Research Article

Neuroprotective Effect of *Adansonia digitata* against Aluminum Chloride-induced Memory Deficits and Hippocampal Damage in Wistar Rats

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Abstract

Aim: Several reports have shown environmental neuron toxins such as aluminium to accumulate in the brain, potentially triggering neurodegenerative disorders. Adansonia digitata (Baobab) has been reported to possess beneficial properties. This study, assessed the neuroprotective properties of aqueous fruit pulp extract of Adansonia digitata (AEAD) on aluminum chloride (AlCl3) – induced memory dysfunctions and hippocampal changes in Wistar rats. The neuroprotective effects of AEAD were assessed with the Morris water maze for learning and memory, oxidative stress biomarkers glutathione (GSH), superoxide dismutase (SOD), and Malondialdehyde (MDA), and histopathological changes on the hippocampal CA3 region using histological techniques. **Materials and Methods:** Thirty male Wistar rats (110 and 150 g) were divided into six groups at random (n=5). The Control group, the animals in Group 1 received 2 ml/kg distilled water. Group 2 received 100 mg/kg of AlCl3. Ascorbic acid at a dose of 595 mg/kg was given to Group 3. 100 mg/kg AlCl3 and different concentrations of the AEAD (500 mg/kg and 1500 mg/kg, respectively) were given to groups 4 and 5. Group 6 received 595 mg/kg of ascorbic acid and 100 mg/kg of AlCl3. **Results:** The latency time spent to locate the escape platform in the Morris water maze test was observed with remarkable ($P<0.05$) improvement in the AEAD treatments compared with the AlCl3-treated group. There was a notable increase in MDA levels and a reduction in SOD and GSH activities in the AlCl3-treated group in relation to the AEAD-treated groups. Histopathological examination of the CA3 hippocampal region treated with AlCl3 revealed neurodegenerative changes, whereas, administration of AEAD ameliorated AlCl3-induced neuronal damages in rats at doses 500mg/kg and 1500mg/kg when compared with the AlCl3-treated group. **Conclusion:** Aqueous fruit pulp extract of Adansonia digitata demonstrated a possible neuroprotection against aluminium chloride-induced memory deficit and CA3 hippocampal neurotoxicity.

Keywords: Oxidative stress, CA3, histology, neurobehavior, phytochemical, wistar rats.

Introduction

Environmental heavy metals are well-recognised exogenous elements that influences brain development and growth. There are reported connections according to the literature between heavy metals and neurodegenerative conditions like Parkinson's disease, Alzheimer's disease, Huntington's disease, frontotemporal dementia, amyotrophic lateral sclerosis, dialysis encephalopathy among others[1,2,3]. Aluminum (Al) is one of the heavy metals implicated in the development of neurodegenerative diseases, because it impacts many cellular metabolic pathways in the central nervous system[4,5,6].

The exposure of humans to Al commonly happens via water and food consumption, cosmetics and antiperspirants, vaccines, and industrial processes etc.,[7,8]. The ionic form of Al has the ability to create resolvable salts promoting ingestion by animals causing disruption of homeostasis initially within the tissue and then within the organ[9]. Al exposure enhances acetylcholinesterase activity, decreases cholinergic activity, and encourages lipid peroxidation[10]. Al can pass through the blood-brain barrier and build up in the brain, with the hippocampus having the greatest quantities[11]. Accumulation of Al causes oxidative stress in the brain and neuronal cellular degeneration, consequently provoking apoptosis and damage to cells of the brain.

Deep within the cerebral temporal lobe is a complex brain structure, the hippocampus, that plays a key role in spatial navigation, memory and learning, control of hypothalamic processes and emotional behavior[12]. A build-up of Al in the hippocampus leads to spatial memory deficits, emotional reactivity, and cognitive impairment primarily by inhibiting long-term potentiation via the glutamate nitric oxide-cyclic guanosine monophosphate pathway[10,13]. Concurrently, Al exposure potentiates mitochondrial dysfunction and NADPH oxidase activation, thereby amplifying

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reactive oxygen species generation and depleting endogenous antioxidants such as glutathione and superoxide dismutase, establishing oxidative stress[9].

Increased oxidative stress is a trigger for many neurodegenerative diseases and age-linked illnesses, and therefore, there is a great deal of interest in finding natural medicinal agents that improve cognitive function and neuroprotection via antioxidant activation[14]. *Adansonia digitata* is also known as *Igi-ose, Kuka*, and *Oshe* in the Yoruba, Hausa, and Igbo Nigerian languages, respectively. It belongs to the family Malvaceae and sub-family Bombacaceae, known as African Baobab[15,16]. In many African nations, different plant parts (such as leaves, bark, and fruit pulp) have long been used as immune-stimulants, anti-inflammatories, analgesics, insect repellents, and pesticides to treat diarrhoea and dysentery. These parts have also been tested as a potential replacement for imported Western medications[17]. Its numerous traditional uses, including medical, nutritional, and cosmetic ones, have drawn the attention of numerous pharmaceutical companies and researchers in the last ten years[18]. Reports of its phytochemicals' high nutritional content and other characteristics have recognized it as a possible nutraceutical agent[19]. Therefore, this study seeks to evaluate the neuroprotective potentials of aqueous fruit pulp extract of *A. digitata* on aluminium chloride-triggered hippocampal neurotoxic of adult male Wistar rats.

Materials and Methods

This study's ethical approval, with approval code ABUCAUC/2018/070 was granted by the Ahmadu Bello University Ethics Committee on Animal Use and Care.

