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

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**Abstract** **Original Research Article**

Alzheimer's, Parkinson's, and Huntington's diseases are among the most disabling conditions affecting the nervous system. The aim of this study is to investigate the effect of coconut water on aluminum chloride–induced neurodegenerative disorder in the cerebellum in Wistar rats. Thirty-two (32) male Wistar rats were used for this study. Animals were grouped into eight (8) with each consisting of 4 animals each; Group I served as the control; Group II received aluminum chloride only (10 mg/kg); Group III received donepezil only; Group IV received aluminum chloride and donepezil; Groups V and VI were treated with aluminum chloride plus coconut water at doses of 500 mg/kg and 1500 mg/kg respectively; while Groups VII and VIII received coconut water 500 mg/kg and 1500 mg/kg. The experiment lasted twenty-eight days. Animals were sacrificed, the cerebellum was collected for histological analyses and blood samples were collected for biochemical analyses (GSH, GPx, CAT, SOD, and MDA). Behavioral assessment using the wire hang test evaluated motor coordination and neuromuscular strength. 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 b ± 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). An increase was observed in the mean time for wire hang test for animals in group I (control group). After treatment the mean time increased in the following groups III, IV, and VI. Coconut water improved motor coordination, and provided neuroprotective effect in Wistar rats.

**Keywords**: Neurodegeneration, $AlCl^3$, Coconut water, Wire hang test, Cerebellum.



## INTRODUCTION

Neurons are the building blocks of the nervous system which includes the brain and spinal cord. Neurons are central to the proper functioning of the human brain since they play a critical role in communication. Most neurons originate in the brain; however, neurons are present everywhere in the body (Lamptey *et al.,* 2022). During childhood, neural stem cells produce the majority of neurons, the number of which is significantly reduced in adulthood. Although neurons are not immortal, the progressive loss of neurons, neuron structure, and/or their functions, known as neurodegeneration, is central to the pathophysiology of several brain disorders and is also a major health concern (Lamptey *et al.,* 2022).

Neurodegenerative diseases (NDD) are diverse conditions characterized by selective dysfunction and ongoing loss of neurons, glial cells and the neural networks in the brain and spinal cord. Accordingly, they cause diverse problems, examples being movement (called ataxias), mental functioning (called dementias) and a person's ability to move, speak and breathe. NDD are incurable and debilitating conditions, and are becoming increasingly prevalent in part due to global population ageing (Technology Networks 2023). The most common neurodegenerative disorders are amyloidoses, tauopathies, and α-synucleinopathies (Dugger & Dickson, 2017).

The cerebellum is an outgrowth from the brainstem that plays a vital role in modulating movements (Carlson,2018). cerebellum is a vital component in the human brain as it plays a role in motor movement regulation and balance control. The cerebellum coordinates gait and maintains posture, controls muscle tone and voluntary muscle activity but is unable to initiate muscle contraction. Damage to this area

in human's results in a loss in the ability to control fine movements, maintain posture, and motor learning (Jimsheleishvili, S., & Diddze, M. 2023). The cerebellum, which is the largest part of the hindbrain, is located in the posterior cranial fossa, behind the fourth ventricle, the pons, and the medulla oblongata (Jimsheleishvili, S., & Diddze, M. 2023)

Coconut water is the clear liquid inside young coconuts. Coconut known as Cocos nucifers is a member of the palm tree family (Arecaceae).

Coconut water is the liquid found in the center of a young, green coconut. It helps nourish the fruit. As the coconut matures, which takes around 10–12 months, some of the liquid remains while the rest ripens into the solid white flesh known as coconut meat. Coconut water typically comes from young coconuts about 6–7 months of age, though it's also found in mature fruit. An average green coconut provides about 1/2–1 cup of coconut water (DiGiacinto & Spritzler, 2023).

Coconut water contains 94% water and very little fat. It should not be confused with coconut milk, which is made by adding water to grated coconut meat. Coconut milk contains about 50% water and is quite high in fat (DiGiacinto & Spritzler, 2023).

