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Mitigation of Lead Neurotoxicity by Aqueous Fruit Extract of *Adansonia digitata* in Adult Wistar Rats

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Aim: The current study seeks to explore the neuroprotective benefits of *Adansonia digitata* against lead induced memory impairment, neurotransmitter/AChE activity imbalance, oxidative stress as well as brain damage.
Methodology: Thirty male adult rats weighing 160g-200g were divided randomly into six groups (I-V1) consisting of five (5) rats in each group. Group I served as control and were administered with distilled water (1 ml/kg) only while groups II -VI were treatment groups. Group II were administered 250 mg/kg of *Adansonia digitata*; group III were administered 30 mg/kg of lead; Group IV were administered 250 mg/kg of *Adansonia digitata* plus 30 mg/kg of lead; Group V were administered 500 mg/kg of *Adansonia digitata* plus 30 mg/kg of lead; Group VI were administered 30 mg/kg of lead plus 10 mg/kg of succimer. All administrations were carried out through oral gavage for a period of 28 days.
Results: Lead administration caused memory impairment, decreased dopamine concentration and AChE activity in brain, induced oxidative stress resulting in brain damage. *Adansonia digitata* treatment significantly (P<.001) attenuated memory impairment, modulated dopamine

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concentration and AChE activity, prevented oxidative stress and ameliorated histopathological changes in the brain of Wistar rats.
Conclusion: The result showed that *Adansonia digitata* ameliorates lead-induced memory impairment in Wistar rats by improving memory index, controlling dopamine concentration and AChE activity, preventing oxidative stress and neuronal degeneration.

Keywords: Lead; Adansonia digitata; brain; memory index; dopamine; oxidative stress.

1. INTRODUCTION

Lead (Pb) is a naturally occurring toxic environmental agent with debilitating effects which is toxic to many organisms even in small quantity [1]. Lead toxicity is one of the major occupational hazards such as in automotive, paint industries and mining [2]. Consequently, substantial efforts have been made to eliminate lead from the environment, but despite some of the strict procedures being taken, there is persisting environmental contamination mainly in air, water, food, soil, paint and industrial wastes which still constitute significant sources of poisoning [3]. Studies have shown that a greater percentage of lead absorption in humans is by inhalation compared to absorption by ingestion, but irrespective of the route of absorption, about 90% of lead intake is retained in the body [4].

Minimal exposure to lead is known to affect both central and peripheral nervous systems; particularly the brain owing to its low rate of mitosis, high lipid content, oxygen tension and low antioxidant content [5]. Brain becomes the major target organ where severe neurological complications may arise after exposure to this heavy metal including damage to the nervous system microvasculature [6]. Lead causes an inappropriate release of neurotransmitters by competing with Ca^{2+} thereby interfering with evoked neurotransmitter release, deregulation of cell signaling, and neurotransmission [7,8]. Lead can cause immediate effects by altering physiology and chemistry of the brain. These functional alterations may result in morphological alterations in the brain that can persist even after lead levels have fallen in the system and induce long-term biochemical, structural, neurological and behavioral effects [9].

Presently, natural antioxidants from plants have come in to limelight to develop novel drugs for the treatment of oxidative stress-related ailments [10]. *Adansonia digitata* commonly known as baobab, exhibits strong antioxidant activity. As such, it has the potential to be used as a neuroprotective agent; as it may minimize the deleterious effects of metal-induced oxidative stress. *Adansonia digitata* is native to the African continent with a lot of antioxidant capabilities due to the presence of several components like vitamin C, Beta carotene, flavonoids, phenols, minerals (calcium, sodium, potassium, iron, copper, magnesium and zinc) and vitamins along with protein, fat, fiber and carbohydrate etc. [11,12]. Moreover, it has also exhibited various pharmacological properties as several traditional uses have been documented collectively in Nigeria and other African countries [13]. *Adansonia digitata* has been used traditionally as an immunostimulant, anti-inflammatory as well as in the treatment of dysentery and diarrhea. Similarly, it is been compared and considered as a substitute for some western drugs [14,15]. A previous study reported that baobab fruit pulp has a strong prebiotic effect on living organisms [15]. It is a known fact that antioxidant helps to prevent diseases caused by oxidative stress such as inflammation, cardiovascular disease, cancer, aging and neurodegenerative diseases [16]. The purpose of the present study was to evaluate the effect of aqueous fruit extract of *Adansonia digitata* on cytoarchitectural damage, brain lead level, MDA and dopamine concentration as well as acetylcholinesterase activity in the brain of rats exposed to lead.

