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Neuroprotective Effects of *Tetracarpidium Conophorum* in Wistar Rats Treated With Cadmium Acetate

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ABSTRACT

Cadmium (Cd) is an environmental heavy metal associated with neurotoxicity. This study investigates the neuroprotective effects of oilseed from *Tetracarpidium conophorum* (Müll. Arg.) on the medulla oblongata of Wistar rats (Rattus norvegicus) exposed to cadmium acetate ($Cd(CH_3COO)_2$) and behavioral changes in the medulla oblongata of Wistar rats. . Thirty adult female Wistar rats were allocated into five groups (n = 6): control (normal saline), sham (pellets/water only), cadmium acetate only (3.5 mg/kg, i.p.), cadmium acetate + T. conophorum oil seed (3.5 mg/kg i.p. + 0.5 ml/kg orally), and oil seed only (0.5 ml/kg orally). Treatments lasted 30 days. Body weight and neurobehavior (beam walk test) were recorded. Brain tissues were examined histologically (H&E, Luxol Fast Blue). Data were analyzed by one-way ANOVA followed by Tukey's post-hoc test; values are mean ± SEM; $p < 0.05$ was considered significant. Results showed that cadmium exposure produced weight loss trends and significant histological alterations in the medulla oblongata, including distorted unmyelinated nerve architecture, vacuolated pyramidal cells, and disrupted myelin staining. Co-treatment with *T. conophorum* oil seed preserved histological architecture relative to cadmium-alone animals, with increased Schwann cell presence and improved myelin staining. Behavioral deficits induced by cadmium were attenuated in the oil-treated group.In conclusion, *T. conophorum* oil seed mitigates cadmium-induced histopathological and behavioral changes in the rat medulla oblongata, supporting its potential as a natural neuroprotectant. Future studies should quantify oxidative stress biomarkers, inflammatory markers, and tissue cadmium to define mechanisms and translational feasibility as dietary or therapeutic agents.

Keywords: *Tetracarpidium conophorum,* cadmium acetate, neuroprotection, medulla oblongata, histopathology, antioxidants.

INTRODUCTION

Cadmium (Cd) is a heavy metal that poses significant health risks due to its widespread environmental presence and toxicity. It is classified as a human carcinogen by the North Carolina National Toxicology Program and is associated with various neurological disorders (Nanuam, n.d.). Cadmium exposure can occur through industrial processes, contaminated food, and smoking, leading to its accumulation in human tissues, particularly in the brain (Faroon et al., 2012). The neurotoxic effects of cadmium are well-documented, with studies indicating that it can disrupt neurotransmitter systems, impair synaptic transmission, and induce oxidative stress, resulting in neuronal damage (Gangolli, 1999; Wang & Du, 2013). The medulla oblongata, a critical region of the brainstem, plays a vital role in autonomic functions such as respiration and heart rate regulation(Cunha Neto et al., 2024). It is particularly vulnerable to oxidative stress due to its high lipid content and oxygen demand (Bauer & Bauer, 1999). Histological studies have shown that

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cadmium exposure can lead to significant alterations in the structure of the medulla oblongata, including distorted unmyelinated nerves and vacuolated pyramidal cells (Afifi & Embaby, 2016). These changes can contribute to cognitive deficits and behavioral disturbances, as evidenced by neurobehavioral assessments in animal models (Viaene et al., 2000).

Given the adverse effects of cadmium, there is a growing interest in identifying natural compounds that may mitigate its toxicity. One such candidate is *Tetracarpidium conophorum* (Müll. Arg.), commonly known as African walnut. This plant is rich in antioxidants, vitamins, and polyphenolic compounds, which have been shown to possess neuroprotective properties (Akomolafe et al., 2015; Kanu et al., 2015). Previous studies have demonstrated that extracts from *Tetracarpidium conophorum* can reduce oxidative stress and improve histological integrity in various tissues (Amaeze et al., n.d.; Tokunbo et al., 2023). However, the specific neuroprotective effects of this plant against cadmium-induced neurotoxicity remain underexplored. This study aims to investigate the neuroprotective effects of oil seed from *Tetracarpidium conophorum* on the histological components of the medulla oblongata in albino Wistar rats exposed to cadmium acetate. By assessing body weight changes, neurobehavioral performance, and histological alterations, this research seeks to elucidate the potential therapeutic role of *Tetracarpidium conophorum* in mitigating cadmium-induced neurotoxicity. The findings may contribute to the development of dietary strategies for preventing heavy metal toxicity and promoting brain health.

