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Original Research Article

Effect of *Coco's nucifera liquid* (Coconut Water) on the Hippocampus of Aluminum Chloride Induced Neurodegenerative Disorder

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Abstract

Neurodegenerative disorders, are marked by progressive neuronal loss and cognitive decline. Aluminum chloride ($AlCl_3$) exposure has been shown to induce neurodegenerative changes through oxidative damage and inflammation. This study investigated the neuroprotective potential of *Cocos nucifera* (coconut water) on $AlCl_3$-induced neurodegeneration in Wistar rats. Thirty-two adult rats were randomly assigned into eight groups: Group I control (distilled water only), Group II, inducement of ND's ($AlCl_3$-only), Group III (donepezil-only), Group IV: ND's + donepezil ($AlCl_3$ and donepezil), Group V: ND's + (coconut water 500mg/kg), Group VI: ND's+ coconut water 1500mg/kg, Group VII: coconut water 500mg/kg only and Group VIII coconut water high dose (1500mg/kg only). Neurodegeneration was induced with $AlCl_3$ (10 mg/kg) for 14 days, followed by 14 days of treatment. Behavioral assessment using the Y-maze evaluated spatial working memory, while biochemical assays measured antioxidant markers—GSH, GPx, CAT, SOD, and MDA. Results revealed that coconut water treatment improved antioxidant status, with significant increases in CAT (group V - 3.60 ± 0.52, group VI - 3.75 ± 0.04) and GPx (group V - 0.04 ± 0.00, group VI - 0.04 ± 0.00) levels ($p < 0.05$) compared to the $AlCl_3$-induced group (CAT - 2.19 ± 0.02 and Gpx - 0.04 ± 0.00) indicating enhanced oxidative defense. Although no statistically significant differences were found in Y-maze performance across groups ($p > 0.05$). In conclusion, coconut water did not avert memory loss induced by AlCl3 toxicity at the dosage administered.

Keywords: Y-maze, Hippocampus, Biochemical assay, Neurodegeneration, Coconut water.

INTRODUCTION

Neurodegenerative disorders (NDs) are conditions characterized by the progressive wasting (atrophy) of structures in the central nervous system (CNS). The most prevalent of these include Alzheimer's disease (AD), Parkinson's disease (PD), Amyotrophic Lateral Sclerosis (ALS), and Huntington's disease (HD). Despite significant research, the occurrence of NDs is increasing rapidly. These disorders can be sporadic or inherited (familial), affecting specific brain regions and leading to severe motor and/or cognitive impairments like aphasia, apraxia, and agnosia. These functional difficulties ultimately disrupt a patient's personality, intellectual capacity, and ability to function socially and occupationally (Choonara *et al.,* 2009)

Coconut water, the transparent fluid from unripe coconuts, is one of the most naturally refreshing drinks available. Due to its excellent potassium content, it serves as a powerful thirst-quenching beverage, making it a popular staple throughout the tropical zone (Gallo *et al.,* 2019)

Coconut water is a traditional natural drink whose global popularity has grown steadily in recent years. In its fresh form, it serves as a hydrating and nutrient-rich beverage widely consumed around the world for its numerous health-promoting benefits (Rethinam & Krishnakumar, 2022). Cytokinins are key plant hormones found in coconut water. These compounds, such as kinetin and trans-zeatin (the first naturally occurring one, isolated in 1963), have also been found to have significant benefits for human health, including anti-aging, anti-cancer, and anti-clotting effects (Kende & Zeevaart, 1997).

Furthermore, micronutrients like vitamins and inorganic ions in the water are vital to the human body's antioxidant system (Evans & Halliwell, 2001).

In conditions that cause high production of reactive oxygen species (free radicals), these micronutrients help prevent oxidative damage to cell components. They function either by directly neutralizing free radicals or by acting as components of essential metalloenzymes (like superoxide dismutase) that remove these oxidizing agents (Evans & Halliwell, 2001).