2. MATERIALS AND METHODS

2.1 Plant Extraction

Adansonia digitata fruit pulp was grinded, 500 g of the fruit powder was soaked in distilled water for 24 hours. The mixture was filtered and the filtrate was evaporated in an oven at 40^{o}C. The extract was weighed and a percentage yield of 5.5 was obtained which was calculated using the formula: % yield = weight of AD/weight of extract x 100%.

2.2 Experimental Animals

A total of thirty (30) male rats of Wistar strain (120g-160g) were obtained from the Faculty of Pharmacy, Ahmadu Bello University (ABU) Zaria, Nigeria. The rats were kept in the Department of

Human Anatomy animal house for two weeks prior to the commencement of the experiments for acclimatization. They had free access to feed (grower mash, Grand Cereal, Nigeria) and water. The research was approved by ABU Zaria Research and Ethical Committee (ABUCAUC/2018/064).

2.3 Study Design

The rats were divided into six groups (n=5). Group I (control rats), received distilled water at 1 ml/kg. Group II rats received AD extract only at 250 mg/kg. Group III rats received Lead acetate only at 30mg/kg. Group IV rats received AD extract at 250 mg/kg) + lead acetate (30 mg/kg). Group V rats received AD extract at 500 mg/kg + lead acetate (30 mg/kg). Group VI rats received Succimer at 10 mg/kg + lead acetate (30 mg/kg). All administrations were done through oral gavage daily for 28 days.

2.4 Behavioural Test

The Morris water maze for special memory and learning used in our study was a black circular pool (140 cm diameter, 60 cm high) filled with water to the depth of 40 cm at 27 ± 1 ^{o}C. The pool was divided into 4 quadrants of equal size by the use of a visible tread. Each quadrant was designated as N (North), S (South), E (East) and W (West). An escape platform (10 cm diameter) was submerged in the middle of one of the quadrants (1 inch below the water surface). The training session consisted of three trials per day for 6 consecutive days which were started from one of the four start positions, used in a random sequence equal for every rat. During training, rats were made to stay on the platform for twenty seconds. Then lowered into the water facing the wall of the pool at one of the starting points. If a rat failed to escape within 60 seconds, it was guided to the platform by the experimenter and allowed to remain on the platform for 30sec and 60sec was allocated to the rat. Once the rat reached the platform, it was allowed to remain for 30 seconds and then placed in a holding cage for an inter-trial interval of 30 seconds. After the last trial, each animal was towel-dried and returned to its home cage. Retention of the spatial memory was assessed every one week from the start of the treatment. Escape latency on the last day of training (day 6) before treatment was considered as an index of learning. Twenty-four hours after the test for latency, probe test was carried out. Here, the platform was removed, and the time spent by rat in the quadrant where the platform was previously placed, was measured within 60 sec. The average time spent by the rats in the target quadrant searching for the hidden platform was considered as an index of memory. During the procedure, the experimenter stood at the same position for the entire experiment during each trial

2.5 Lead Concentration

To measure Lead level in the brain tissues, dried tissues were crushed to powder. 4 ml of perchloric acid, 24 ml Conc HNO_3 and 2ml Conc H_2SO_4 were added to 0.2g of grinded brain tissue under fume hood. The content was heated gently on a hot plate under perchloric acid fume hood showing dense white fume. The content was transferred into pyrex volumetric flask and top up with distilled water to 50 ml. The content was filtered using Whatman filter paper and the filtrate was transferred into lead aspirator and fixed into the Atomic Absorption Spectrophotometer (AAS-AA500G, PG instrument, England) for reading. The actual reading was taken by multiplying the value from AAS by volume of the digest divided by weight of sample (value from AAS ×50÷0.2) and was expressed in µg/g.

2.6 Preparation of Homogenate

Immediately after the sacrifice with the aid of ketamine with the dose of 75mg/kg intraperitoneally, the brain was rapidly excised, dissected on ice pack. The brain was rapidly divided into two halves by the use of razor blade. One half of each brain was weighed and homogenized immediately in 0.1 M phosphate buffer (Ph 7.4) at the ratio of 1 g tissue/ 5 ml of 0.1 M phosphate buffer [17], stored in plain sample bottles. The homogenate was centrifuged for 10 min at 3000 rpm the supernatant was stored in a refrigerator and used for MDA level, dopamine assay as well as acetylcholinesterase activity.

2.6.1 Malondialdehyde (MDA) level

From the supernatant, analysis for malondialdehyde (MDA) level in the samples were carried out using Rat malondialdehyde ELISA Kits from WKEA Med Supplies corporation, Changchun 130012 China. The procedure for ELISA technique guided by the manufacturer's manual were strictly followed.