MATERIALS AND METHODS

Study Location

This research was conducted at the Animal House of the Department of Physiology, Benjamin Carson School of Medical Sciences, and Babcock University Teaching Hospital, Babcock University, Ilishan-Remo, Ogun State, Nigeria.

Experimental Animals

A total of thirty adult female Wistar rats (*Rattus norvegicus)* weighing between 150 to 200 g were procured from Babcock University Farmhouse. The rats were housed in plastic cages with net covers to ensure proper ventilation. They were acclimatized for fourteen days with access to distilled water and were fed twice daily with standard rat pellets. Wood shavings were used as bedding, which was changed every two days.

Preparation of Plant Material

Fresh fruits of *Tetracarpidium conophorum* (Müll. Arg.) were purchased from a local market in Ilishan-Remo, Ogun State, Nigeria. The fruits were taxonomically identified and confirmed by a botanist from the University of Medical Sciences in Ogun State. The fruits were washed with soft, moist cotton wool, and the seeds were carefully removed and baked at 65°C for four hours to facilitate oil extraction.

Extraction of Oil

The oil from the seeds of *Tetracarpidium conophorum* was extracted using the Soxhlet extraction method as recommended by the Association of Official Agricultural Chemists (AOAC, 1996). Approximately 5.0 g of the dried seeds was placed in a pre-weighed thimble and subjected to n-hexane extraction (N-hexane, Sigma-Aldrich, 3050 Spruce St, St. Louis, MO 63103, USA) at a reflux temperature of 40 to 60°C for six hours. After extraction, the thimble was removed, dried at 100°C for thirty minutes to evaporate the solvent, cooled in a desiccator, and weighed to determine the oil yield.

Administration of Test Substances

The experimental rats were divided into five groups, each consisting of six rats. The administration of test substances was as follows:

Group I: Control group received 3.5 mg/kg of normal saline orally.

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Group II: Rats were fed with rat pellets and water only.

Group III: Rats were injected intraperitoneally with 3.5 mg/kg of cadmium acetate (chemical formula: $Cd(CH_3COO)_2$; Cadmium acetate, Fisher Scientific, 300 Industry Drive, Pittsburgh, PA 15275, USA) only. *Group IV*: Rats were injected intraperitoneally with 3.5 mg/kg of cadmium acetate and concurrently received 0.5 ml/kg of *Tetracarpidium conophorum* oil seed orally.

Group V: Rats received 0.5 ml/kg of *Tetracarpidium conophorum* oil seed only.

The treatment lasted for thirty days, after which the animals were sacrificed for further analysis.

Neurobehavioral Study

The beam walking test was conducted to assess the neurobehavioral effects of cadmium exposure and the protective effects of the oil seed . The apparatus consisted of a wooden beam measuring 100 cm in length and 2 cm in width, elevated 40 cm above the ground. The following parameters were measured during the test: *Distance traveled*: The number of lines crossed by the rat.

Foot slips: The number of times the rat's back feet slipped from the beam.

Number of turns: The number of times the rat turned to the other direction.

Termination of Experiment and Sample Collection

At the end of the treatment period, the rats were weighed and sacrificed under chloroform anesthesia. The brain tissues were harvested and washed with normal saline, then fixed in 10% neutral buffered formalin (Formalin, Fisher Scientific, 300 Industry Drive, Pittsburgh, PA 15275, USA) for histological examination.

Histopathological Study

The brain tissues were processed for histological examination. After fixation in 10% neutral buffered formalin, the tissues were subjected to conventional tissue processing, which included dehydration, clearing (Xylene, Thermo Fisher Scientific, 168 3rd Ave, Waltham, MA 02451, USA), and embedding in paraffin wax. Sections of 5 µm thickness were cut using a microtome.

Staining Techniques

Histological sections were stained using Hematoxylin and Eosin (H&E) and Luxol Fast Blue staining techniques to evaluate the histological integrity of the medulla oblongata.

Hematoxylin and Eosin Staining: Sections were deparaffinized in xylene, rehydrated through graded alcohols, and stained with hematoxylin (Hematoxylin, Sigma-Aldrich, 3050 Spruce St, St. Louis, MO 63103, USA) for three to five minutes. After differentiation in acid alcohol, sections were counterstained with eosin (Eosin Y, Sigma-Aldrich, 3050 Spruce St, St. Louis, MO 63103, USA) for one to four minutes, dehydrated, cleared, and mounted with a suitable mounting medium (Sheehan & Hrapchak, 1980).

