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Antioxidant Effects of Ethanol Extract of Acrostichum Aureum (Golden Leather Fern) Against Lead Acetate Induced Oxidative Damage in Albino Rats

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ABSTRACT

This study evaluated the antioxidant effects of ethanol extract of Acrostichum aureum (golden leather fern) in lead acetate induced oxidative damage. Thirty male albino rats of mean weight 120 g were divided into 5 groups of six rats each. Groups 1 - 3 served as normal control, lead acetate control (100 mg/kg body weight), extract group only (400 mg/kg body weight), and while groups 4 and 5 were lead acetate induced groups treated with 200 and 400 mg/kg body weight of the extract respectively. Treatment lasted for 28 days, after which the animals were sacrificed under mild ether anaesthesia. Blood samples were collected for biochemical analysis. The results of the in vivo antioxidant activity showed a significant ($P<0.05$) increase in catalase, glutathione peroxidase, reduced glutathione, superoxide dismutase in the treatment groups when compared with the lead acetate control. There was a significant ($P<0.05$) decrease in malondialdehyde (MDA) in the treatment groups when compared with lead acetate control. Also the results of the in vitro antioxidant activity showed a significant ($P<0.05$) increase in the 2,2-diphenyl-1-picryhydrazyl (DPPH), ferric reducing antioxidant power (FRAP) in the treatment groups when compared with the lead acetate control. These findings suggest that ethanol extract of Acrostichum aureum attenuated the effect of lead acetate induced oxidative stress. Therefore, the extract could be used in managing lead acetate induced oxidative damage.

Keywords: Antioxidants, Acrostichum aureum, Lead acetate, Oxidative damage, Catalase

1. Introduction

Lead (Pb) is a toxic metal that induces a wide range of behavioural, biochemical and physiological effects in humans. Even though blood lead levels continue to decline over the past two decades, specific populations like infants, young children and working class are still at a higher risk[1,2]. As lead exposure tends to be sub-acute, produces only subtle clinical symptoms. Chronic exposure cases are more common than acute toxicity. Lead via gastrointestinal absorption is first taken up by the red blood cells and is distributed to all vascular organs[3]. Pathogenesis of lead poisoning is mainly attributed to lead induced oxidative stress. Chronic lead exposure is known to disrupt the pro oxidant/ antioxidant balance existing within the mammalian cells[4,5]. Lead is reported to cause oxidative stress by generating the release of reactive oxygen species (ROS) such as superoxide radicals, hydrogen peroxide and hydroxyl radicals and lipid peroxides[6-8].

As oxidative stress has been mainly implicated in the lead toxicity, reducing the possibility of lead acetate interacting with cellular metabolism biomolecules and decreasing the reactive oxygen species generation by the use of antioxidant nutrients has received considerable attention in the recent past[4,9,10]. There has been increased interest among phytotherapy researchers to use medicinal plants with antioxidant activity for protection against heavy metal toxicity[6,8].

Acrostichum aureum Linn is a plant that is widely distributed in Mangrove forests around the world including Nigeria[11,12]. It belongs to the genius, Pteridaceae an d Acrostichum respectively. It is found in mangrove swamps, salt marshes, and canal margins[13]. It is commonly known as mangrove fern, golden leather fern, tiger fern, and swamp fern[14]. The plant is locally used in Nigeria for the treatment of stomachache, internal heat, skin diseases, and migraines[15]. It is also used in other countries for the treatment of fever, skin infections, boils, ulcers, wounds, diabetes, pharyngitis, hemorrhoids, dysentery, hernia, chest pain, constipation, and snake bites[16-18]. Other documented traditional medicinal uses of the plants are in the treatment of asthma, sore throat, elephantiasis, asthma, worm infection, hypotension, and digestive problems[19]. Moreover, the young fronds of the plant are consumed as vegetables in some Asian countries[20]. The plant is rich in beneficial secondary metabolites and valuable phytochemicals including quercetin and its glycosides, isotachioside, kaempferol, lupeol, campesterol, stigmasterol, β and γ- sisosterol, pterosin P and C, tetracosane, patriscabratine and α-amyrin[21].