Plant Material Collection, and Identification

Dried *Adansonia digitata* (Baobab) fruits were obtained from the locality (Samaru market, Kaduna state), and validated in the Department of Biological Sciences (Herbarium Unit), Ahmadu Bello University (ABU), Zaria Nigeria and a Voucher specimen Number 7170 was provided.

Extract Preparation and Phytochemical Screening

Adansonia digitata fruit pulp extraction and phytochemical screening were conducted. The method of maceration as described by Al-Qarawi *et al*. [21] for the extraction of aqueous fruit pulp extract of *Adansonia digitata* (AEAD) was adopted, and the technique of Trease and Evans [22] was adopted for phytochemical assessment.

Acute toxicity study

An acute toxicity study of AEAD aqueous extract was conducted according to the method reported by Lorkes [23].

Experimental Animals

Wistar rats (110 to 150 g) were obtained and kept in standard cages in the Animal Facility of the Department of Human Anatomy ABU, Zaria, Nigeria and were habituated for two weeks prior to the start of the study. The rats were provided with a typical rodent pelletised food and water *ad libitum*. The rats were categorized into control and treatment groups. Experimentation lasted for four weeks. The rats were weighed using digital weighing scale (Acculab Vicon VIC-511 Precision Balance/Scale, USA, 0.001 g) before and after experimentation, and observed for any physical changes.

Drugs

Aluminium chloride ($AlCl_3$), manufactured by Guangdong Limited, Shantou, China was obtained and used as a neurotoxicant for the experiment.

Vitamin C (Ascorbic acid), manufactured by Emzor Limited, Lagos, Nigeria was obtained and used as a standard antioxidant.

Experimental Procedure

Thirty apparently healthy male Wistar rats (110-150 g) were randomly assigned into six groups of five rats each. The groupings were H_2O (2 ml/kg) –treated group which was administered 2 ml/kg distilled water, 100 mg/kg $AlCl_3$-treated group which received 100 mg/kg aluminium chloride only, 595 mg/kg ascorbic acid-treated group received only 595 mg/kg Vitamin C, 100 mg/kg $AlCl_3$ + 500mg/kg AEAD-treated, and 100 mg/kg $AlCl_3$ + 1500mg/kg AEAD-treated groups were both administered 100 mg/kg aluminium chloride only, and different doses of AEAD at 500 and 1500 mg/kg respectively, and 100 mg/kg $AlCl_3$ +595 mg/kg ascorbic acid–treated group which received 100 mg/kg aluminium chloride and 595 mg/kg Vitamin C. All administration were through oral route.

Morris Water Maze (MWM)

The method described by Morris, [24] for spatial learning and memory was adopted for this study. Rats were tested in a water-filled water maze that measured 180 cm in diameter and 60 cm in height. Halfway between the wall and the center of the pool, in one of the four quadrants, an escape platform was fixedly positioned 2 cm below the water's surface.

Procedure

Before treatment, the rats were trained for six days. The training session consisted of three consecutive trials (with a 30-second gap between trials) during which each rat was randomly placed in one of four quadrants, of the pool and permitted to freely swim to the platform for escape or until 60 seconds had elapsed. Should a rat be unable to locate the platform within 60 seconds, it was taken and placed there to stay for 20 seconds. In between treatments, they were put back in their own cages. After swimming to the platform and remaining there for 10 seconds, each rat was taken out of the water for 30 seconds before being relocated to the next starting area. Using a stopwatch, the latency time to locate the platform was recorded for every session. For six days, this training process was repeated, but the beginning points—the axis of one hypothetical quadrant—were changed in a pseudo-randomized way. The test sessions were subsequently conducted once weekly for four consecutive weeks.

Euthanasia

Following completion of the experiment, the rats were euthanized using chlorate hydrate (350 mg/kg) intraperitoneally. The rats were decapitated and the brains were removed. The dissected whole brains were sectioned into halves at the sagittal plane, one half of the brain was removed and homogenized for biochemical assessment and the other half was fixed in Bouins fluid for histological studies.

Biochemical studies

Harvested brains were weighed and manually homogenized (1 g tissue/4 ml) in 0.1 M phosphate buffer (pH 7.4) in accordance to the technique described by Ige *et al*. [25]. Homogenate was

analysed for oxidative stress biomarkers such as reduced glutathione (GSH), superoxide dismutase, (SOD), and malondialdehyde (MDA). Enzymatic antioxidant activity was estimated by assaying GSH activity in accordance to the procedures described by Rukkumani *et al*. [26] and SOD activity according to the methods of Fridovich [27]. The Ohkawa *et al*. [28] method was used to evaluate the amounts of lipid peroxidation using the MDA (thiobarbituric acid reactive substance) assay.

Histological Studies

The fixed brain was processed using histological techniques which included fixation, dehydration, clearing, infiltration and embedding in paraffin wax. It was stained with Haematoxylin and Eosin stain to access the cytoarchitecture of the hippocampus (CA3 region). The stained sections were observed under a light microscope and photomicrographs were obtained at a magnification of x250.

Data Analysis

The Statistical Package for Social Scientist (SPSS version 20.0) was used to analyze the data obtained. The results were presented as mean ± SEM, and one-way analysis of variance (ANOVA) with Turkey's *post hoc test* for significance was used to determine whether there were significant differences between the group means. Significant values were defined as $p<0.05$.

Results

Extract Preparation and Phytochemical Analysis

It was observed that the aqueous extract had a yield of 20.13%. Phytochemical assessment of AEAD produced a positive reaction for the following metabolites: cardiac glycosides, saponins, flavonoids, tannins, glycosides, steroids and triterpenes. However, a negative reaction was gotten for anthraquinones, tannins and alkaloids.