Coconut water possesses various functional properties, including antioxidant, antimicrobial, antidiabetic, anti-inflammatory, anti-hyperlipidemic, anti-cancer, anti-ulcerogenic, and cardioprotective effects (Shi et al.,2025). It has been incorporated into traditional medicine and dietary interventions in numerous cultures, particularly in Africa, India, and the Philippines, for the treatment of a multitude of conditions, including gastroenteritis, coronary heart disease, kidney diseases such as urolithiasis, and urinary tract infections (Shi. et al.,2025). Additionally, the content of phenolic compounds in coconut water increases with maturity, enhancing its antioxidant properties and nutritional value.Key bioactive constituents include catechin and several phenolic acids, such as syringic acid, salicylic acid, m-coumaric acid, p-coumaric acid, and p-hydroxybenzoic acid, which serve as significant sources of antioxidants in the prevention of disease The catechin extracted from coconut water showed antioxidant, antibacterial, and anti-cancer effects, interestingly, green coconut water exhibits a stronger free radical scavenging activity, higher antioxidant and anti-aging activities, and a richer profile of bioactive compounds compared to mature coconut water(Shi *et al.,* 2025).

## 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 degrees celcius to produce a concentrated paste.

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

The Ld50 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) adults 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)

**Wire hang test**

**Equipments**

The equipment used for the wire hang test included a metal wire grid with standard spacing of approximately 1 cm. soft bedding material or foam padding, was placed beneath the apparatus to cushion potential falls. A stopwatch or timer was used for time measurement, and a data recording sheet was used for documentation. Gloves were optionally used for animal handling.

**Pre-Test Preparation**

The testing area was prepared by placing adequate soft bedding or padding underneath the wire apparatus to minimize injury from falls. The wire grid was positioned at an appropriate height, typically 30 cm above the padding. Animals were acclimated to the testing room for 30 minutes prior to testing to reduce stress-related variability. A maximum trial duration was set as 180 seconds.

**Testing Procedure**

- Each animal was gently placed on top of the wire grid and allowed to grip the wire naturally with the 2 front paws. Care was taken to ensure the animal was calm and had a secure grip before proceeding.
- The wire was kept horizontally at a height of approximately 30 cm above the padded surface.
- Timing commenced immediately after the animal had gotten a grip on the wire. The wire was maintained in a steady horizontal position throughout the duration of the test.
- Timing was stopped when any of the following occurred: the animal fell completely from the wire; the maximum time limit of 180 seconds was reached.
- The amount of time spent by each animal hanging was recorded
- The procedure was repeated for 2 trials with a 3-minute interval between the 2 trials

**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

In the present study, a decrease in the mean time for wire hanging test was observed in group 2 (Induction of Neurodegenerative disorders only). Administration of Coconut water decreased the mean time in the following groups, 5 (NDs + Coconut water 500mg/kg), 7 (Coconut water 500mg/kg only) and 8 (Coconut water 1500mg/kg) (Table 1). An increase was observed in the mean time for wire hanging test for animals in group 1(control group). After treatment the mean time increased in the following groups, 3 (Induction of Donepezil standard drug only), 4 (NDs + donepezil standard drug), and 6 (NDs + Coconut water 1500mg/kg) (Table 1)

**Table 1: Effect of *Coco's nucifers liqiud* on the neuromuscular strength and motor coordination of wistar rats induced with neurodegenerative disorder using the wire hang test**

