida was collected in February 2017, from Wiiluere farm, Nyogor-Beeri in Khana Local Government Area of Rivers state, Nigeria. It was identified and authenticated by Dr. N. L. Edwin-Wosu of the Department of Plant Science and Biotechnology, University of Port Harcourt, Choba, Rivers State, Nigeria, and specimen with voucher number UPHV-1033 deposited at the University herbarium. Plants were uprooted with a spade and the roots removed, transported to the Department of Biochemistry laboratory, University of Port Harcourt, washed and air-dried under shade.

Experimental animals

Thirty-six (36) healthy rats weighing 210 – 290 g of mixed-sex, procured from the Department of Biochemistry, University of Port Harcourt, were used for the study. The rats were maintained in plastic cages, under standard housing conditions of humidity (49– 70%), temperature (34 ± 1°C), and 12 hours light-darkness cycle, with free access to clean water *ad libitum* and grower's marsh feed (Premier Feed Mill Co. Nig. Ltd) procured from mile one market, Port Harcourt, for10 days. Physical appearances and feeding behaviors of

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the rats were used to confirm the health status of the experimental rats. The study was done according to the United States National Institute of Health (NIH) "Principles for Laboratory animals use and care",[12] as approved by the University of Port Harcourt ethics committee on laboratory animals (UPH/BCH/AEC/2016/025C).

Chemicals and reagents
Chemicals and reagent kits used for this study were of analytical grades. Ethanol (40%) was purchased from Austin Labs and store, Alakahia, Port Harcourt, Rivers State, Nigeria, and silymarin provided by Micro Labs Ltd (India).

Preparation of plant extract
Root barks were removed, reduced to smaller bits with a machete and were air-dried under shade for10 days. The dried root barks were pulverized using hand grinding machine (Corona-16D). Extraction was done by soaking 500 g of powdered root materials in 5.5 liters of hot water for about 3 hours and stirred. The mixture was filtered with filter paper (Whatman no. 1) and the filtrate evaporated to dryness with a rotary evaporator (Heidolph 4000, Schwabach, Germany) carefully regulated at a low temperature (34^{o}C) and with additional traps (porcelain boiling chips). The dried *S. nitida* root bark extract was kept in the refrigerator at 4^{o}C until required for use.

Acute toxicity test
The acute toxicity test (LD_{50}) of the extract of *S. nitida* root bark was studied using a modified Lorke method, [13] with 24 rats divided into 6 groups of 4 rats each. The rats were fasted overnight before treatments and 2 ml each of 5, 100, 500, 1000, 3000, and 5000 mg/kg of the extract were orally given to rats in groups 1 to 6 accordingly. They were monitored for signs of toxicity including mortality for 72 hours post administration. LD_{50} was calculated by taking the square root of the product of the highest dose that could not cause mortality and the least dose that could cause mortality.

Experimental design and procedure
The study was conducted with 36 Wistar rats that were randomly divided into 6 separate groups A – F, with 6 rats per group. Group A received physiological saline *per os* daily for 36 days and serves as the normal control (NC). Group B received ethanol (40%) *per os* daily for 18 days followed by physiological saline *per os* daily for further 18 days, and serves as the induction control (IC). Groups C to E received ethanol (40%) *per os* daily for 18 days followed by 800, 1000, and 1200 mg/kg body weight/day of extract of *S. nitida* root bark *per os* for further 18 days, and were used as extract treated (ET1-3). While group F received ethanol (40%) *per os* daily for 18 days followed by 25 mg/kg bodyweight/day of silymarin *per os* for further 18 days and was used as the reference control (RC). On day 37, animals were anaesthetized with formalin, sacrificed by cervical dislocation and the liver used for evaluations of lipid peroxidation (LPO), reduced glutathione (GSH), and some antioxidant enzymes, which include catalase (CAT), superoxide dismutase (SOD), glutathione S-transferase (GST), glutathione perioxidase (GP), and glutathione reductase (GR). Standard methods were used to determine the dosages and volume of the extract and drug (silymarin) used.[14] Treatments of animals with alcohol, the plant extract, and standard drug were done using a metal gavage.