Acute Toxicity

Signs of toxicity or mortality were not observed in the rats upon administration of AEAD up to the dose of 5000 mg/kg, which indicates that the LD_{50} of AEAD is safe.

Physical Observation

Throughout experimentation, the rats were observed for any changes in physical activities and behavioural patterns. The H_2O (2 ml), and 595 mg/kg ascorbic acid-treated groups were observed to be very active, reacting to stimuli, moving around the cage. The rats in 100 mg/kg $AlCl_3$ only group, 100 mg/kg $AlCl_3$ +500 mg/kg AEAD, 100 mg/kg $AlCl_3$ +1500 mg/kg AEAD and 100 mg/kg $AlCl_3$ +595 mg/kg ascorbic acid –treatment groups were observed to show signs of decreased physical activities, gnawing and restlessness, scratching of mouth and nostril, nose bleeding, frequent urination, and watery faeces during the first two weeks of administration. However, improved physical activities were observed in 100 mg/kg $AlCl_3$ +500 mg/kg AEAD, 100 mg/kg $AlCl_3$ +1500 mg/kg AEAD, and 100 mg/kg $AlCl_3$ +595 mg/kg ascorbic acid -treatment groups during the last two weeks of administration. The effect of A. digitata on changes in body weight in Wistar rats exposed to aluminium chloride is shown in Figure 1.

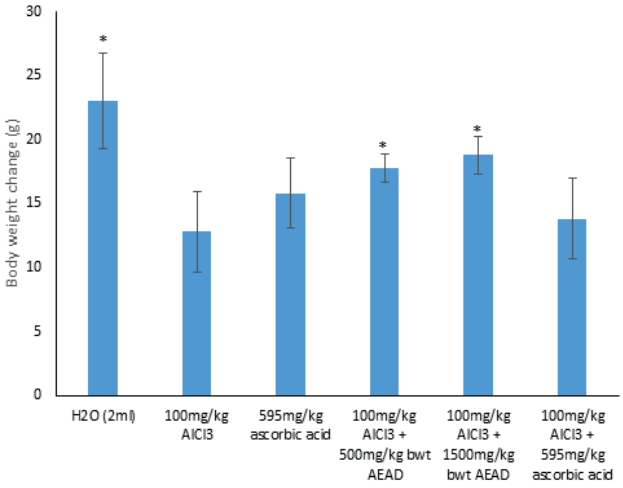

Figure 1: Effect of A. digitata on body weight changes of Wistar rats exposed to Aluminium chloride

n=5, One-way ANOVA followed by Tukey post hoc test, * significant ($p<0.05$) difference when compared with 100 mg/kg AlCl3 –treated group. AlCl3: Aluminum chloride; AEAD: Aqueous fruit pulp extract of *A. digitata*

The Wistar rats' weights were recorded both before and after the study and the weight differences were subjected to statistical analysis. There was a notable ($p<0.05$) increase in body weight of 100 mg/kg $AlCl_3$ +500 mg/kg AEAD, and 100 mg/kg AlCl3 +1500 mg/kg AEAD – treated groups in relation to the 100 mg/kg $AlCl_3$–treated group (Figure 1).

Morris Water Maze Studies

The effect of A. digitata on the latency time of Wistar rats exposed to aluminium chloride is shown in Figure 2. Rats were tested for spatial learning and memory using MWM. By the end of the training, all of the rats had figured out how to find the hidden platform, as seen by the mean training time, which showed that they could find the platform quickly. However, on days 14, 21, and 28, the 100 mg/kg $AlCl_3$-treated group, showed learning and memory impairment by increased ($p<0.05$) in the time (sec) to locate the escape platform when compared to the other groups (Figure 2).

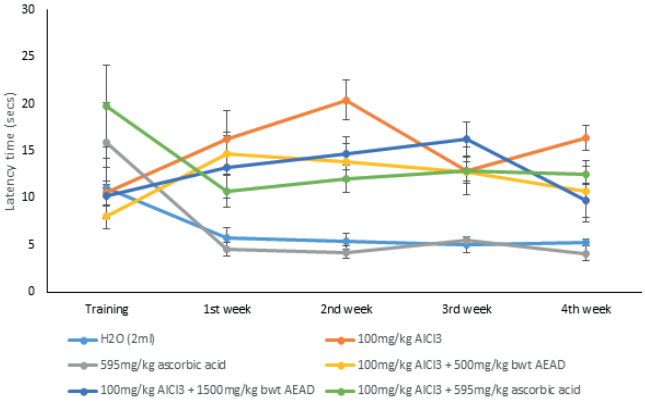

Figure 2: Effect of A. digitata on Latency time of Wistar rats exposed to Aluminium chloride

n=5, One-way ANOVA followed by Tukey post hoc test, * significant ($p<0.05$) difference when compared with 100 mg/kg AlCl3–treated group. AlCl3: Aluminum chloride; AEAD: Aqueous fruit pulp extract of *A. digitata*

Biochemical Analysis

The effect of A. digitata on reduced glutathione (GSH) activity in Wistar rats exposed to aluminium chloride is shown in Figure 3.1; the effect of A. digitata on superoxide dismutase (SOD) activity in Wistar rats exposed to aluminium chloride is shown in Figure 3.2; The effect of A. digitata on malondialdehyde levels in Wistar rats exposed to aluminium chloride is shown in Figure 3.3. The results of the biochemical analyses (Figure. 3) for oxidative stress markers: antioxidant enzyme activity GSH, SOD, and MDA were evaluated in brain tissue homogenate of the rats. There was a remarkable ($p<0.05$) decrease in GSH and SOD activities in the 100 mg/kg $AlCl_3$-treated group in relation to the other groups (Figures 3.1 and 3.2). The result obtained showed that there was notable ($p<0.05$) increased in MDA level in 100 mg/kg $AlCl_3$-treated group in relation to H_2O (2 ml) –treated group, 595 mg/kg ascorbic acid –treated group, 100 mg/kg $AlCl_3$ +500 mg/kg AEAD –treated group, 100 mg/kg $AlCl_3$ +1500 mg/kg AEAD –treated group, and 100 mg/kg $AlCl_3$ +595 mg/kg ascorbic acid –treated group (Figure. 3.3).