Coconut water also contains a variety of other organic compounds, including sugars, lipids, amino acids, and enzymes. It is this vast and varied chemical composition, with many known and unknown compounds, that gives coconut water its recognized biological properties. This paper aims to summarize the known chemical makeup of coconut water (Tulecke *et al.,*1961).

MATERIALS AND METHODS

Preparation of Plant Extraction

After purchase of the coconut, the fibrous layer was removed manually and the coconut washed properly. The coconut shell was cracked and the water was extracted. After which the water was filtered using filter paper to get rid of the debris. The coconut water was boiled in a water bath at 40^0 C to produce a concentrated paste.

After this the coconut water extract (concentrate) was stored in a fridge at cooling temperature.

The Ld_{50} study was determined using Lorke's method. This method involves two phases; Phase I: 9 mice were used for the present study. Animals were divided into 3 groups with each group consisting of 3 animals. Group I received 500 mg/kg, group 2 received 1000 mg/kg and group 3 received 1500 mg/kg of extract. After 24 hours no death was recorded, phase 2 was carried out. Phase II: This phase also involved 9 mice divided into 3 groups with each group containing 3 mice each. GroupIV received 2000 mg/kg, groupV will receive 3000 mg/kg and groupVI 5000 mg/kg.

Study Models and Animal Handling

Thirty-two (32) adult male wistar rats weighing between 160g and 180g were obtained for the study and housed in the Animal House unit of the Department of Animal and Environmental Biology, Faculty of Sciences, Rivers State University, Nigeria. Standard cages were used and the models were maintained under the 12hr light/dark cycle with free access to feeds and water throughout the study. The route of donepzil and aluminum chloride administration was intraperitoneal route and coconut water administration was oral route.

Experimental Protocol

32 rats were grouped according to weight into 8 different groups, 4 animals per group
Group 1 served as the normal control group and received normal saline.
Group 2 served as the disease control group and received aluminum chloride only for the induction of neurodegenerative disorder.
Group 3 served as the standard drug control group and received donepezil only.
Group 4 served as the standard treatment group and received aluminum chloride for induction of neurodegenerative disorder followed by treatment with donepezil.
Group 5 served as the low-dose test group and received aluminum chloride for induction of neurodegenerative disorder followed by treatment with coconut water extract at a dose of 500 mg/kg body weight.
Group 6 served as the high-dose test group and received aluminum chloride for induction of neurodegenerative disorder followed by treatment with coconut water extract at a dose of 1500 mg/kg body weight.
Group 7 served as the extract control group and received coconut water extract only at a dose of 500 mg/kg body weight.
Group 8 served as the extract control group and received coconut water extract only at a dose of 1500 mg/kg body weight.

Induction of Neurodegenerative disorder

Aluminum chloride was administered 14 days at a dosage of 10mg/kg (Zarneshan *et al.,* 2025)

Y-maze procedure

Equipments

- Y-maze apparatus (3 arms of equal dimensions, 30 cm long, 10 cm wide, 15 cm high walls)
- Video camera and recording system
- Stopwatch or timer
- Cleaning supplies (70% ethanol)
- Data recording sheet

Before the test, the maze was thoroughly with 70% ethanol and allowed to dry completely
Camera was positioned to capture all three arms clearly from above
Arms were labeled as A, B, and C
The room was acclimatized to appropriate lighting, minimal noise
Animals were brought to the testing room 30 minutes before testing for habituation

Procedure

Trial 1

Step 1: The animal was placed in the center of the Y-maze facing away
Step 2: Timer and video recording were started immediately
Step 3: The animal was allowed to explore freely for 5 minutes

Step 4: Time spent in each arm (A, B, C) during exploration was recorded. (An animal is considered "in an arm" when all four paws have entered that arm).
Step 5: After the trial ended, the animal was removed and returned to its home cage.
Step 6: All arms were cleaned thoroughly with 70% ethanol to remove odor cues.
Step 7: The maze was allowed to dry for 2 minutes to allow odors to dissipate.
Trial 2
Step 8: An inter-trial interval of 30 minutes was observed.
Step 9: Steps 1–6 was repeated exactly as in Trial 1.
Step 10: The maze was cleaned again after completion.