2.6.2 Dopamine assay

The resultant supernatant was used for glutamate analysis. Glutamate concentration was

measured using Rat Dopamine Elisa Kit: Finetest-EU0392 (Wuham Fine Biotech Company Limited, Wuhan,), using standard glutamate stock solution to produce standard curve, dopamine levels in the samples were detected by microplate reader (RT-6000) which measured the absorbance at 450nm and was expressed as ng/ml.

2.6.3 Acetylcholinesterase analysis

From the supernatant, analysis for acetylcholinesterase (AChE) activity in the samples was carried out in accordance with the method of Ellman et al. [18]. In this method, the esterase activity was measured by providing an artificial substrate, acetylthiocholine (ATC). Thiocholine released because of the cleavage of ATC by AChE reacted with 2-nitrobenzoic acid (DTNB), which was reduced to thionitrobenzoic acid, a yellow-coloured anion with an absorption maximum at 412nm was obtained. The concentration of thionitrobenzoic acid was detected using a spectrophotometer and was taken as a direct estimate of the AChE activity expressed as nmol/min/ml.

2.7 Statistical Analysis

The Data were presented as Mean ± standard error of mean (SEM) and analysed using statistical package for social sciences (SPSS) version 23.0. One-way analysis of variance (ANOVA) was used to compare the mean difference between and within the groups and P-value (P<.05) was considered statistically significant.

3. RESULTS

3.1 Cognitive Ability

Lead treated group showed a significant increase in latency, while AD treated group showed significant decrease in latency when compared to the control (p<.05) with respect to the pre-treatment period. Groups treated with lead plus extract and lead plus succimer also showed significant (p<.05) reduction in latency when compared to lead treated group (Table 1).

3.2 Probe Test

Lead treated group showed a significant decrease in index of memory when compared to the control (P<0.01). Groups treated with AD extract only, AD extract plus lead and succimer plus lead showed significant increase in index of memory when compared to lead treated group at P<0.01 (Fig. 2).

3.3 Lead Concentration

Lead concentration increased significantly in lead treated group when compared with the control group (P<.05). There was a significant decrease in the lead concentration of rats treated with AD250, AD250 + Pb, AD500 + Pb and DMSA10 + Pb treated groups when compared with Pb treated rats atP<.05 (Table 1).

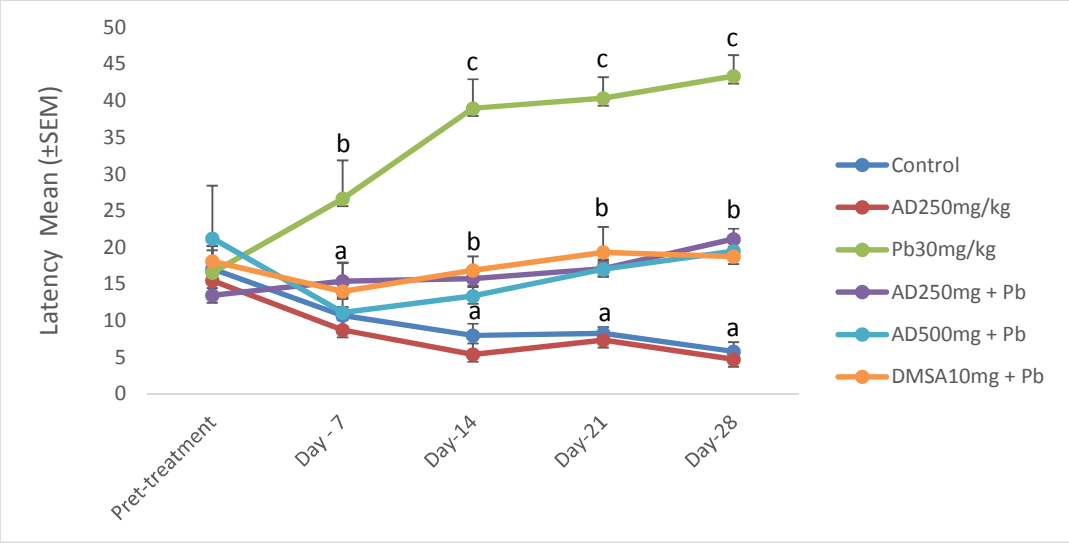

Fig. 1. Latency on Morris water maze of Wistar rats treated with Pb, AD, AD + Pb and DMSA + Pb. Values with different letters (a, b, & c) are significantly different at p<.05 (n = 4)