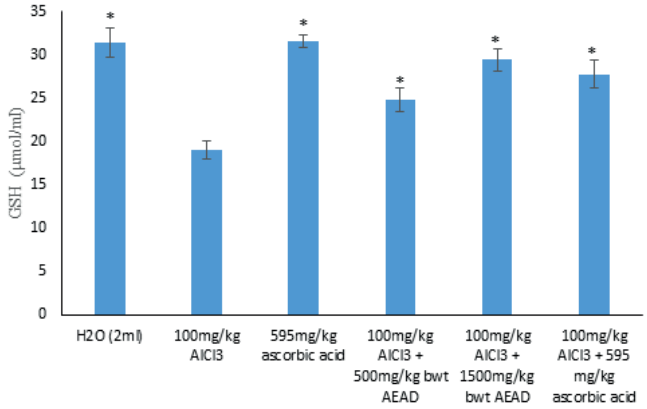

Figure 3.1: Effect of A. digitata on reduced glutathione (GSH) activity of Wistar rats exposed to Aluminium chloride

n=5, One-way ANOVA followed by Tukey post hoc test, * significantly (p<0.05) difference when compared with 100 mg/kg AlCl3 –treated group. AlCl3: Aluminum chloride; AEAD: Aqueous fruit pulp extract of A. digitata

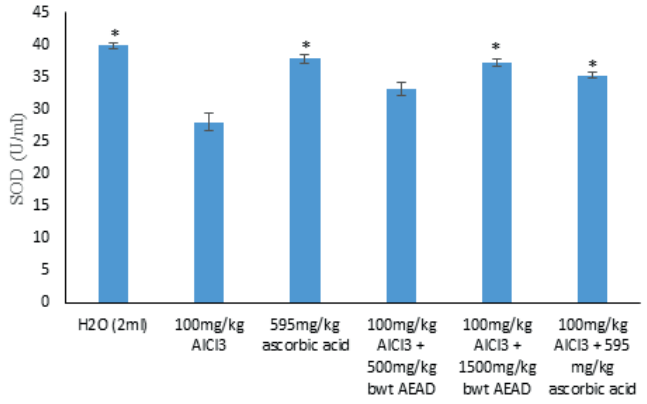

Figure. 3.2: Effect of A. digitata on Superoxide dismutase activity (SOD) of Wistar rats exposed to Aluminium chloride

n=5, One-way ANOVA followed by Tukey post hoc test, * significantly (p<0.05) difference when compared with 100 mg/kg AlCl3 –treated group. AlCl3: Aluminum chloride; AEAD: Aqueous fruit pulp extract of A. digitata

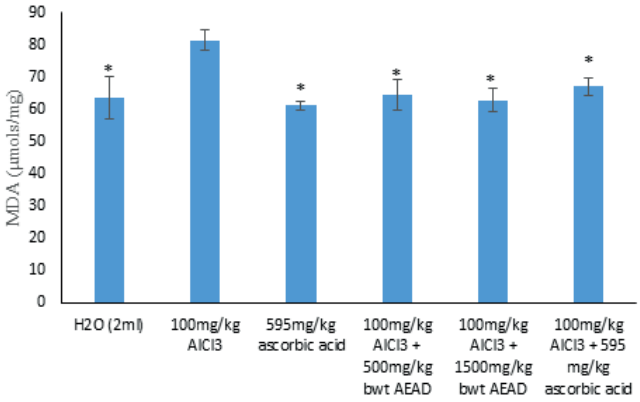

Figure. 3.3: Effect of A. digitata on Malondialdehyde levels of Wistar rats exposed to Aluminium chloride

n=5, One-way ANOVA followed by Tukey post hoc test, * significantly (p<0.05) difference when compared with 100 mg/kg AlCl3 –treated group. AlCl3: Aluminum chloride; AEAD: Aqueous fruit pulp extract of A. digitata

Histological Examination

A micrograph of the CA3 region of the hippocampus in Wistar rats (H&E, ×250) is shown in Figure 4. Light microscopic examination of the sections of CA3 hippocampal region stained with histological stains (H&E) revealed relatively normal histoarchitectural features of pyramidal cells with intact nuclei while the 100 mg/kg $AlCl_3$-treated group revealed degenerative changes in pyramidal cells which were evident with karyolytic and pyknotic nuclei. However, examinations of the 595 mg/kg ascorbic acid–treated group revealed mild distortion of the CA3 region. Moreover, 100 mg/kg $AlCl_3$ +500 mg/kg but AEAD–treated group, 100 mg/kg $AlCl_3$ +1500 mg/kg but AEAD–treated group, and 100 mg/kg $AlCl_3$ +595 mg/kg ascorbic acid–treated group revealed restoration of the neurodegenerative changes (Figure. 4).