Data Analysis

Data was expressed as mean ±SD. Mean difference between the control and treatment was tested using one-way analysis of variance (ANOVA). Values were considered statistically significant when p is less than or equal to 0.05($p \leq 0.05$).

The difference between the sample mean was evaluated using the Bonferroni post-hoc test.

RESULTS

The table presents the Mean ± standard Deviation (SD) for the percentage alternation variable for each of the eight groups in two different states: Before and After. The low F-values and high p-values (around 0.8) strongly suggest there was no significant difference in the percentage alternation measure across the eight groups, neither before nor after the intervention.

Table 1: The effect of coconut water on the memory of rats induced with neurodegenerative disorder using Y-maze

GROUPS	% Alternation (Before) Mean ± SD	% Alternation (After) Mean ± SD
Group1	$24.67^{a} \pm 6.81$	$23.33^{a} \pm 2.89$
Group2	$14.00^{a} \pm 12.77$	$13.33^{a} \pm 25.17$
Group3	$21.25^{a} \pm 14.57$	$25.25^{a} \pm 3.86$
Group4	$18.25^{a} \pm 3.50$	$19.00^{a} \pm 14.94$
Group5	$12.50^{a} \pm 17.68$	$12.50^{a} \pm 17.68$
Group6	$20.50^{a} \pm 3.32$	$17.50^{a} \pm 11.90$
Group7	$24.33^{a} \pm 9.02$	$23.67^{a} \pm 2.31$
Group8	$18.50^{a} \pm 12.50$	$20.75^{a} \pm 5.67$
F-value	0.479	0.502
p-value	0.838	0.822

The same superscript shows no significant difference among the groups at $p<0.05$.

ANTIOXIDANT STUDY

In the present study statistically, significant differences were observed among all the groups for the antioxidant markers GSH, GPX, and CAT (Table 4.2). For GSH and CAT, Group 1 shows the highest mean levels, indicating a stronger effect in this group compared to most others.

Conversely, there is no statistically significant difference among the groups for SOD and MDA ($p>0.05$), meaning these parameters are not affected by the difference between the groups.

Table 2: Antioxidant effect of coconut water on rats induced with neurodegenerative disorder using aluminum chloride

GROUPS	GSH Mean ± SD	GPX Mean ± SD	CAT Mean ± SD	SOD Mean ± SD	MDA Mean ± SD
Group1	$2.53^{a} \pm 0.11$	$0.06^{c} \pm 0.00$	$3.87^{b} \pm 0.40$	$0.43^{a} \pm 0.02$	$0.27^{a} \pm 0.02$
Group2	$1.82^{b} \pm 0.08$	$0.04^{a} \pm 0.00$	$2.19^{a} \pm 0.02$	$0.25^{a} \pm 0.03$	$0.53^{a} \pm 0.03$
Group3	$2.04^{b} \pm 0.21$	$0.04^{b} \pm 0.00$	$3.21^{a} \pm 0.44$	$0.31^{a} \pm 0.01$	$0.46^{a} \pm 0.01$
Group4	$1.95^{b} \pm 0.04$	$0.04^{b} \pm 0.00$	$2.56^{a} \pm 0.16$	$0.30^{a} \pm 0.02$	$0.50^{a} \pm 0.01$
Group5	$1.86^{b} \pm 0.04$	$0.04^{b} \pm 0.00$	$3.60^{b} \pm 0.04$	$3.32^{a} \pm 4.29$	$0.45^{a} \pm 0.06$
Group6	$2.00^{b} \pm 0.04$	$0.04^{b} \pm 0.00$	$3.75^{b} \pm 0.52$	$0.37^{a} \pm 0.06$	$0.39^{a} \pm 0.12$
Group7	$1.89^{b} \pm 0.05$	$0.04^{b} \pm 0.00$	$3.53^{b} \pm 0.60$	$0.39^{a} \pm 0.02$	$0.36^{a} \pm 0.10$
Group8	$2.00^{b} \pm 0.01$	$0.05^{b} \pm 0.00$	$3.94^{c} \pm 0.23$	$21.19^{a} \pm 29.43$	$0.34^{a} \pm 0.09$
F-value	12.03	25.30	$3.60^{b} \pm 0.04$	0.963	0.058
p-value	0.001	0.000	0.010	0.513	0.742