| GROUP | BEFORE MEAN ± SD | AFTER MEAN ± SD |
|---|---|---|
| **Group 1(control)** | 108.33 ± 24.66 | 141.67 ± 30.55 |
| **Group 2(Induction of NDs)** | 108.67 ± 35.81 | 55.00 ± 32.79 |
| **Group 3(Donepezil only)** | 105.75 ± 24.73 | 122.50 ± 17.08 |
| **Group 4(NDs + Donepezil)** | 68.50 ± 31.20 | 126.00 ± 20.77 |
| **Group 5(NDs + coconut water 500mg/kg)** | 97.00 ± 32.53 | 37.50 ± 23.33 |
| **Group 6(NDs + coconut water 1500mg/kg)** | 87.50 ± 43.00 | 103.00 ± 8.29 |
| **Group 7(coconut water 500mg/kg)** | 129.33 ± 62.14 | 79.33 ± 50.66 |
| **Group 8(coconut water 1500mg/kg)** | 178.50 ± 25.53 | 143.25 ± 15.84 |
| F- value | 6.122 | |
| P- value | 0.001 | |

In the present study statistically, significant differences was observed among all the groups for the antioxidant markers GSH, GPX, and CAT (Table 2). For GSH and CAT, Group 1 shows the highest mean levels, indicating a stronger effect in this group compared to most others. 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 *Coco's nucifers liqiud* on wistar rats induced with Neurodegenerative disorders**

| GROUPS | GSH MEAN ± SD | GPX MEAN ± SD | CAT MEAN ± SD | SOD MEAN ± SD | MDA MEAN ± SD |
|---|---|---|---|---|---|
| **Group1** | $2.53^{a\pm}$ 0.11 | $0.06^{c}$ ± 0.00 | $3.87^{b}$ ± 0.40 | $0.43^{a}$ ± 0.02 | $0.27^{a}$ ± 0.02 |
| **Group2** | $1.82^{b}$ ± 0.08 | $0.04^{a\pm}$ 0.00 | $2.19^{a}$ ± 0.02 | $0.25^{a}$ ± 0.03 | $0.53^{a}$ ± 0.03 |
| **Group3** | $2.04^{b}$ ± 0.21 | $0.04^{b}$ ± 0.00 | $3.21^{a}$ ± 0.44 | $0.31^{a}$ ± 0.01 | $0.46^{a}$ ± 0.01 |
| **Group4** | $1.95^{b}$ ± 0.04 | $0.04^{b}$ ± 0.00 | $2.56^{a}$ ± 0.16 | $0.30^{a}$ ± 0.02 | $0.50^{a}$ ± 0.01 |
| **Group5** | $1.86^{b}$ ± 0.04 | $0.04^{b}$ ± 0.00 | $3.60^{b}$ ± 0.04 | $3.32^{a}$ ± 4.29 | $0.45^{a}$ ± 0.06 |
| **Group6** | $2.00^{b}$ ± 0.04 | $0.04^{b}$ ± 0.00 | $3.75^{b}$ ± 0.52 | $0.37^{a}$ ± 0.06 | $0.39^{a}$ ± 0.12 |
| **Group7** | $1.89^{b}$ ± 0.05 | $0.04^{b}$ ± 0.00 | $3.53^{b}$ ± 0.60 | $0.39^{a}$ ± 0.02 | $0.36^{a}$ ± 0.10 |
| **Group8** | $2.00^{b}$ ± 0.01 | $0.05^{b}$ ± 0.00 | $3.94^{c}$ ± 0.23 | $21.19^{a}$ ± 29.43 | $0.34^{a}$ ± 0.09 |
| **F-value** | 12.03 | 25.30 | 6.10 | 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 Disnutase
**MDA-** Malondialdehyde