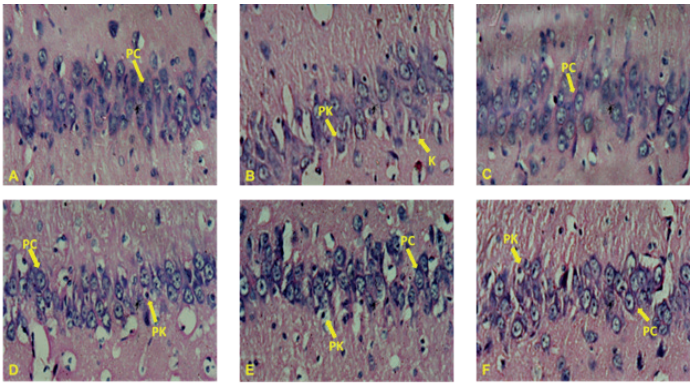

Figure. 4: Micrograph of CA3 Region of Wistar rats' Hippocampus (H&E x250).

A= H2O (2ml) –treated group with relatively normal cytoarchitectural features: pyramidal cells (PC) B= 100 mg/kg AlCl3 -treated group with distortion in the cytoarchitectural features: pkynosis (PK) and karyolysis (K). C= 595 mg/kg ascorbic acid–treated group with relatively normal cytoarchitecture feature: pyramidal cells (PC). D= 100 mg/kg but AlCl3 +500 mg/kg but AEAD –treated group with relative preservation of cytoarchitecture features: pyramidal cells (PC) and pyknosis (PK) E= 100 mg/kg AlCl3 +1500 mg/kg but AEAD –treated group with relative preservation of cytoarchitecture features: pyramidal cells (PC) and pyknosis (PK) F= 100 mg/kg

AlCl3 + 595 mg/kg ascorbic acid –treated group with relative preservation of cytoarchitecture features pyramidal cells (PC) and pkynosis (PK).

Discussion

This study assessed the neuroprotective activity of AEAD on cognitive functions, and the hippocampal CA3 region of Wistar rats exposed to aluminium chloride using phytochemical, neurobehavioral, histological, and biochemical analysis.

The phytochemical assessment of the AEAD; showed the presence of glycosides, flavonoids, triterpenes and saponins in the extract. Anani *et al*.[29], and Ogunleye *et al*.[30] reported similar phytochemicals constituents as observed in this study which have been suggested as possible substitute in reducing inflammation and oxidative stress linked to degenerative illnesses[14,31].

The physical observation showed that rats were very active with no visible physical changes observed while decreased physical activity, and signs of toxicity; gnawing and restlessness, scratching of the mouth and nostrils, watery faeces and loss of appetite were observed in the rats treated with aluminium. Abodunrin *et al*.[32] reported signs of weakness in Wistar rats exposed to aluminium sulphate. Kpatcha *et al*.[33] had also reported an increase in physical activities in Wistar rats administered with *A. digitata* when compared with other groups.

The toxic level of a substance and its implication on health can be observed through the body weight analysis[32,34]. This study clearly showed a progressive increase in the mean body weights of the rats throughout the co-administration of aluminium chloride with *A. digitata* fruit pulp extract, and aluminium chloride with ascorbic acid. However, the degree of weight gain by the rats treated with only aluminium chloride throughout the experiment was less than that of the weight gain by rats treated with *A. digitata* fruit pulp extract and ascorbic acid. The significant difference in body weight could be attributed to the toxic effect of aluminium chloride that might have slowed down the degree of weight gain by the rats exposed to aluminium. Justin *et al.* [35], Abodunrin *et al*. [34], and Leal *et al*. [36] reported a reduction in body weight gain from aluminium exposure as a result of less water and food consumed with transient diarrhoea. Oyewopo *et al.*[37] reported an improved body weight gain in Wistar rats after treatment with *A. digitata* fruit pulp aqueous extract following chemically-induced toxicity. The result of the ascorbic acid-treated group is in line with the findings of Sallam *et al.*[38] who reported that ascorbic acid was able to ameliorate the toxic effect of aluminium on the body weight of animals treated with ascorbic acid.

Memory is the mechanism by which knowledge is stored from learning, which gathers knowledge about experiences throughout time[39,40]. The administration of aluminum significantly increased the amount of time it took the experimental animals to find the concealed platform in the Morris water maze test for memory and learning. This could be linked to memory impairment, which is brought about by neuronal death, distortion in the pyramidal cells' overall morphology, and the hippocampal CA3 region. These changes indicate that the brain region that projects into the pyramidal layer and the CA3 region of the hippocampus will no longer be active, including in terms of memory and learning[41,42]. The results of this study showed a remarkable decrease in the mean-time it took the animals to trace the hidden platform in the Morris water maze test for spatial learning and memory in the treatments with *A. digitata* fruit pulp extract and ascorbic acid groups. Thus, the study has showed the ameliorative effects of *A. digitata* fruit pulp extract and ascorbic acid on spatial learning and memory in the experimental animals induced with aluminium chloride toxicity. The ameliorative properties of *A. digitata* on aluminium-induced memory impairment could be a result of the aforementioned phytochemical constituent which are rich in antioxidants that act by scavenging free radicals, thereby preventing oxidative stress[43].

Several heavy metals such as mercury, cadmium, lead, aluminium, and other organic compounds can damage the nervous tissues[44]. Aluminium has been reported to cause decreased cellular population, neurodegenerative changes and change in the overall shape of the hippocampal CA3 area and pyramidal cells[44]. These changes suggest that the area of the brain that projects into the pyramidal layer and the CA3 region of the hippocampus may lose its capacity for memory and learning[35,41].