*The different superscript shows a significant different among the groups at $p<0.05$

Key: GSH – Glutathione; GPx - Glutathione peroxidase CAT- Catalase; SOD- Superoxide Dismutase MDA- Malondialdehyde

HISTOLOGY RESULTS

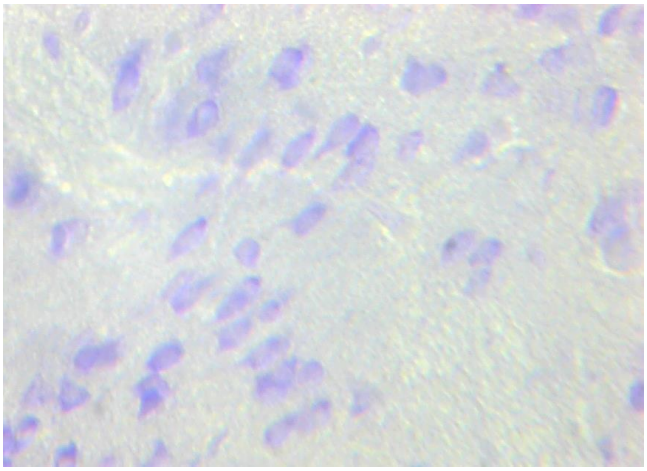

Plate I: Section of the hippocampus showed how normal hippocampus with normal pyramidal cells with vesicular nuclei, molecular layer (orange arrow) and polymorphic region with normal glial cells (blue arrow) x400

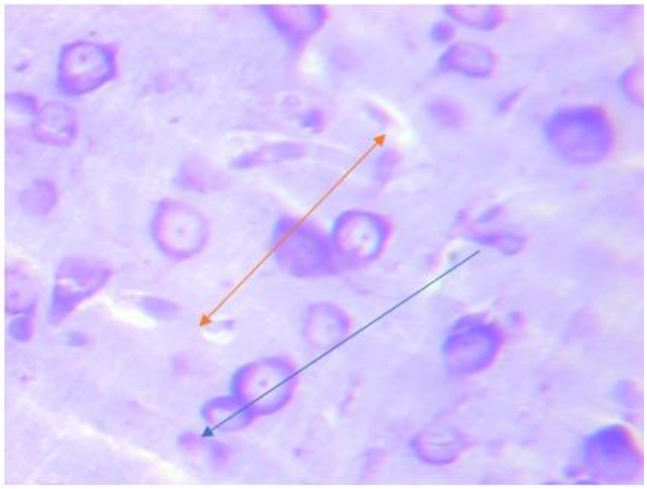

Plate II: Section of the hippocampus showed hippocampus dark stained cells with irregular sizes and shapes (blue arrow) and vacuolation noted in areas (orange arrow). The three different layers are also not intact (yellow arrow) x400