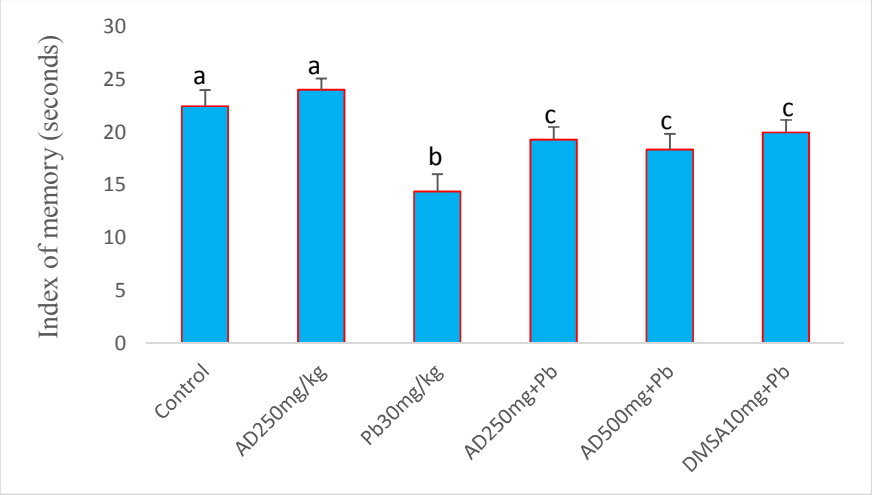

Fig. 2. Probe test on Morris water maze of Wistar rats treated with Pb, AD only, AD + Pb and DMSA + Pb. Values with different letters (a, b, & c) are significantly different at P<0.01 (n = 4).

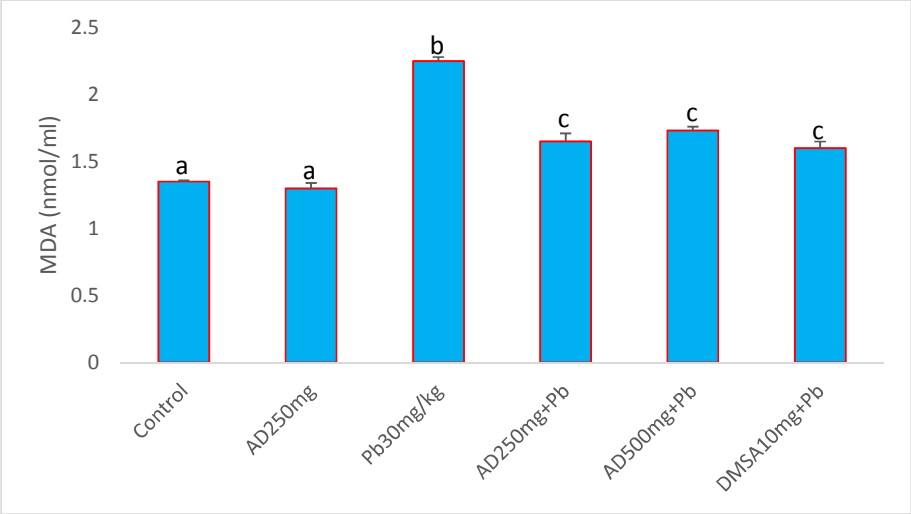

Fig. 3. MDA level in the brain tissue of rats treated with AD, Pb, AD + Pb and DMSA + Pb. Values with different letters (a, b, & c) are significantly different at P<0.01 (n = 4)

Table 1. Lead concentration (µg/g) in the brain tissue of rats treated with AD, Pb, AD + Pb and DMSA + Pb. (n = 4); Means ± SEM with a different letter ((a, b, c & d)) are significantly different (P<.05) from each other (One-way ANOVA, post hoc test)

Control	0.023 ± 0.015^{a}
AD250mg/kg	0.015 ± 0.010^{a}
Pb30mg/kg	0.377 ± 0.016^{d}
AD250mg + Pb	0.162 ± 0.012^{c}
AD500mg + Pb	0.121 ± 0.015^{b}
DMSA10mg + Pb	0.097 ± 0.011^{b}
F	2.676
p-Value	0.001

3.4 Malondialdehyde (MDA) Level

Malondialdehyde level increased significantly in lead treated group when compared with the control group (P<0.01). There was a significant decrease in the MDA level of rats treated with AD250, AD250 + Pb, AD500 + Pb and DMSA10 + Pb treated groups when compared with Pb treated rats at P<0.01 (Fig. 3).