Histologically, when comparing the $AlCl_3$-treated group to the control group, sections of the hippocampal CA3 region of the rats showed varying degrees of histological changes, including the presence of degenerating pyramidal cells that were visible with karyolytic and pyknotic nuclei indicating the toxic effects of aluminum exposure. This suggests that the hippocampus's functions in learning, memory formation, information storage, and retrieval may be compromised. The results of this investigation concur with those of Justin et al. [35] and Yu et al. [41] who reported that rats treated with aluminium revealed hippocampal neurodegenerative alterations that were linked to the animals' memory deficit. The results of these investigations are similar to other studies that reported that several heavy metals including mercury, cadmium, lead, and other organic compounds can damage the nervous tissues[45,46].

The histological sections of the AEAD-treated groups and the vitamin C-treated groups showed remarkable restoration of the hippocampal histoarchitecture with mild neurodegenerative changes. This outcome is consistent with the findings of the beneficial effects of *A. digitata* aqueous extracts on histopathological indices such as neuroprotection[37], and hepatoprotection[21]. The observed amelioration of histopathological degeneration in the hippocampus could equally be attributed to the antioxidant property of the plant [47] and free radical scavenging actions of the phytochemicals flavonoids and saponins in aqueous extract and as suggested by Musa *et al.*[31]. The ascorbic acid-treated group, in relation to the aluminium-exposed group, was observed to have ameliorated the histological insults incurred by the latter. Comparatively, the 1500 mg/kg doses of aqueous extract of *A. digitata* ameliorated the neurohistopathologic effects of aluminium much better than that observed with the ascorbic acid-treated group. This lends belief to the reports that *A. digitata* extracts compare favourably with ascorbic acid in protecting the brain against neurotoxicity in rats because of its high antioxidant properties[43,48].

In conclusion, the results obtained from this study demonstrated the neuroprotective potential of aqueous fruit pulp extract of *Adansonia digitata* against aluminium chloride-induced CA3

hippocampal neurotoxicity in Wistar rats. The phytochemicals present in the extract which are known for their antioxidant properties may contribute to this neuroprotective property. However further studies are recommended to ascertain the efficacy of *Adansonia digitata* as a therapeutic option for oxidative stress-associated neuropathologies in animal models. This will aid in the development of effective therapies in the future.

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Patient informed consent

There is no need for patient informed consent.

Ethics committee approval

This study's ethical approval, with approval code ABUCAUC/2018/070 was granted by the Ahmadu Bello University Ethics Committee on Animal Use and Care.

Conflict of interest

There is no conflict of interest to declare.

Financial support and sponsorship

No funding was received.

Author contribution subject and rate

- Sadiya Suleiman (20%): Design the research, data collection and analyses, wrote the whole manuscript and contributed with comments on research design and slides interpretation.
- Sunday Samuel Adebisi (10%): Organized the research, supervised the article write-up and organized the research and supervised the article write-up.
- Sunday Abraham Musa (10%): Design the research, data collection analyses and wrote the whole manuscript, organized the research and supervised the article write-up.
- Ubong Udeme Ekpo (20%): Contributed with comments on research design, slides interpretation, contributed with comments on manuscript organization and write-up.
- Stephen Samuel Lazarus (20%): Contributed with comments on research design, slides interpretation, contributed with comments on manuscript organization and write-up.
- Gbenga Peter Oderinde (20%): Contributed with comments on research design, slides interpretation, contributed with comments on manuscript organization and write-up