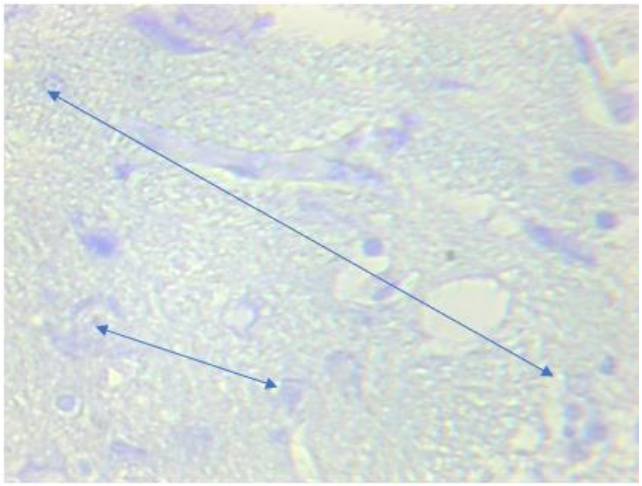

Plate III: Section of the hippocampus showed hippocampus with normal looking cells with intact layers (yellow arrow). However, there is mild cellular degenerative changes in areas. (blue arrow) x400

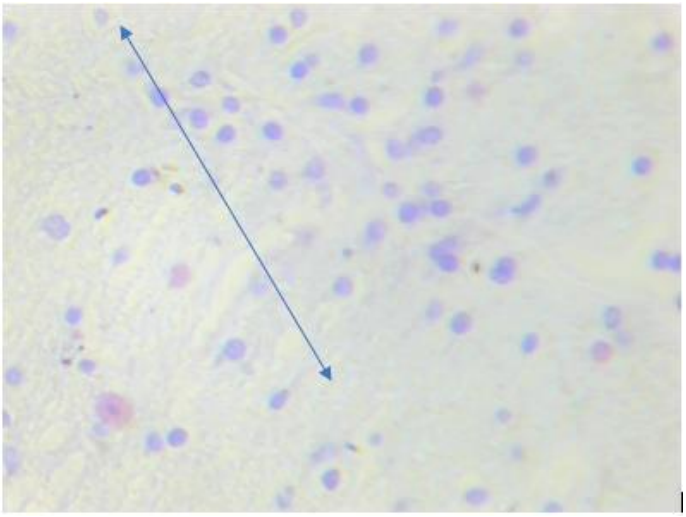

Plate IV: Section of the hippocampus showed hippocampi with normal layers and glial cells. There is however reduced number of layers in the pyramidal region with mild cellular shrinkage noted in areas (blue arrow) x400

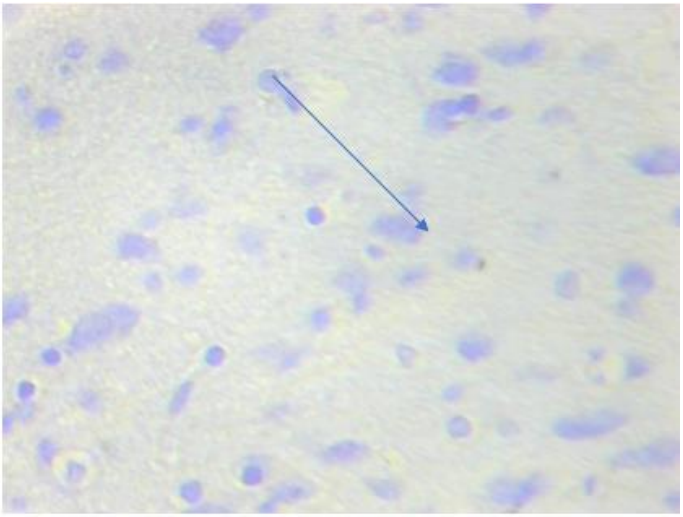

Plate V: Section of the hippocampus showed hippocampi with reduced number of layers (orange arrow) with mild cellular shrinkage in the pyramidal layer (blue arrow). x400