## RESULTS

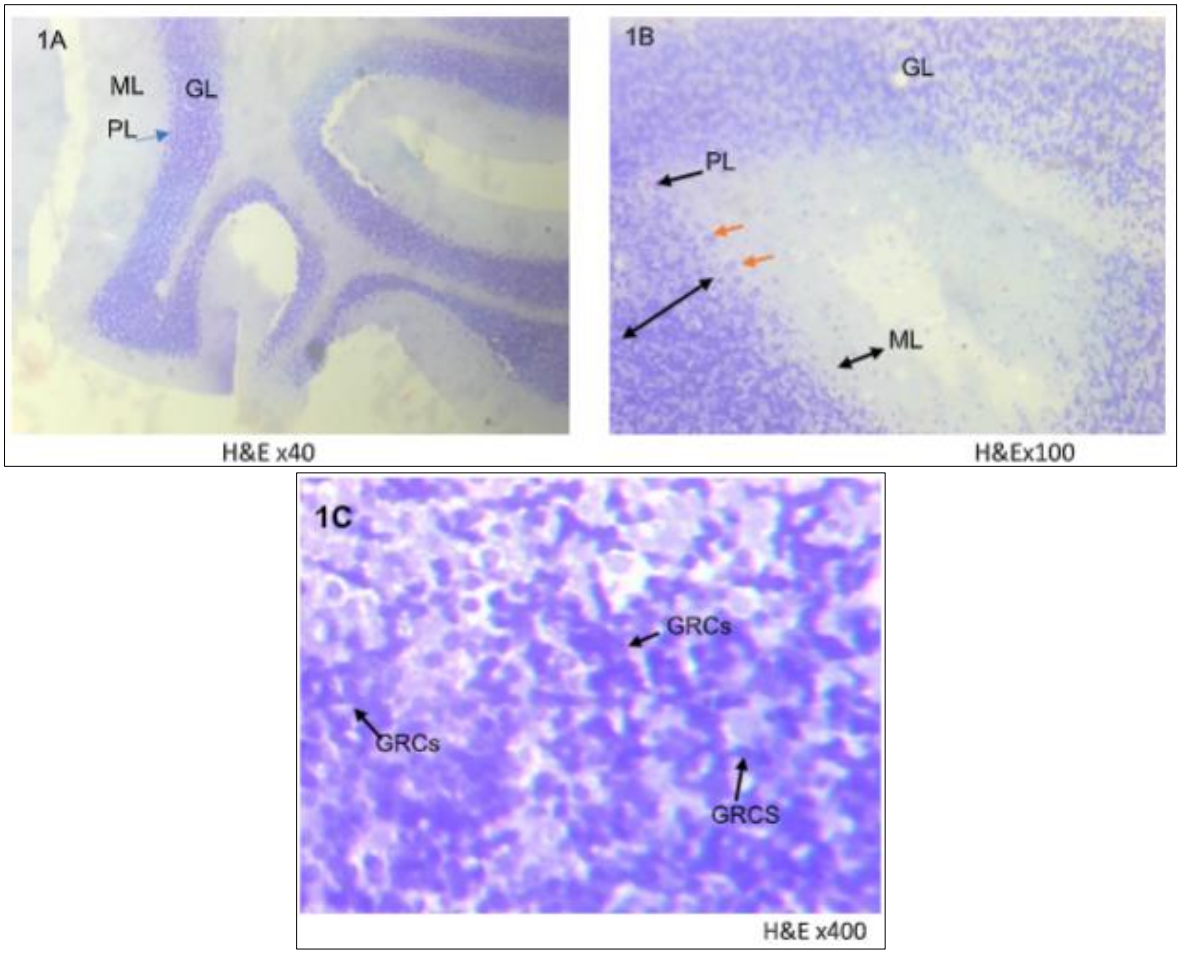


**Plate I: Section of cerebellum of brain tissue taken from Wistar rat from control group showed 1A with the molecular layer (ML), pyramidal cell layer (PCL) and the granular layers (GL). Section 1B showed the molecular layers with the stellate cells and basket cells, the pyramidal cell layer with the pyramidal cells (PC) and the granular layer with granule rosette cells. Section 1C displayed the granular layer (GL) with dark purple stained granule rosette cells (GRCs)**