3.5 Dopamine Concentration

Dopamine concentration decreased significantly in lead treated group when compared with the control group (P<0.01). There was a significant

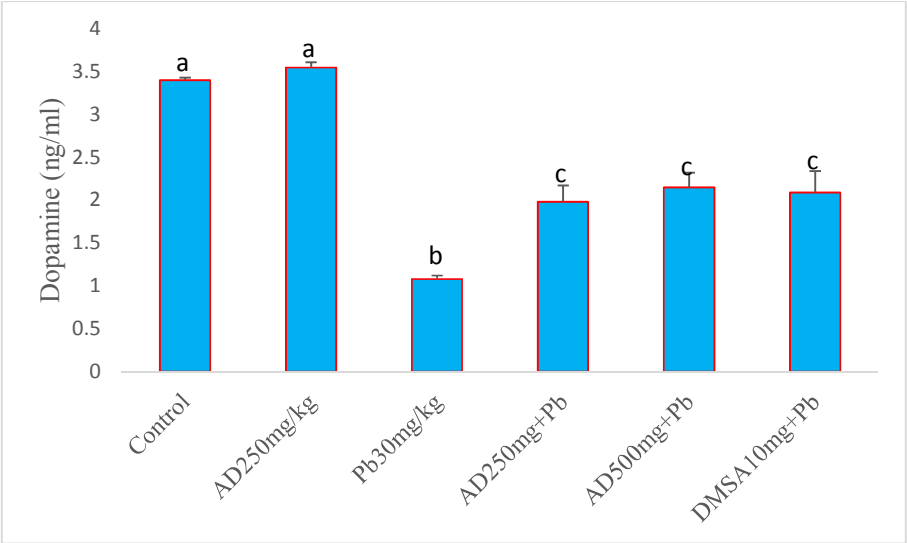

Fig. 4. Dopamine concentration in the brain tissue of rats treated with AD, Pb, AD + Pb and DMSA + Pb. Values with different letters (a, b, & c) are significantly different at P<0.01 (n = 4)

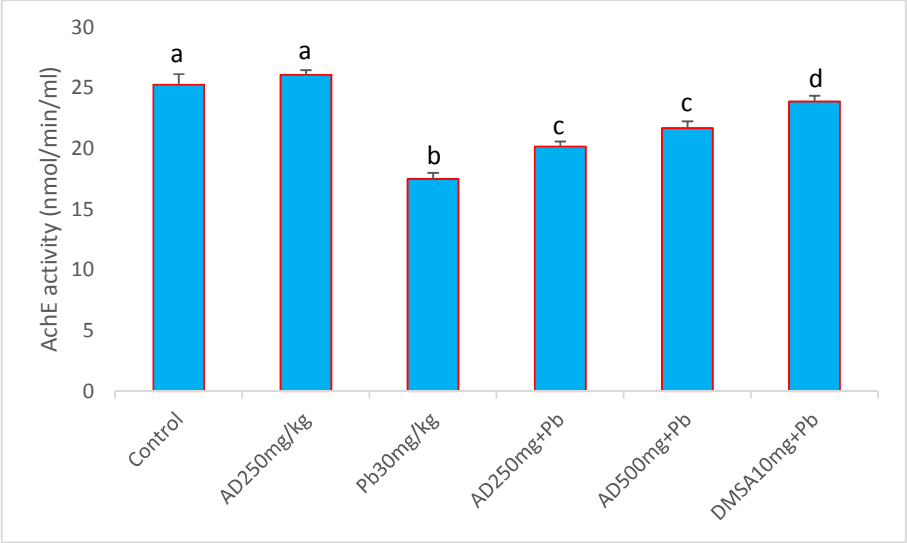

Fig. 5. AchE activity in the brain tissue of AD, Pb, AD +Pb and DMSA + Pb treated rat. Values with different letters (a, b, c & d) are significantly different at P<0.01 (n = 4)

increase in the dopamine concentration of rats treated with AD250, AD250 + Pb, AD500 + Pb and DMSA10 + Pb treated groups when compared with Pb treated rats at P<0.01 (Fig. 4).

3.6 Acetylcholinesterase Activity

Tissue acetylcholinesterase activity decreased significantly in all the treated groups when compared to the control (P<0.01). A significant increase in tissue acetylcholinesterase activity was observed in AD, AD + Pb and DMSA + Pb treated groups when compared with Pb only group at P<0.01 (Fig. 5).

3.7 Histopathological Studies

The microarchitecture of the hippocampal CA1 and CA3 areas of the Control and AD250 only showed Closely packed, linearly arranged pyramidal cells with well-defined shape (Fig. 5 A, B G & H). By contrast, architectural distortion in

the pyramidal layer, neuronal cell loss with cellular disarray were observed in Pb treated group; the group also showed some cytoplasmic features suggestive of cell death (Fig. 5 C & I). Distortions and neuronal degenerations were significantly reduced in the AD250mg + Pb, AD500mg + Pb as well as DMSA + Pb groups; when compared to the control. (Fig. 5 D, E, F, H, J, K & L).