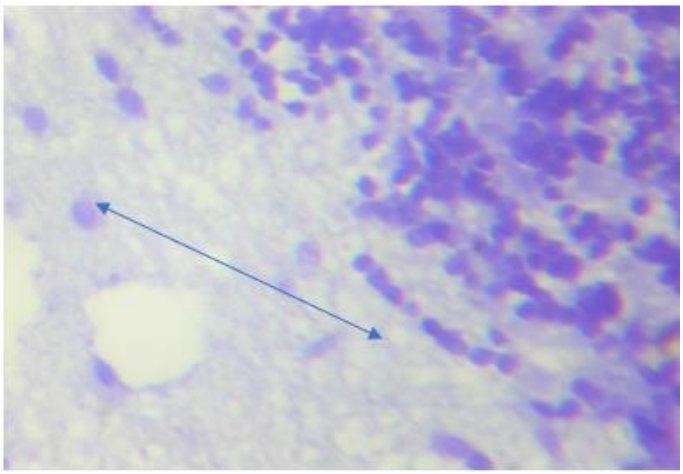

Plate VII: Section of the hippocampus showed normal looking hippocampus with abundant glial cells with vesicular nuclei (blue arrow) in the different layers of the hippocampus x400

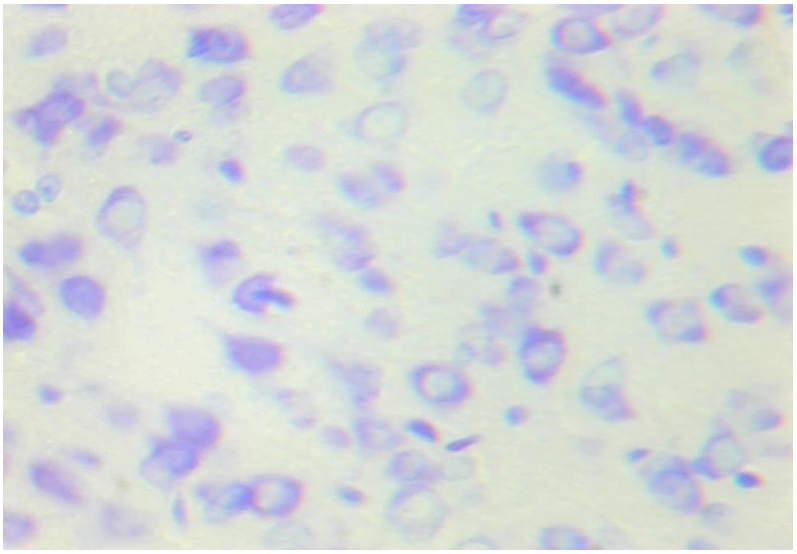

Plate VIII:

DISCUSSION

In the present study, MDA reduced, SOD, CAT and GPx increased when compared to the group induced with NDs. Significant increase was observed in CAT and GPx. This implies that coconut water may have provided antioxidant defense in AlCl3 induced NDs. This agrees with Alizadeh & Kheriouri (2019). They reported that plant extract capable of reducing MDA and increasing SOD, CAT and GPx enhanced the efficacy of the antioxidant defense system.

The y-maze test was used to evaluate spatial working memory and exploratory behavior before and after treatment. The highest Y-maze percentage of alternation was observed in Group 1 (control group). It was observed that the Y-maze % alternation for the group induced with $AlCl_3$ reduced. This implies impairment of spatial working memory. This agrees with Adelodun *et al.,* (2021). They reported a reduction in $AlCl_3$ induced group suggesting impairment in spatial working memory as a consequence of $AlCl_3$ exposure. This also agrees with Zarneshan *et al.,* (2025) They reported that a decrease in the activity of the AlCl3 induced group compared with the control group with a decrease in percentage alternation

In the present study, treatment with coconut water reduced percentage alternation in the high dose group. This implies that there was no recovery from memory loss. This result disagrees with Zarneshan *et al.,* (2025) they reported that treatment with polydatin increased percentage alternation thereby averting memory loss.

CONCLUSION

Coconut water did not avert memory loss induced by AlCl toxicity at the dosage administered.

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