Determinations of hepatic oxidative stress parameters.
Preparation of liver homogenate
The liver homogenate was prepared by manual grinding of 8 g of the liver in normal saline using mortar with pestle. The crude homogenate was sieved and the filtrate acidified with 130 µl of 25 % trichloroacetic acid (TCA). Then the precipitated protein homogenate was centrifuged at 4000 rpm for 10 minutes and allowed to stand for 5 minutes. The supernatant was used for the assessment of malondialdehyde (MDA), GSH, CAT, SOD, GST, GP, and GR.

Estimation of LPO level
The assay for membrane LPO was performed using the method described by Wright *et al*.[15] The supernatant (0.4 ml) from homogenate was added to 1.6 ml of tris-KCl buffer. Then 0.5 ml of 30 % TCA and 0.5 ml of 0.75 % thiobarbituric acid (TBA) was added and this was mixed properly. The setup was incubated for 15 minutes in a boiling water bath (BG-7311, Electrochemical, England) and then placed on ice. The test tubes were centrifuged at 3000 rpm for 10 minutes and the concentration of malondialdehyde (MDA) read at 532 nm with a UV spectrophotometer (Surgispec SM-23D, Surgifield Medical, England) against the blank containing 0.4 ml of distilled water. The concentration of MDA = absorbance of test sample – absorbance of blank/molar extinction coefficient (1.56×10^{-5} M^{-1} cm^{-1}), and result expressed as nmol MDA/100g tissue.

Estimation of reduced glutathione (GSH) level
The level of reduced GSH was measured according to Jollow *et al* method.[16] The supernatant (0.2 ml) from the liver homogenate was added to 1.8 ml of distilled water and mixed with 3 ml of precipitating agent (4 % sulfosalicylic acid). The setup was made to stand for about 7 minutes and then spun for 10 minutes at a speed of 1200 rpm. 1 ml of the resulting supernatant was mixed with 4 ml of 0.1 M phosphate buffer plus 0.5 ml of Ellman's reagent (5,5-dithio-bis-[2-nitrobenzoic acid]), DTNB and maintained for 15 minutes at ordinary room temperature. Absorbance was read at 412 nm with a UV spectrophotometer against the blank of 3 ml of distilled water. The GSH concentration was obtained from a standard curve prepared using concentrations that vary between 10 – 160 µmol, and results obtained were expressed as µmol GSH/min/mg tissue.

Measurement of catalase (CAT) activity
The activity of CAT was measured according to Claiborne method.[17] About 4 ml of 50 mM phosphate buffer was dispensed into a test tube plus 1 ml of 100 mM of hydrogen peroxide (H_2O_2) and 0.2 mL of supernatant from liver homogenate. The setup was then maintained for 2 min at 37^{o}C and the change in absorbance was read with a UV spectrophotometer at 240 nm. Calculation of catalase activity was done with a 43.591 $mol^{-1}cm^{-1}$ molar extinction coefficient of H_2O_2. A unit of catalase activities equals the quantity of protein changed to 1 µmol H_2O_2/min. Catalase activity was recorded as µmol H_2O_2/min/mg protein.

Measurement of superoxide dismutase (SOD) activity
The method of Misra and Fridovich was used to determine SOD activity.[18] The supernatant (100 µL) from the liver homogenate was mixed with 1000 µL of distilled water to make a 1:10 dilution, and 200 µl of the diluted supernatant added to 2.5 ml 0.05 M carbonate buffer (pH 10.2) and equilibrated in the UV spectrophotometer. Then 0.3 ml of fresh 0.3 mM epinephrine was added and mixed by inversion. In the blank, 0.2 ml of distilled water was dispensed, in which 2.5 ml of carbonate buffer and 0.3 ml of epinephrine were added. The SOD activity was read with a UV spectrophotometer at 480 nm and an increase in absorbance was monitored every 30 seconds for 2.5 min. Increase in absorbance/min = absorbance at 150 sec - absorbance at 30 seconds/150 seconds. The % inhibition = increase in absorbance x 100/blank. One unit of SOD activity = amount of SOD necessary to cause 50 % inhibition of oxidation of epinephrine to adrenochrome in 60 seconds. Results were expressed as Unit/mg protein.