The plant has been reported to have antitumor, antiulcer, anthelmintic, antibacterial, antidiarrheal, analgesic, antiviral, and tyrosinase inhibitory properties[22]. The in vitro antioxidant activity and the reducing power of different extracts of the plant have also been demonstrated[23]. Recently Wu, et al.[24] showed that a polar extract of the plant protected against ethanol induced ulcers in rats by reducing oxidative stress and inflammation. However, there is a paucity of information on the effect of the plant on lead acetate induced oxidative damage. Consequently, the lead acetate rat model of oxidative toxicity was used to evaluate the antioxidant effect of ethanol extract of Acrostichum aureum.

2. Materials and Methods

2.1. Collection and identification of specimen

Fresh frond (leaves) and stalk of Acrostichum aureum were collected in the mangrove vicinity of University of Lagos and was identified and authenticated by a Taxonomist, Dr. Akinnibosun Henry Adewale in the department of Plant Biology and Biotechnology, University of Benin, and given a voucher number UBH-A650.

2.2. Preparation of sample and extraction

Fresh frond (leaves) of Acrostichum aureum were rinsed in running tap water to remove debris and then air dried under a shade for two weeks. The leaves were pulverized using a mechanical blender to coarse powder. Three hundred (300) g of pulverized sample was macerated in 2100 ml of ethanol and shaken severally. After 72 hrs, total extract obtained were filtered using a muslin cloth and subsequently with Whattman filter paper No. 1 (125 mm). Filtrate was there after concentrated using a rotary evaporator at 45°C to obtain the crude extract.

2.3. Experimental design

Thirty (30) wistar albino rats of mean weight 120 g were purchased from the animal facility Centre of the Faculty of Pharmaceutical Sciences, University of Nigeria Nsukka. The animals were allowed to acclimatize for two weeks prior to start of experiment, at the animal facility Centre of the Department of Biochemistry, Michael Okpara University of Agriculture, Umudike, with access to standard rodent feed and water ad libitum.

The animals were fasted overnight and randomly distributed into five groups of six rats each. Among the five (5) groups of animals, Group I received only food and water serving as normal control, while group 2 received lead acetate only (100 mg/kg body weight), group 3 received extract only (400 mg/kg body weight), Group 4 received lead acetate and extract (200 mg/kg), and Group 5 received lead acetate and extract (400 mg/kg body weight). Treatment lasted for 28 days after the animals were sacrificed under mild ether anaesthesia, blood samples were collected for biochemical analysis.

2.4. Evaluation of In vitro antioxidant activity

2,2-diphenyl-1-picrylhydrazine (DPPH) scavenging activity was carried out as described by[25]. Ferric reducing antioxidant power (FRAP) assay was carried out following the method described by Benzie and Strain[26].

2.5. Evaluation of in vivo antioxidant activity

The activity of catalase was assayed by the method of[27]. Superoxide dismutase activity was assayed by the method of[28] as contained in Randox kit. Estimation of reduced glutathione and glutathione peroxidase was done according to the method of[29]. Estimation of malondialdehyde (MDA) concentration was estimated by measuring spectrophotometrically the level of the lipid peroxidation product, malondialdehyde (MDA) as described by[30].

2.6. Statistical analysis

The results obtained was analysed statistically using one-way analysis of variance (ANOVA) to get the grouped mean which was used to determine the significant difference between the

group means. The Duncan Multiple Range test and post hoc test were used for comparison of the means of the various doses. A probability of 95 % level of confidence, using the statistical products and service solutions (IBM SPSS Statistics 22.0), and $P < 0.05$ was considered statistically significant between the test and control groups.