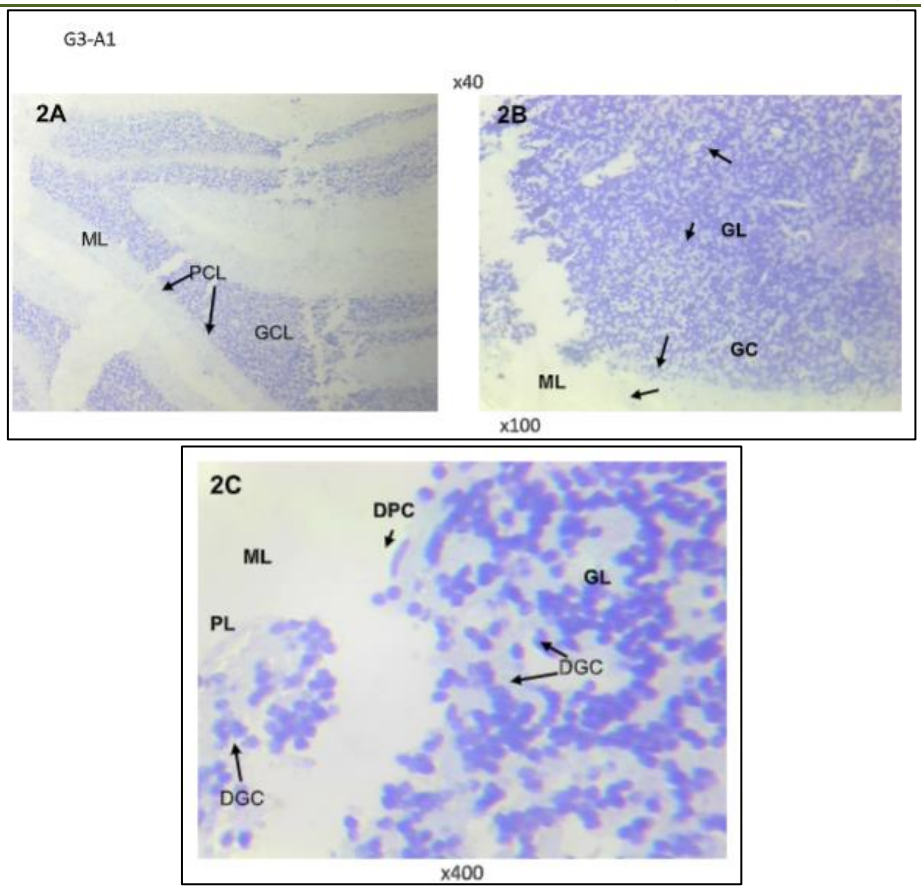


**Plate II: Photomicrograph of a section of cerebellum of brain tissue of Albino Wistar from group G3-A1. Section showed. Fig 2A showed the three layers of molecular layer (ML), pyramidal cell layer (PCL) and granular layer (GL), fig 2B showed that there is cellular degeneration of the 3 layers of the cerebellum with less glial cells in the molecular layer, degenerated pyramidal cells in the pyramidal layer and disorganized granular cells in the granular cell layer**