Histological observations with cresyl fast violet stain showed normal neuronal organization and well stained nissl granules in the control and AD250mg groups (Fig. 6 A, B, G & H). By contrast, architectural distortion, neuronal degeneration and loss of nissl granules (chromatolysis) were observed in Pb treated group (Fig. 6 C & I). Mild neuronal degeneration and loss of the nissl substances were noticed in CA1 and CA3 regions of AD250mg + Pb, AD500mg + Pb as well as DMSA + Pb treated rats when compared with the control (Fig.5 D, E, F, H, J, K & L).

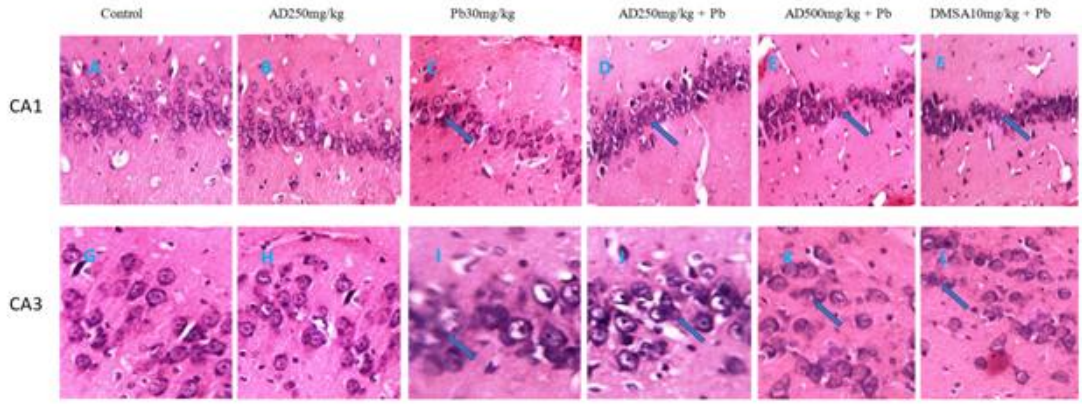

Fig. 6. Composite photomicrograph of CA1 and CA3 hippocampal subfields of the brain of rats treated with AD, Pb, AD + Pb and DSMA + Pb showing normal pyramidal cells in A, B, G & H; neuronal degeneration (arrow) in C, D, E, F, I, J, K & L. (H & E × 250)

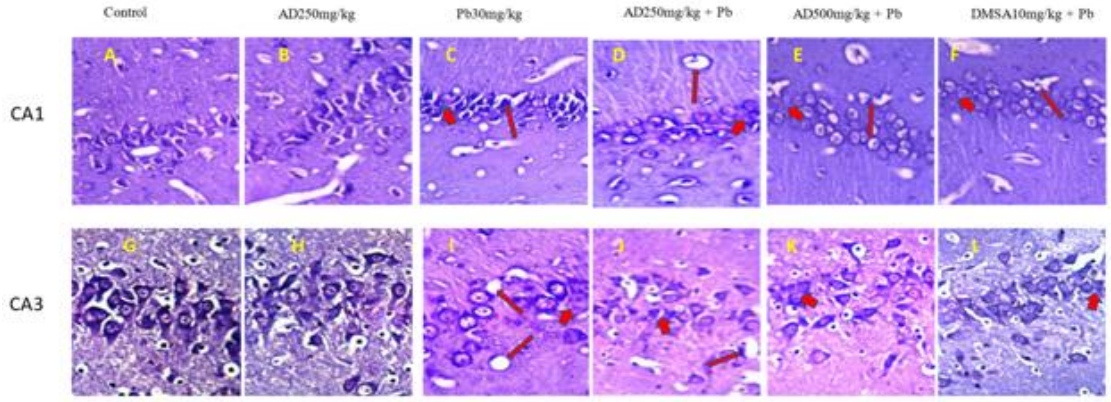

Fig. 7. Composite photomicrograph of CA1 and CA3 hippocampal subfields of the brain of rats treated with AD, Pb, AD + Pb and DSMA + Pb showing normal pyramidal cells with nissl granules in A, B, G & H; neurodegeneration (arrow head) and loss of nissl substances (arrow) in C, D, E, F, I, J, K, L. (CFV x 250)

4. DISCUSSION

The current findings demonstrated the damaging effects of Pb and the neuroprotective role of *Adansonia digitata* on a Pb induced brain damage in rats. From the study, it was evident that administration of Pb in rats resulted in the accumulation of lead in the brain causing

elevated MDA level, reduction in the concentration of dopamine as well as decreased acetylcholinesterase activity in the brain leading to memory impairment. The results also showed that treatment by *Adansonia digitata* ameliorated the deleterious effects of lead which was comparable to the effect of succimer. Moreover, a probable mechanism involved in these procedures was hypothesized to be due to the present of antioxidant in the extract, hence protection against brain tissues oxidative damage.