3. Results

3.1. Result of *in vivo* antioxidant assay

The effect of crude extract administration of Acrostichum aureum on MDA, GSH, GPx, SOD and CAT are shown in figure 1 to figure 5 below. For MDA, there is a significant ($P<0.05$) decrease in the level of malondialdehyde for all treated groups when compared to the lead acetate control **(Figure 1)**. However, there is a significant ($P<0.05$) increase in reduced glutathione and glutathione peroxidase activity in crude extract treated groups when compared to the lead acetate control **(Figures 2,3)**. Furthermore, there is a significant ($P<0.05$) increase in both catalase and superoxide dismutase activity in crude extract treated groups when compared to the lead acetate control **(Figures 4,5)**.

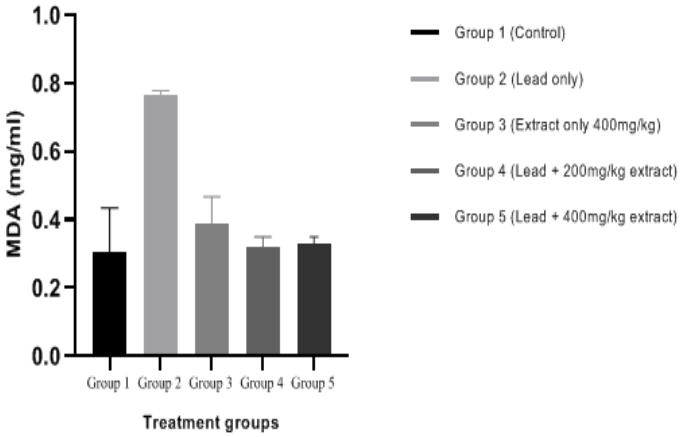

Figure 1: Effect of administration of crude extract of Acrostichum aureum on Malondialdehyde.

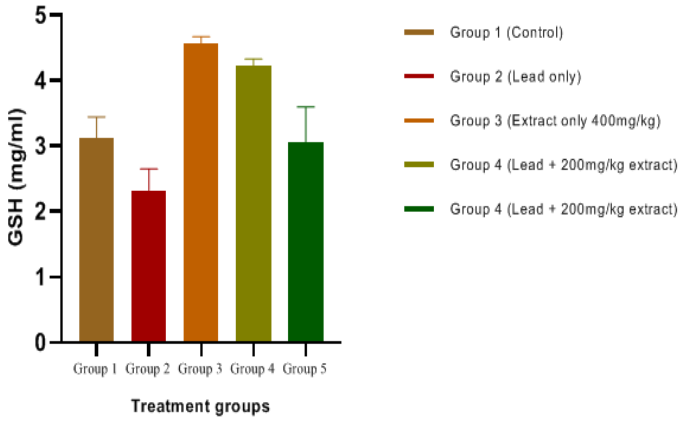

Figure 2: Effect of administration of crude extract of Acrostichum aureum on reduced glutathione.

3.2. Result of in vitro antioxidant assay

The in vitro antioxidant activity was evaluated and IC_{50} values were obtained. The result showed significant ($P<0.05$) increase in radical scavenging activity in both DPPH and FRAP models in the crude extract treated groups when compared with lead acetate control **(Tables 1 and 2)**.

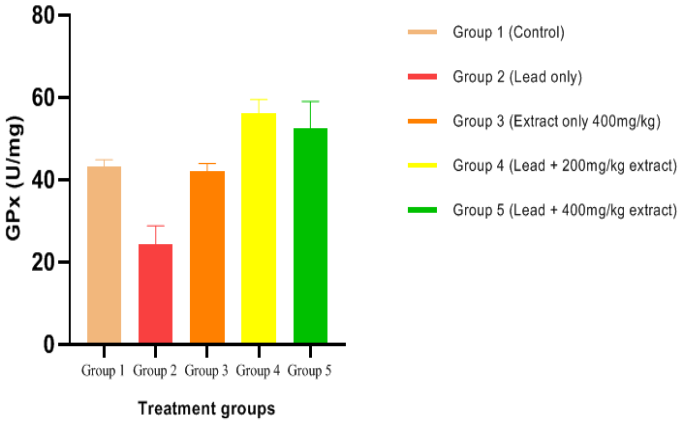

Figure 3: Effect of administration of crude extract of Acrostichum aureum on glutathione peroxidase (GPx).