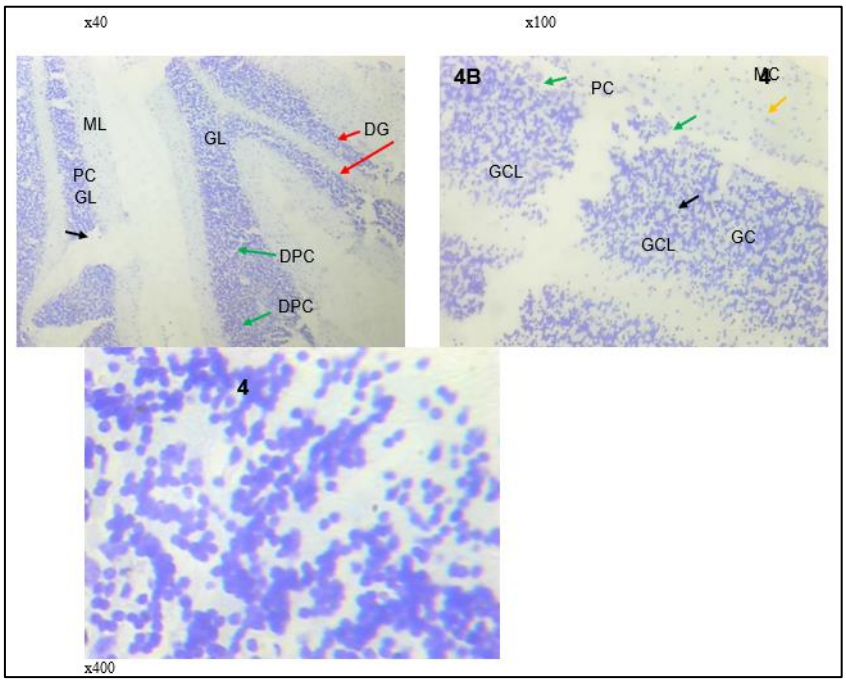


**Plate III: Photomicrograph of a Section of cerebellum tissue taken from the brain of Wistar rat from group G5-A1. Section showed distorted and disorganized Molecular layers (ML), Pyramidal cell layers (PL) and Granular areas (GL) of the cerebellum in Fig 4A. Fig 4B displayed degenerated pyramidal cells (DPC-Green arrows) in the pyramidal layer and Fig 4C showed cellular distortion and disorganization of the pyramidal cells (DPC) and granular cells (DGC) red arrows.**