Exposure to lead has been shown to cause damages to various systems in the body especially nervous system through various mechanisms. Lead is known to cross the blood-brain barrier and disrupt the biochemical as well as structural components of brain by causing injury to the neuronal cells including glial cells, primarily in the cerebellum, hippocampus and cerebral cortex [19]. Lead deposited in these brain regions selectively and causes behavioral abnormalities such as impaired cognitive functions, learning impairment, decreased hearing and neuromuscular weakness both in humans as well as experimental animals [20]. It can also result in many neurochemical alterations resulting in a variety of neurological disorders like behavioral problems, nerve damage, mental retardation, schizophrenia and Parkinson's as well as Alzheimer's disease [21,22].

In this study, we observed an increased level of lead in the brain tissue of exposed rats when compared to the control, which further provides experimental evidence that some heavy metals such as lead can cross blood-brain barrier and impart its toxic effects in the brain at cellular and subcellular level. Studies have shown that exposure to lead from different sources leading to considerable accumulation of this heavy metal as time passes will leads to a critical defect in the brain with the most disruptive effect on the hippocampus [23,24]. Interestingly, AD treated groups were seen to significantly reduced the concentration of the lead in the brain similar to succimer. The mechanism for the action of AD in reducing the concentration of lead in the brain is not well understood, but could be attributed to the general neuroprotective role some antioxidants have on the brain. Some plants are seen to exhibit varieties of biological activities including strong anti-oxidant, anti-apoptotic, anti-inflammatory and neuroprotective activity [25,26].

The results of this study also showed increased MDA concentrations in the brain tissues of the rats exposed to lead when compared with the control. Researches have shown that exposure of animals to lead causes an increase in lipid peroxidation and impaired antioxidant defense enzymes in brain following lead exposure, suggesting that lead enhances oxidative stress [27,28]. Lead toxicity has been linked to the generation of free radicals associated with the enhancement of oxidative stress. This is seen in the Fenton reaction where lead catalyzed the reaction between iron and hydrogen peroxide resulting in the formation of free radicals [29]. The generated free radical then act on the lipid membrane of the brain tissues resulting in the process called lipid peroxidation. MDA is a maker of lipid peroxidation which has been reported as an evidence of lead-induces brain tissue oxidative stress [30,31]. The groups that we gave AD showed a significant reduction in the MDA level indicating that AD due to constituent such flavonoids, phenols, ascorbic acids etc. with strong antioxidant capabilities has the ability to reduce MDA level in the brain thereby preventing oxidative stress caused by chemicals or heavy metal [12]. Several plants material with antioxidant potential have been seen to reduce MDA level in animals exposed to heavy metals such as lead [32].

We also investigated the effectiveness of *Adansonia digitata* in attenuating memory impairment caused by lead toxicity and its ability in modulating reactive oxygen species due to its antioxidant constituent. We have shown from our previous study, that Adansonia digitata possesses several bioactive chemicals with strong antioxidant capabilities and a great potential in deactivating reactive oxygen species [12]. In assessing for cognition, we used MWM in evaluating animals for escape latency as well as index of memory using probe test. From our finding, lead treated group showed a significant increase in latency time, with a significant reduction in the probe time when compared to the control. Change in oxidative stress marker such as increase in MDA level due to Pb toxicity observed in this study supports other finding that induction of memory impairment by Pb could be associated with oxidative stress [33]. For AD treated groups, there was a significant reduction in latency time as well as significant increase in the time spent on the quadrant which had the escape platform (probe time) when compared with the lead treated group. Interestingly, the result we obtained concerning cognition in AD

was comparable to the group treated with succimer. Phytochemical analysis of AD showed compounds such as ascorbic acids, flavonoids and other phenolic compounds which have been known to act as a potent antioxidant by directly scavenging free radicals thereby improving memory impairment and brain damage caused by oxidative stress [11]. Study has proven the used of antioxidant-rich plant such as arbutin in maintaining the antioxidant system in biological system in protecting the brain from chemical-induced brain damage and ultimately improved cognition in MWM activity [34].