Measurement of glutathione S-transferase (GST) activity
The method of Habig *et al* was used for measuring GST activity.[19] 30 µl of glutathione (1 mM) was dispensed into test tubes plus 150 µl of 1 mM 1-chloro-2,4-dinitrobenzene (CDNB) and 2.79 ml of 0.1 mM of phosphate buffer (pH 6.5), plus 30 µl of supernatant from homogenate, while 62.82 ml of phosphate buffer (pH 6.5), equal volume of other reagents and 30 µl of distilled water were used as blank. The test tubes were kept for 60 seconds, and changes in absorbance were read at 340 nm with a UV spectrophotometer. Enzyme activities were expressed as nmol CDNB/min/mg protein, using the molar extinction coefficient of 9.6×10^{3} M^{-1} cm^{-1}.

Measurement of glutathione peroxidase (GP) activity
The activity of GP was assessed according to Mohandas *et al* method.[20] Into test tubes were measured 0.1 ml of supernatant from liver homogenate, plus 1.49 mL of 0.1 mM phosphate buffer (pH 7.4), 0.1 ml of 1.0 mM ethylene diaminetetra-acetic acid (EDTA), 0.1 ml of 1 mM sodium azide, 0.05 ml of glutathione reductase (1 U/ml), 0.05 ml of 1 mM GSH, 0.1 ml of 0.2 mM reduced nicotinamide adenine

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dinucleotide phosphate (NADPH), and 0.01 ml 0.25 mM H_2O_2. The disappearance of NADPH was read with a UV spectrophotometer at 340 nm against the blank. The enzyme activity was expressed as nmol NADHP oxidized/min/mg protein, using a molar extinction coefficient of 6.22 x 10^3 M^{-1} cm^{-1}.

Measurement of glutathione reductase (GR) activity

Measurement of GR activity was done with the method of Carlberg and Mannervik.[21] The reaction mixture contained 1.65 ml of 0.1 mM phosphate buffer (pH 7.6), 0.1 ml of 0.5 mM ethylene diaminetetra-acetic acid (EDTA), 0.05 ml of 1.0 mM oxidizing NADPH plus 0.1 ml of supernatant from liver homogenate in a test tube, while distilled water was used as blank. The enzyme activity was obtained by measuring the disappearance of NADPH at 340 nm with a UV spectrophotometer against the blank. Results were expressed as nmol NADPH oxidized/min/mg protein using molar extinction coefficient of 6.22 x 10^3M^{-1} cm^{-1}.

Statistical analysis

Data were statistically analyzed using a one-way analysis of variance (ANOVA) with the SPSS statistical package version 22. Multiple comparisons were done using the Scheffe's post hoc test. Results were presented as mean ± standard error of the mean (SEM) and were considered significant at a 95% confidence level ($p < 0.05$).