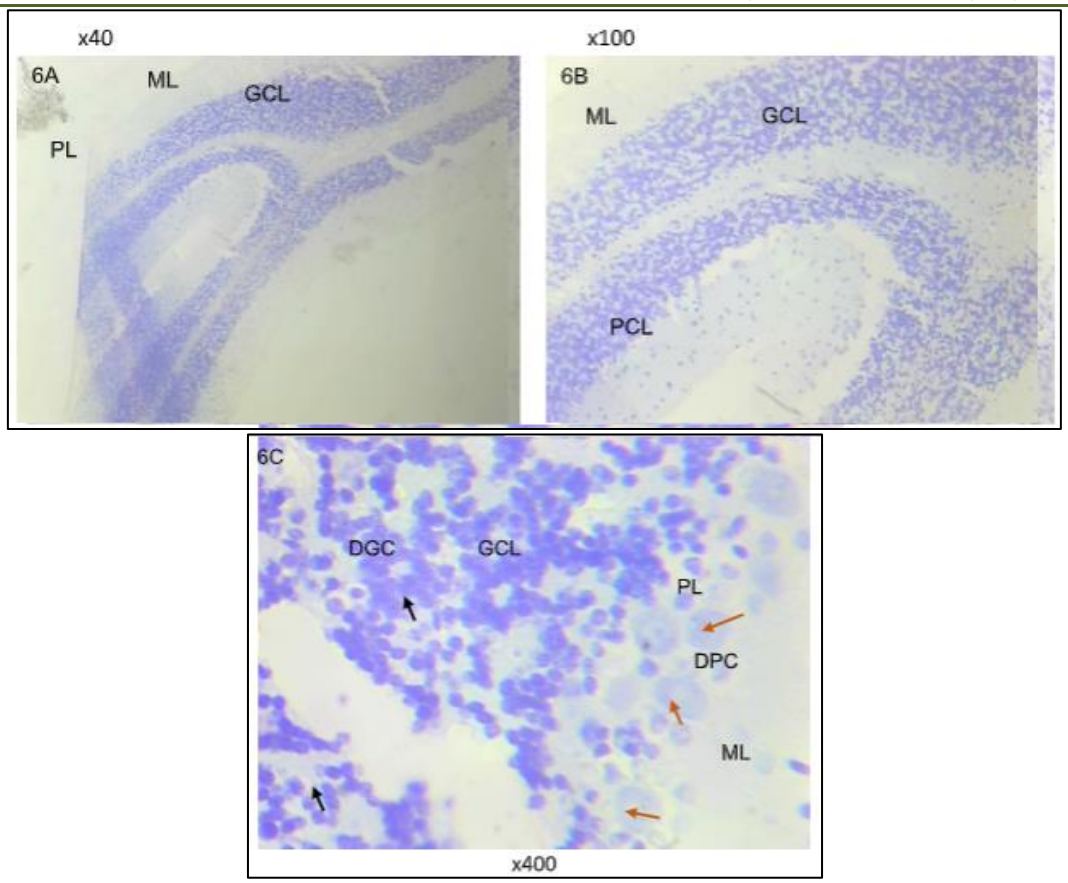


**Plate IV: Photomicrograph of a section of cerebellum of brain tissue taken from Wistar rats of group G6-A2, Section showed all three layers of the cerebellum with degeneration of basket and stellate cells at the molecular layer, karyolitic pyramidal cells (brown arrows) in the pyramidal cell layer (brown arrows) and degenerated granule cells**

G7-A1

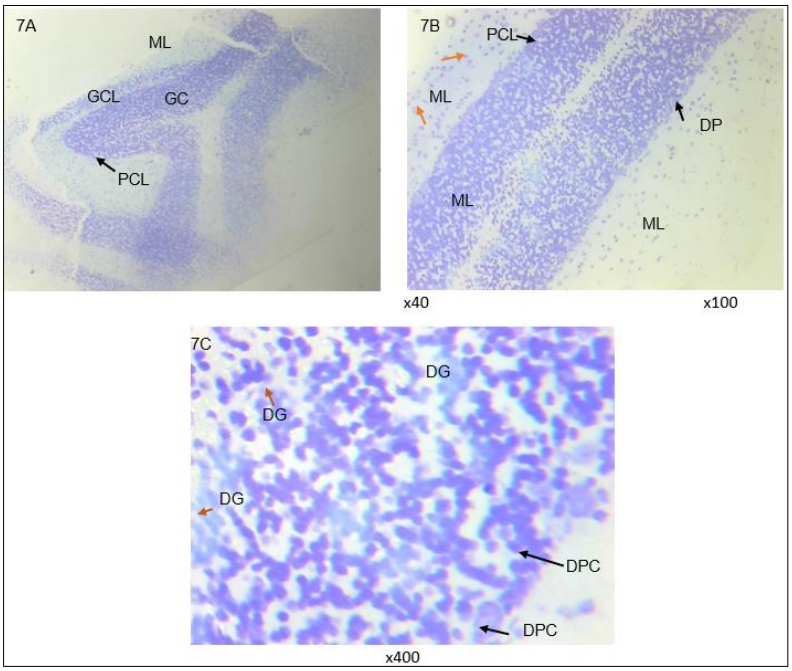


**Plate V: Photomicrograph of a section of cerebellum tissues taken from brain of Wistar from G7-A1. Section 7A showed the molecular layer (ML), Pyramidal cell layer (PL) and the granular cell layer (GCL). Section 7B showed stellate cells (Orange arrows) in the ML, pyramidal cells-PC (black arrows) in the pyramidal layer (PL) and karyolitic rosette granule cells (Brown arrows) at the granular layer (brown arrows)**

## DISCUSSION

In group 3(Donepezil only standard drug),4(NDs + Donepezil standard drug) and 6(NDs + coconut water 1500mg/kg high dose) an increase in grip strength was observed in the treatment groups. This implies that there may be an increase in motor co-ordination and muscle tone due to the treatment administered. This agrees with Adebiyi *et al.,* (2016), they reported that any substance capable of increasing the grip strength of an animal may likely increase motor coordination and muscle tone.

In group 6(NDs + coconut water 1500mg/kg high dose) the mean time (secs) increased. This implies that there was improvement in neuromuscular strength which may be due to administration of the plant extract (coconut water). This agrees with saadullah *et al.,* (2022) they reported an increased neuromuscular strength in animals with an increased mean time.

In the present study, MDA was 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.

## CONCLUSION

Coconut water improved motor coordination, and provided a neuroprotective effect in Wistar rat.

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