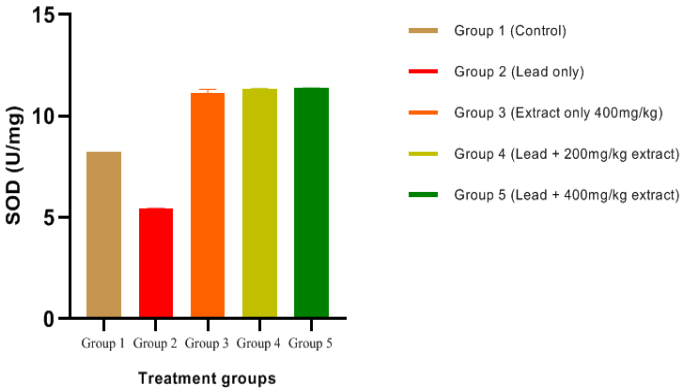

Figure 4: Effect of administration of crude extract of Acrostichum aureum on Superoxide dismutase (SOD).

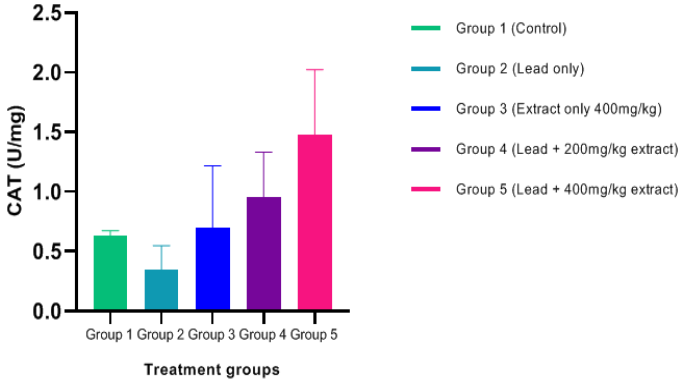

Figure 5: Effect of administration of crude extract of Acrostichum aureum on catalase (CAT).

Table 1: In Vitro antioxidant activity of crude extract on DPPH model.

/SAMPLE	IC_{50} VALUE (µg/ml)
Acrostichum aureum crude extract	420.0
Ascorbic acid (Standard)	96.35

Table 2: In Vitro antioxidant activity of crude extract in FRAP model.

SAMPLE	IC_{50} VALUE (µg/ml)
Acrostichum aureum crude extract	677.0
Ascorbic acid (Standard)	177.0

4. Discussion

Lead is known to cause oxidative damage in various tissues by bringing about imbalance in the generation and removal

of reactive oxygen species[31]. Although the exact mechanisms by which lead induces oxidative stress in various tissues are not completely understood, evidence indicates that multiple mechanisms may be involved. Numerous plant products have been shown to have high potent antioxidant activity. Recently, bioflavonoids and polyphenols of plant origin have been used extensively for free radical scavenging and to inhibit lipid peroxidation[32].

Lead is known to produce oxidative damage in various organs by increasing lipid peroxidation[33]. Lipid peroxidation will inactivate cell constituents by oxidation and ultimately lead to loss of membrane integrity[34].

Lead acetate is known to cause free radical damage in tissues by two mechanisms: Increased generation of ROS, including hydroperoxides, singlet oxygen and hydrogen peroxides, and by causing direct depletion of antioxidant reserves[35]. Superoxide dismutase, glutathione peroxidase and glutathione S-transferase enzymes take part in maintaining glutathione homeostasis in the tissues. These antioxidant enzymes are involved in the defence system against free radical mediated tissue or cellular damage after lead exposure[32]. The observed decrease in circulating antioxidants and decrease in serum total antioxidants confirm the lead acetate induced depletion of antioxidants depletion.