Results and Discussion

The result for the acute toxicity test (LD_{50}) was 2236.1 mg/kg, and there was no sign of toxicity, including mortality observed in any of the treated animal group during the test even at a dose level of 5000 mg/kg of aqueous extract of *S. nitida* root bark. Also, the results of the study on the free radical scavenging effect of aqueous extract of *S. nitida* root bark against alcohol-induced hepatic stress in rats are displayed in figures 1 - 6. From figure 1, it was observed that oral administration of 40 % ethanol to rats in the induction control (group B) significantly increased ($p < 0.05$) the level of MDA by 62.96 % compared to that of the normal rats in the normal control (group A). Also, the level of GST in the induction control (IC) rats in group B that were given 40 % ethanol was non-significantly ($p > 0.05$) raised compared to the normal control (NC) rats in group A. Treatments of alcohol-induced hepatic stress in the experimental rats in the extract treated groups C-E with 800, 1000, 1200 mg/kg body weight/day of the *S. nitida* root bark extract and reference control, RC (group F) with 25 mg/kg body weight/day of silymarin respectively show significant decreases ($p < 0.05$) in MDA compared to those in IC (group B),and non-significant decrease ($p > 0.05$) in GST levels compared to those in IC (group B) rats (Figures 1 and 2). Also, treatment with alcohol significantly decreased ($p < 0.05$) the levels of GSH, CAT, SOD, GP, and GR in the IC rats in group B compared to the normal control rats in group A (see Figures 2-6). Administration of different doses of the extract and reference drug to experimental rats non-significantly increased ($p > 0.05$) GSH level in the extract treated rats in group C (ET1), and significantly increased ($p < 0.05$) GSH levels in the extract treated rats in groups D (ET2), E (ET 3), and in the RC rats in group F compared to the IC rats in group B (Figure 3). A significant increase ($p < 0.05$) in levels of CAT in the ET1-3 rats in groups C, D, E, and RC rats in F compared to rats in IC (group B), were also observed (Figure 4). It is seen from figure 5 that treatments of alcohol-induced liver injury in the experimental rats with graded doses of the extract and the reference drug significantly increased ($p < 0.05$) the levels of SOD and non-significantly increased ($p > 0.05$) the level of SOD in group C rats. Also, figure 6 revealed that the levels of GP and GR in the extract treated (ET) rats in groups C-E, and in the RC rats in group F were significantly increased ($p < 0.05$) compared to those of their counterparts in the IC (group B).

It was observed that the effects of the extract of *S. nitida* root bark in the experimental rats with ethanol-induced hepatic injuries were dose-dependent, and that there were no significant differences ($p < 0.05$) between the extract treated rats in groups D and E, and those of the reference control (group F) rats. For GSH and CAT, results showed that the effects of the extract of *S. nitida* root bark in the group E rats were slightly higher than those of the reference control (group F) rats that were treated with the drug.

Too much intake of alcohol activates the formation of free radicals and alters the levels of endogenous antioxidant systems,[22] resulting in oxidative stress. These free radicals damage the plasma membrane of cells and other biomolecules.[23,24] The result for the oral toxicity test (2236.1 mg/kg body weight) recorded in this study show that *S. nitida* root bark is very safe and is non-toxic. This is because substances with high LD_{50} are slightly toxic or non-toxic.[25] The elevated level of MDA seen in the IC rats in group B in this work might be due to the production of excess free radicals or reduced level of antioxidants,[26,27] resulting in peroxidation of membrane lipids in hepatocytes. The free radical productions may arise from the metabolism of ethanol in the liver by alcohol dehydrogenase forming toxic acetaldehyde that interacts with proteins and lipids in the cell causing free radical generations and cell damages. The increase in GST level recorded in the IC (group B) rats in this work might be due to oxidative stress,[28] as a defensive response to detoxify the metabolic products of alcohol metabolism, or might indicate a reduction in the concentration of ROS, since GST catalyzes the conjugation of GSH to a variety of substrates resulting in detoxification. The decrease in the levels of GSH, CAT, SOD, GP, and GR recorded in the extract treated groups (ET1-3) rats in groups C - E in this study might be consequences of oxidative stress and metabolism of ethanol by CYP2E1.[7] The observed reduction in the concentration of GSH in the IC (group B) rats in this study might be due to the rise in ROS generation or fall in the level of other endogenous antioxidants or both.[26,27] A decrease in CAT activity observed in this work in the IC (group B) rats might be due to exhaustion of the antioxidant enzymes induced by ethanol or caused by cross-linking and/or inactivation of the enzyme concern with lipid peroxidations,[28] while the decrease in SOD activity also seen in the IC rats in group B might not be unconnected with the excessive hydrogen peroxides produced as a result of CAT inactivation,[29] or due to liver injury. Decreases in both SOD and CAT activities in rats with ethanol-induced oxidative stress in group B (IC) may cause the accumulation of free reactive radicals which will result in loss of cell integrity and function.[30] The significant decrease in activities of GP and GR caused by ethanol in the IC rats in group B might be due to the formation of lipid peroxides or ROS that inactivate antioxidant enzymes, and impairment in the process of glutathione reduction.[26,31]