Acetylcholinesterase (AChE) is an enzyme present basically on extracellular surface of neurons in the synaptic area bound with local collagen and glycosaminoglycans. It catalyzes the breakdown of acetylcholine and of some other choline esters that function as neurotransmitters thereby setting the basis for rapid, repetitive, responses and enabling reuptake and recycling [35]. The predominant localization of this enzyme at the presynaptic terminal makes it an important marker enzyme for studying the process of nerve conduction [36]. AChE estimation can be used as an indicator for neurotoxicity [37]. Our study showed that AChE activity decreased significantly in the lead treated group when compared to control. Studies have shown a similar decrease in the activity of AChE in animals exposed to lead which was attributed to the production of ROS caused by lead toxicity as increased lipid peroxide and decreased activity of antioxidant enzymes in the brain lead to decrease in the AChE activity [38,39]. Moreover, [35] observed that inhibition of energy production by lead exposure appears to limit the availability of acetylcoenzyme A, the enzyme essential for acetylcholine production which is believed to be the indirect effect of lead on AChE activity. Interestingly, AD extract significantly restored AchE activity of the brain in lead treated rats, suggesting that AD extract could preserve living organism against neurotoxicity by reversing the AchE imbalance caused by lead. The protective efficacy of this extract can be attributed to its antioxidant action as well as the presence of carbohydrate which is a good source of acetylcoenzyme A required for the production of acetylcholine. These findings are consistent with the fact that plants materials rich in natural antioxidant significantly improve AChE activity [40].

Dopamine is an organic chemical of the catecholamine and phenethylamine families. It functions both as a hormone and a neurotransmitter, and plays several important roles in the brain and body. In the brain, dopamine functions as a neurotransmitter; a chemical released by neurons (nerve cells) to send signals to other nerve cells. In this study, brain tissue dopamine level decreased significantly in lead group when compared to control group. This reduction in the concentration of dopamine in lead group is in line with the work of [41,42]. They attributed the decrease to the effect of lead on the activity of tyrosine hydroxylase, DOPA decarboxylase and dopamine β-hydroxylase enzymes which are involved in dopamine synthesis. Research has shown that Ca^{2+} is very important for dopamine regulation and release by stimulating catecholamine secretion, acting through the calcium calmodulin-dependent protein kinase II system. But when a system is exposed to Pb, it competes with Ca^{2+} for common binding sites and get itself incorporated into calcium transport systems in the nervous system thereby disrupting the normal dopamine synthesis process [43]. In this study, AD increases the brain tissue concentration of dopamine. This could be attributed to the ability of the extract to prevent the formation of ROS which causes the reduction in the activities of the enzymes that catalyzed the synthesis of dopamine. According to [44], the availability of glucose is also believed to increase the synthesis of dopamine due to the formation of coenzyme A during the metabolism of glucose. Coenzyme A is one of the precursors needed for the production of dopamine. Phytochemical analysis of AD shows a reasonable concentration of carbohydrates, as such it can be hypothesized that increase concentration of glucose from carbohydrate metabolism in the extract also plays a part in stimulating the synthesis of dopamine thereby increasing the concentration of dopamine in the animals treated with the extract.

Microscopically, brains (Hippocampus) of the control group rats revealed normal histological structure. Moreover, brains of AD group rats showed no histopathological changes. Meanwhile, brains of Pb group rats revealed necrosis of neurons as well as chromatolysis. However, the brains of Pb + AD group rats revealed an improved picture and the examined sections showed some level of neurodegeneration and chromatolysis similar to succimer group. Our results are in tandem with the work of [45] who observed that the toxicity of lead destroys the ordinary histological structure of the brain and influences the physiological

functions it performs. [46] observed that exposure to lead resulted in hippocampal damage with a microscopic study revealing the reduction in the overall thickness and neuronal loss of pyramidal cells particularly in the deeper regions showing empty spaces with vacuoles due to neuronal degeneration. Negative changes in neuronal cells that appeared in this examination may be attributed to the depletion of antioxidant reserves due to lead toxicity and the generation of ROS resulting in oxidative stress since oxidative stress plays a major role in the brain damage of living organisms. The administration of Ad extracts helps to reduce histo-architectural damage, relieve inflammation and oxidative stress similar to the succimer group. This may be due to the presence of constituents that have antioxidant potentials and thus prevent the formation of free radicals and reduce the damage caused by oxidative stress. There is evidence indicating that compounds rich in antioxidant has the ability to protect the histoarchitecture of the brain against metals induced neuronal damage [33].

5. CONCLUSION

The result showed that *Adansonia digitata* mitigates lead-induced memory impairment in Wistar rats by improving memory index, controlling dopamine concentration and AChE activity, preventing the formation of ROS hence lessened oxidative stress and neuronal degeneration.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by TETFund.

CONSENT

It is not applicable.

ETHICAL APPROVAL

The research was approved by ABU Zaria Research and Ethical Committee (ABUCAUC/ 2018/064).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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