References

1. Abu-Taweel, G.M., Ajarem, J.S., & Ahmad, M. (2012). Neurobehavioral toxic effects of perinatal oral exposure to aluminum on the developmental motor reflexes, learning, memory and brain neurotransmitters of mice offspring. Pharmacology, Biochemistry, and Behavior, 101, 49-56.
2. Nampoothiri, M., John, J., Kumar, N., Mudgal, J., Nampurath, G.K., & Chamallamudi, M.R. (2015). Modulatory role of simvastatin against aluminium chloride induced behavioural and biochemical changes in rats. Behavioural Neurology, 2015, 210169.
3. Anwar, H.M., Georgy, G.S., Hamad, S.R., Badr, W.K., El Raey, M.A., Abdelfattah, M.A.O., Wink, M., & Sobeh, M. (2021). A Leaf Extract of Harrisonia abyssinica Ameliorates Neurobehavioral, Histological and Biochemical Changes in the Hippocampus of Rats with Aluminum Chloride-Induced Alzheimer's Disease. Antioxidants, 10, 947.
4. Cao, Z., Wang, F., Xiu, C., Zhang, J., & Li, Y. (2017). Hypericum perforatum extract attenuates behavioral, biochemical, and neurochemical abnormalities in Aluminum chloride-induced Alzheimer's disease rats. Biomedicine & Pharmacotherapy, 91, 931-937. [CrossRef]
5. Hussien, H.M., Abd-Elmegied, A., Ghareeb, D.A., Hafez, H.S., Ahmed, H.E.A., & El-moneam, N.A. (2018). Neuroprotective effect of berberine against environmental heavy metals-induced neurotoxicity and Alzheimer's-like disease in rats. Food and Chemical Toxicology, 111, 432-444. [CrossRef]
6. Aboelwafa, H. R., El-Kott, A. F., Abd-Ella, E. M., & Yousef, H. N. (2020). The possible neuroprotective effect of silymarin against aluminum chloride-prompted Alzheimer's-like disease in rats. Brain Sciences, 10(9), 628.
7. Niu, Q. (2018). Overview of the relationship between aluminum exposure and health of human being. Neurotoxicity of Aluminum, 1-31.
8. Fernandes, R. M., Corrêa, M. G., Aragão, W. A. B., Nascimento, P. C., Cartágenes, S. C., Rodrigues, C. A., ... & Lima, R. R. (2020). Preclinical evidences of aluminum-induced neurotoxicity in hippocampus and pre-frontal cortex of rats exposed to low doses. Ecotoxicology and environmental safety, 206, 111139.
9. Klotz, K., Weistenhöfer, W., Neff, F., Hartwig, A., van Thriel, C., & Drexler, H. (2017). The health effects of aluminum exposure. Deutsches Ärzteblatt International, 114(39), 653.
10. Olanrewaju, J., Akinola, O., Sunday, Y.O., Joshua, O., Sunday, F., Ukonne, U., Desalu, A., & Olanrewaju, J. (2018). Effects of virgin coconut oil on aluminium chloride-induced alzheimer-like dementia in the prefrontal cortex. Journal of Advanced Medical and Pharmaceutical Sciences, 18, 1-12.
11. Li, Q., Liu, H., Alattar, M., Jiang, S., Han, J., Ma, Y., & Jiang, C. (2015). The preferential accumulation of heavy metals in different tissues following frequent respiratory exposure to PM 2.5 in rats. Scientific Reports, 5, 1-8.
12. Onimisi, B.O., Zagga, A.D., Okolo, R.U., Abubakar, M.S., Oderinde, G.P., Usman, Z., Akor, J., Ahmed, A.M., & Idris, S. (2023). Moringa oleifera Ameliorates Chlorpyrifos-Induced Hippocampal Region Histoarchitectural Changes in Wistar rat. Journal of Anatomical Sciences, 14(1).
13. Prakash, A., Shur, B., & Kumar, A. (2013). Naringin protects memory impairment and mitochondrial oxidative damage against aluminum-induced neurotoxicity in rats. International Journal of Neuroscience, 123, 636-645.
14. Edobor, H. D., Musa, S. A., Umana, U. E., Oderinde, G. P., & Agbon, A. N. (2021). Neuroprotective effect of Phoenix dactylifera (date palm) on paraquat triggered cortico-nigral neurotoxicity. Journal of Neurobehavioural Sciences, 8, 199-208.
15. Rimpav, V., Cerdan, C., Vila, R. and Iglesias, J. (1999). Antioxidant activity of some extracts from plants used in the traditional medicine of North-African countries (II). Phytotherapy Research, 13(2): 128-132.
16. Venter, S. M., and Witkowski, E. T. F. (2010). Baobab (Adansonia digitata L.) density, size-class distribution and population trends between four land-use types in Northern Venda, South Africa. Forest Ecology and Management, 259: 294-300.
17. El-Rawy, E. M., Gergis, S. M., Bazaid, S., & El-Mougy, S. A. (1997). The immunostimulant effect of Adansonia digitata on the immune response of chicken vaccinated with avian cholera vaccine. Journal of Egyptian Veterinary Medical Association, 57(4), 959-970.