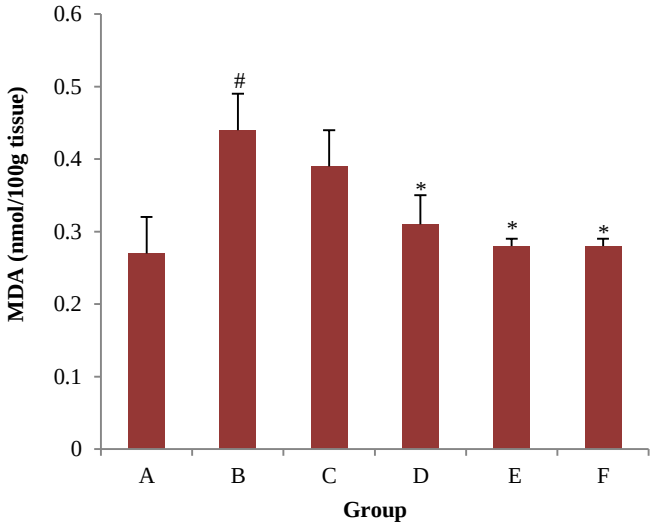

Figure 1: Effect of extract of root barks of *S. nitida* on MDA (nmol/100g tissue) in rats with ethanol-induced hepatic stress. Results = mean values ± standard error; # indicate significance ($p < 0.05$) compared to the normal and * indicate significance ($p < 0.05$) compared to the induction controls; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

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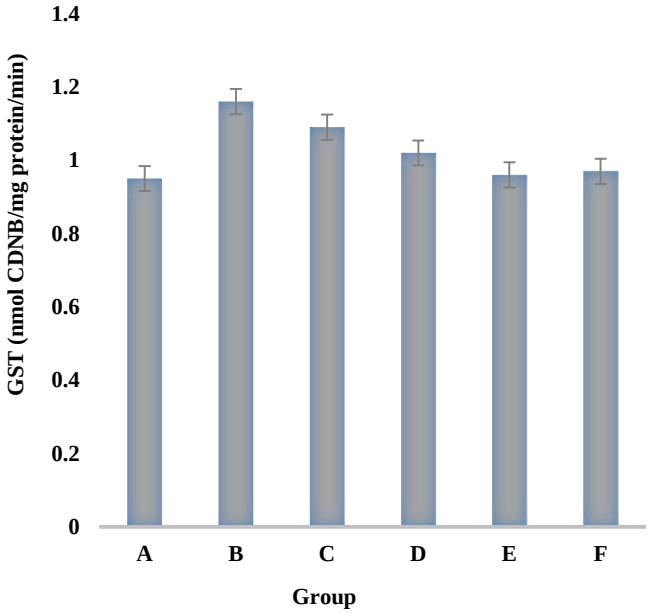

Figure 2: Effect of extract of root barks of *S. nitida* on GST (nmol CDNB/mg protein/min) in rats with ethanol-induced hepatic stress.
Results = mean values ± standard error; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