The present study showed that lead acetate administration to the animals resulted in severe oxidative stress. The selective dose of lead acetate used in the current study was based on previous literature[36]. Findings from this study showed that oral administration of ethanol extract of Acrostichum aureum attenuated the effect of lead acetate induced oxidative damage by increasing GSH and GPx levels, catalase and SOD activities and decreasing MDA levels. Our study revealed that lead acetate caused a significant increase in the MDA levels, which was decreased following administration with low and high doses of Acrostichum aureum extract. This implied that Acrostichum aureum extract attenuates oxidative damage induced by lead acetate by lowering MDA. One of the reasons for increasing levels of MDA is that high levels of ROS transverse the cell membrane and destroys neighbouring cells which result in an increase in the ROS that facilitates the cellular damage[37].

For the SOD antioxidant system assay, our findings revealed that oral administration of Acrostichum aureum extract at low and high doses enhanced the activities of SOD in experimental animals administered lead acetate and Acrostichum aureum extract. This observation indicated that the chemical constituents of Acrostichum aureum extract activated SOD isoenzyme activity, which ameliorated oxidative damage induced by lead acetate[38]. It was clearly shown by the dose significant response in the activity of SOD of animals in the treatment groups when compared to lead acetate control group.

Redox biomarkers such as GSH, GPx and CAT have been implicated in understanding the mechanisms related to the action of mixtures of xenobiotics on animal oxidative profile based on the current toxicological approach termed 'the real-life exposure scenario'[39]. However, the administration of ethanol extract of Acrostichum aureum to lead acetate induced animals led to a dose dependent significant increase in levels of GSH, GPx and CAT. This implied that exogenous administration of Acrostichum aureum extract increases GSH, GPx and CAT antioxidant levels, which was depleted by lead acetate. This is really a significant observation since the increase of these antioxidants in Acrostichum aureum treated groups attenuated lead acetate induced impairment in the intrinsic antioxidant defense mechanisms. Although, there is no report of the antioxidative activity of Acrostichum aureum in lead induced toxicity, however, the antioxidative properties observed, in which Acrostichum aureum increased CAT, GSH, and GPx levels are in line with the findings of Wu, et al.[24]. Also, Acrostichum aureum may have exerted similar antioxidant activity with flaxseed isolate incorporated with lemon juice which was reported to exert protective properties on lead induced kidney and liver toxicity[40].

The in vitro assay of this study further buttressed the antioxidant property of this plant. In comparison to standard ascorbic acid, Acrostichum aureum showed plausible antioxidant activities in DPPH model (IC_{50} value of 420 ug/ml) and FRAP model (IC_{50} value of 677 ug/ml). These results were significantly comparable to those obtained with ascorbic acid in both DPPH and FRAP models at IC_{50} value of 95.35 ug/ml and 660 ug/ml observed for ascorbic acid respectively. Antioxidants prevent lead acetate induced toxicity by inactivating the activities of generated ROS at the gene level, lead ion chelating, and prevention of ROS formation in its maintenance in a redox state thus contributing to its weakness in reducing molecular oxygen[41]. Also, assessing the hazard index (HI) and hazard quotient (HQ) of lead will shed more light in understanding the level of risk and toxicity of lead in the experimental animals so as to proffer an analysis for the mechanism of any outcomes with exposure to lead and Acrostichum aureum[42].

5. Conclusion

The findings from this study have demonstrated that oral administration of ethanol extract of Acrostichum aureum attenuated the effect of lead acetate induced oxidative stress by increasing GSH, GPx, catalase and SOD activity and decreasing MDA level. The results of this study also shows that the in vitro antioxidant activity (DPPH and FRAP) of the crude extract of Acrostichum Aureum has a potent antioxidative activity which may be due to the presence of the high phenols and flavonoid content which can act synergistically as free radical scavengers by donating an electron or hydrogen. Therefore, the extract could be useful in managing lead induced oxidative damage and these findings will contribute significantly to the search for locally available medicinal plant for managing and/or treatment of lead induced oxidative damage.

6. Ethical Approval

The authors hereby declare that "Principles of laboratory animal care" (NIH Publication No. 85- 23, revised 1985) were followed in this study, as well as specific national laws, where applicable. All experiments were examined and approved by the appropriate ethical committee.

7. Acknowledgement

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8. Conflicts of Interest

The authors declare that they have no conflict of interest.

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