18. Addy, H.A. (2009). Adansonia digitata L. (baobab) - a review of traditional uses, phytochemistry and pharmacology. African Journal of Traditional, Complementary and Alternative Medicines, 6(4), 282-285.
19. Hussain, M. I., Farooq, M., & Syed, Q. A. (2020). Nutritional and biological characteristics of the date palm fruit (Phoenix dactylifera L.)–A review. Food Bioscience, 34, 100509.
20. Younas, A., Naqvi, S. A., Khan, M. R., Shabbir, M. A., Jatoi, M. A., Anwar, F., ... & Aadil, R. M. (2020). Functional food and nutra-pharmaceutical perspectives of date (Phoenix dactylifera L.) fruit. Journal of food biochemistry, 44(9), e13332.
21. Al-Qarawi, A. A., Abdel-Rahman, H., Ali, B. H., Mousa, H. M., & El-Mougy, S. A. (2005). The ameliorative effect of dates (Phoenix dactylifera L.) on ethanol-induced gastric ulcer in rats. Journal of ethnopharmacology, 98(3), 313-317.
22. Trease, G. E., & Evans, W. C. (1989). Pharmacognosy (13th edn). Bailliere Tindall, London, 176-180.
23. Lorke D. (1983). A new approach to practical acute toxicity testing. Archives of toxicology, 54(4), 275–287.
24. Morris, R. (1984). Developments of a water-maze procedure for studying spatial learning in the rat. Journal of neuroscience methods, 11(1), 47-60.
25. Ige, S. F., Akhigbe, R. E., Edeogho, O., Ajao, F. O., Owolabi, O. Q., Adegunlola, G. J., & Oyekunle, O. A. (2011). Hepatoprotective activities of Allium cepa in experimental liver injury. Pharmacognosy Research, 3(1), 16-20.
26. Rukkumani, R., Aruna, K., Varma, P. S., Rajasekaran, K. N., & Menon, V. P. (2004). Comparative effects of curcumin and an analog of curcumin on alcohol and PUFA induced oxidative stress. J Pharm Pharm Sci, 7(2), 274-283.
27. Fridovich, I. (1989). Oxygen radicals from acetaldehyde. Free radical biology & medicine, 7(5), 557-558.
28. Ohkawa, H., Ohishi, N., & Yagi, K. (1979). Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Analytical biochemistry, 95(2), 351-358.
29. Anani, K. (2000). Investigation of medicinal plants of Togo for antiviral and antimicrobial activities. Pharmaceutical Biology, 38, 40-45.
30. Ogunleye, O. O., Jatau, I. D., Natale, A. J., Obaloto, O. O., Adetutu, A. E., & Salifu, A. O. (2019). Aqueous extract of fruit pulp of Adansonia digitata (Linn): Phytochemical screening and in vitro antitrypanosomal effect. Nigerian Veterinary Journal, 40(1), 35-43.
31. Musa, S., Edobor, H., Umana, U., Oderinde, G., Olatomide, O., Zachariah, R., ... & Agbon, A. (2022). Histological Assessment of the Effect of Phoenix Dactylifera (Date Palm) on Cortico-ponto-cerebellar Structures in Paraquat Model of Neurotoxicity: Date and Cortico-ponto-cerebellar Structures. Journal of Medical and Basic Scientific Research, 3(2), 105-116.
32. Abodunrin, K., Fakunle, B., Saka, O., Abijo, A., Akinboro, A., & Ige, S. (2020). Effects of Aluminium Sulphates on Weight and Structure of Adrenal Gland of Adult Wistar Rats. International Journal of Human Anatomy, 2, 40-46.
33. Kpatcha, T., Dosseh, K., Idoh, K., Agbonon, A., & Gbéassor, M. (2016). Effect of Adansonia digitata L. on physical performance and haematological parameters in rats. African Journal of Science Research, 5(5), 01-06.
34. Sharma, P., & Mishra, K. (2006). Amelioration of fumonisin B1 hepatotoxicity in mice by depletion of T cells with anti-Thy-1.2. Reproductive Toxicology, 21, 313-321.
35. Justin, T. A., William Raja, T. R., Manivasagam, T., Janakiraman, U. and Essa, M. M. (2016) Hesperidin ameliorates cognitive dysfunction, oxidative stress and apoptosis against aluminium chloride induced rat model of Alzheimer's disease. Nutrition Neuroscience(in press).
36. Leal, F. A., Gonçalves, G. D., Soncini, J. G. M., Staurengo-Ferrari, L., Fattori, V., Verri Jr, W. A., & Fernandes, G. S. (2022). Exposure to aluminium chloride during the peripuberal period induces prostate damage in male rats. Acta Histochemica, 124(1), 151843.
37. Oyewopo, O. A., Olaniyi, K. S., Oywopo, C. I., & Morakinyo, K. S. (2017). Adansonia digitata ameliorates carbon tetrachloride-induced cerebello-pituitary dysfunction in adult male Wistar rats. International Journal of Health and Allied Sciences, 6, 158-162.
38. Sallam, S. M., Naseer, M. E., Yousef, M. S., El-morsy, A. M., Mahmoud, S. A., & Yousef, M. I. (2005). Influence of Aluminium Chloride and Ascorbic Acid on performance, digestibility, caecal microbial activity and biochemical parameters of rabbits. Research Journal of Agriculture and Biological Sciences, 1(1), 10-16.
39. Gupta, A., Singh, M. P., & Sisodia, S. S. (2018). A review on learning and memory. Journal of Drug Delivery and Therapeutics, 8(2), 153-157.
40. Angelopoulou, E., & Drigas, A. (2021). Working memory, attention and their relationship: A theoretical overview. Research, Society and Development, 10(5), e46410515288-e46410515288.
41. Yu, L., Jiang, R., Su, Q., Yu, H., & Yang, J. (2014). Hippocampal neuronal metal ion imbalance related oxidative stress in a rat model of chronic aluminium exposure and neuroprotection of meloxicam. Behavioural and Brain Functions, 10, 6.
42. Schröder, H., Moser, N., Huggenberger, S., Schröder, H., Moser, N., & Huggenberger, S. (2020). The mouse hippocampus. Neuroanatomy of the Mouse: An Introduction, 267-288.
43. Otong, S., Musa, S. A., Danborno, B., Sambo, S. J., & Dibal, N. I. (2021). Adansonia digitata ameliorates lead-induced memory impairments in rats by reducing glutamate concentration and oxidative stress. Egyptian Journal of Basic and Applied Sciences, 9(1), 1-10.
44. Ogunlade, B., Adelakun, S. A., & Agie, J. A. (2022). Nutritional supplementation of gallic acid ameliorates Alzheimer-type hippocampal neurodegeneration and cognitive impairment induced by aluminum chloride exposure in adult Wistar rats. Drug and Chemical Toxicology, 45(2), 651-662.
45. Budson A. E. (2009). Understanding memory dysfunction. Neurologist, 15:71-79.
46. Farina, M., Rocha, J. B. and Aschner, M. (2011). Mechanisms of methylmercury-induced neurotoxicity: Evidence from experimental studies. Journal of life Sciences, 89:555–563.
47. Parfait, B., Jean, B. G., Roger, P., Hervé, N. A., Balbine, K. K., Guillaume, C. W., & Germain, S. T. (2022). Antioxidant and Anticholinesterase Properties of the Aqueous Extract of Balanites aegyptiaca L. Delile Fruit Pulp on Monosodium Glutamate-Induced Excitotoxicity in Swiss Mice. Evidence-Based Complementary and Alternative Medicine, 2022(1), 7576132.
48. Rita, K., Moncada, M., Pintão, A., Bernardo, A., Silva, L., & Mesquita, F. (2019). Effect of Adansonia digitata L. fruit on postprandial glycaemia in non-diabetic adults. Annals of Medicine, 51(sup1), 159-159.