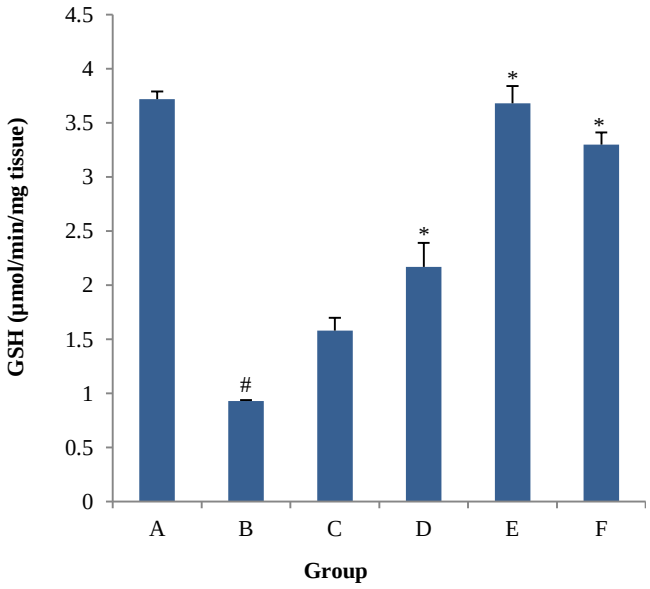

Figure 3: Effect of extract of root barks of *S. nitida* on GSH (µmol/min/mg tissue) in rats with ethanol-induced hepatic stress.
Results = mean values ± standard error; # indicate significance ($p < 0.05$) compared to the normal and * indicate significance ($p < 0.05$) compared to the induction controls; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

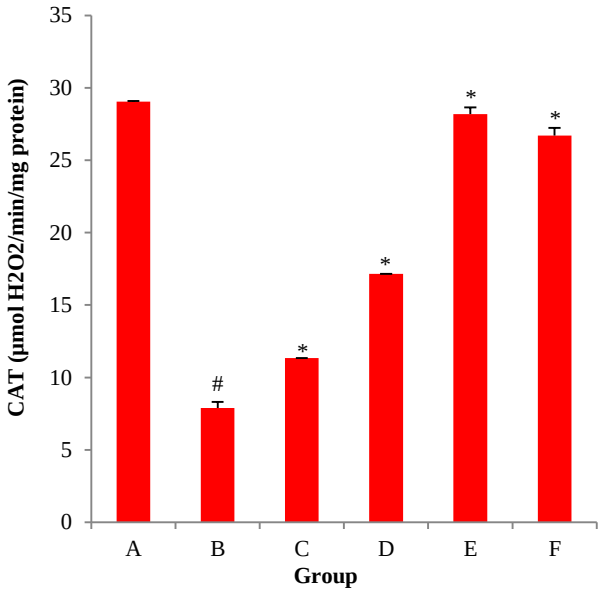

Figure 4: Effect of extract of root barks of *S. nitida* on CAT (µmol H_2O_2/min/mg protein) in rats with ethanol-induced hepatic stress.
Results = mean values ± standard error; # indicate significance ($p < 0.05$) compared to the normal and * indicate significance ($p < 0.05$) compared to the induction controls; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

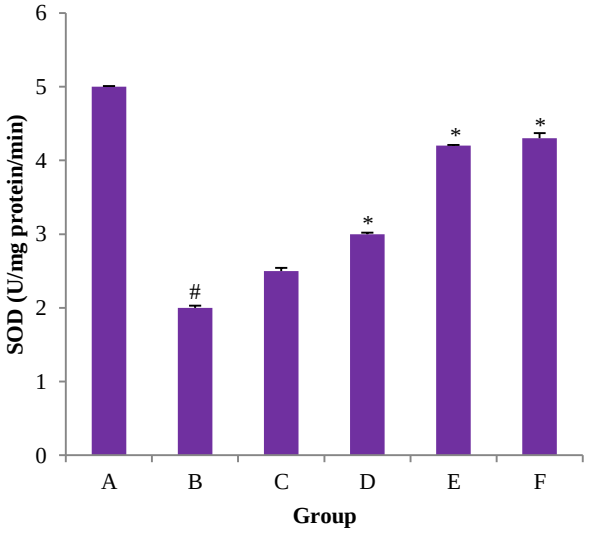

Figure 5: Effect of extract of root barks of *S. nitida* on SOD (U/mg protein/min) in rats with ethanol-induced hepatic stress.
Results = mean values ± standard error; # indicate significance ($p < 0.05$) compared to the normal and * indicate significance ($p < 0.05$) compared to the induction controls; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

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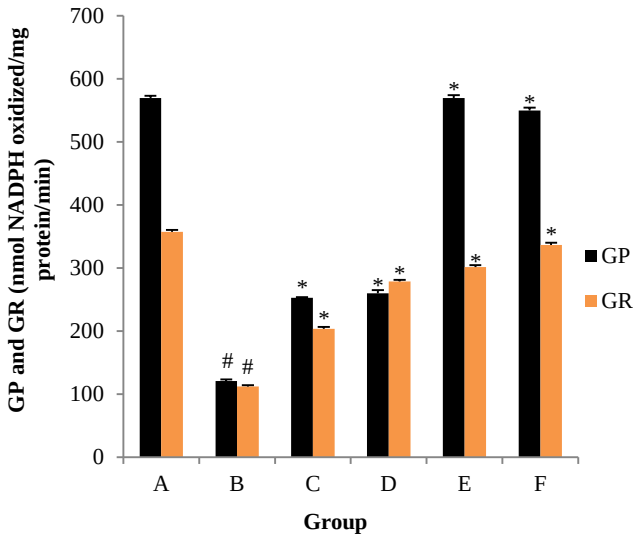

Figure 6: Effect of extract of root barks of *S. nitida* on GP and GR (nmol NADPH oxidized/mg protein/min) in rats with ethanol-induced hepatic stress.
Results = mean values ± standard error; # indicate significance ($p < 0.05$) compared to the normal and * indicate significance ($p < 0.05$) compared to the induction controls; n = 6. A = normal control rats (group A), B = ethanol treated (induction control, IC) rats, C-E = ethanol challenged rats treated with 800, 1000, and 1200 mg/kg body weight/day of *S. nitida* root bark, F = ethanol challenged rats treated with 25 mg/kg body weight/day of silymarin.

Our results showed that ethanol-treated rats in the IC group (group B) were in tandem with reports of Antonenkov and Panchenko,[29] who revealed that SOD and CAT activities increase after administration of excess ethanol in albino rats; Das and Vasudevan,[32] who reported that consumption of ethanol disrupts the antioxidant system and are dose- and time-dependent; and that of MacDonald *et al*.,[33] who also reported that chronic intakes of alcohol induces oxidative stress in rats. The results obtained revealed that administration of graded doses of aqueous extract of root back of *S. nitida* to ethanol-exposed rats significantly decreased ($p < 0.05$) the level of MDA and activity of GST, and increased the level of GSH and activities of CAT, SOD, GP, and GR compared to those of the induction control rats in group B. Though, it was observed that the effects of treatment with the aqueous extract of *S. nitida* root bark was dose-dependent, results revealed that the efficiency of the aqueous extract of *S. nitida* root bark was better than that of the experimental drug used for treatments of ethanol-challenged rats in the reference control group (group F).The ability of the extract of *S. nitida* root bark to restore the antioxidant concentrations in the experimental rats in extract treated groups (ET1-3) might be due to the presence in it, of phytochemicals with antioxidant properties like the quinolone alkaloids, flavonoids, phenolics, tannins, anthocyannins, and phytates.[34-38] Lunamarine and ribalinidine alkaloids have shown radical scavenging ability.[39] Flavonoids were reported to ameliorate LPO and liver damage by activating antioxidant enzymes, improved GSH levels, and reduced the activity of CYP2EI in human hepatocytes.[40,41] The therapeutic effects of most medicinal plants and their isolated compounds in the treatments of some diseases including cancer and malaria might be attributed to their antioxidant properties.[42-44] *Stachytarphyta jamaicensis* has been reported to exhibit good antioxidant activities due to the present of flavonoids and phenolics.[45] Pigments from grapes, coloured potatoes, and seed coats of black soybean containing anthocyanin have been reported to activate the production of antioxidant enzymes in cells.[46,47] So, the above mentioned phytochemicals with antioxidant potentials present in the extract of *S. nitida* root bark might be responsible for the radical scavenging effect exhibited by the plant part in this study.

Conclusion

In conclusion, results from the present study suggest that the aqueous extract of *S. nitida* root bark ameliorated oxidative stress in hepatic tissues of the experimental rats ingested with 40% ethanol with the implication of protecting the liver of humans if consumed. Therefore, this plant part might be useful in the manufacture of novel antioxidants that could be used to manage liver problems.

Conflict of interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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