Luxol Fast Blue Staining: Sections were deparaffinized and hydrated to 95% alcohol, stained in Luxol Fast Blue solution (Luxol Fast Blue, Sigma-Aldrich, 3050 Spruce St, St. Louis, MO 63103, USA) overnight at 60°C, rinsed, and differentiated in lithium carbonate (Lithium Carbonate, Sigma-Aldrich, 3050 Spruce St, St. Louis, MO 63103, USA). They were then counterstained with eosin and mounted (Oh, 2001).

Statistical Analysis

Data were analyzed using GraphPad Prism 6.0.1 software. One-way analysis of variance (ANOVA) was performed, and results were expressed as mean ± standard error of the mean (SEM). A p-value of less than

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0.05 was considered statistically significant. Each experimental treatment was replicated three times, and individual experiments were duplicated to ensure reliability of the results.

Disposal of Used Materials

All biological materials were treated as potentially infectious. Used specimens were autoclaved at 121ºC, 15 psi for fifteen minutes and disposed of by incineration following the completion of the study.

RESULTS AND DISCUSSION

Results

The results of this study are presented in terms of morphological analysis, body weight changes, neurobehavioral assessments, and histological findings. The data demonstrate the effects of cadmium acetate and the neuroprotective role of oil seed from *Tetracarpidium conophorum* on the medulla oblongata of Wistar rats.

Morphological Analysis

Physical Observation

Table I summarizes the mean weights of the animals before and after the administration of cadmium acetate and *Tetracarpidium conophorum* oil seed. The mean weights of the animals before administration were 193.5 g, 208.2 g, 213.2 g, 208.3 g, and 185 g, while the mean weights after administration were 193.8 g, 204.2 g, 208.2 g, 201.5 g, and 182 g.

Table i. Mean Weight of Animals

Group	Mean Weight of Animal Before Administration	Mean Weight of Animals After Administration
1	193.5g	193.8g
2	206.8g	204.2g
3	213.2g	208.2g
4	208.3g	201.5g
5	181.7g	182g

Initial Animal Body Weight

Figure 4. illustrates the initial body weights of the experimental animals across all groups in statistical bar chart. There was a significant difference in body weight among the groups ($p = 0.0001$; $F = 17.14$). Group 3 (213.2 ± 3.701 g) and Group 4 (208.3 ± 3.593 g) showed an increase in body weight, while Group 1 (193.5 ± 3.510 g) and Group 5 (181.7 ± 1.783 g) exhibited a decrease compared to the control group (206.8 ± 2.535 g).

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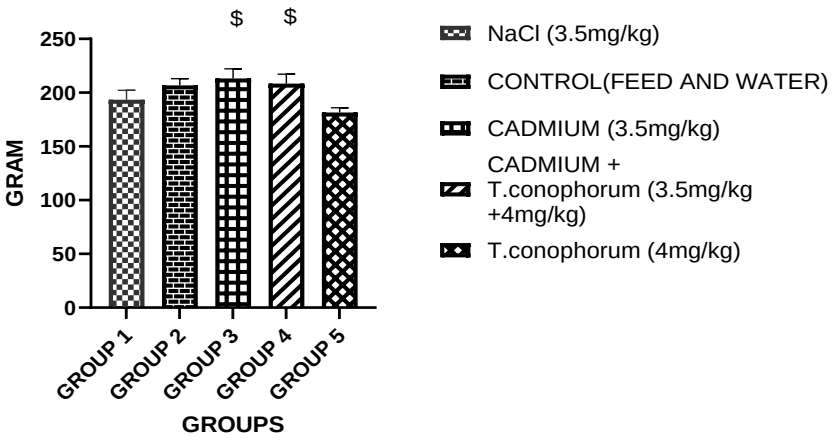

Figure 4. Bar chart showing the initial body weights of animals across all groups. Values are mean SEM of data obtained; p values ($p<0.05$). **$=** indicates an increase in group 3 and group 4 when compared to the control group and the other groups.

Final Animal Body Weight

Figure 5 presents the final body weights of the animals in simple statistical bar charts. A significant difference was observed across all groups ($p = 0.001$; $F = 6.405$). Group 3 (208.2 ± 4.331 g) showed an increase in body weight, while Group 1 (193.8 ± 2.750 g), Group 4 (201.5 ± 3.423 g), and Group 5 (182.0 ± 6.319 g) had decreased body weights compared to the control group (204.2 ± 2.315 g).

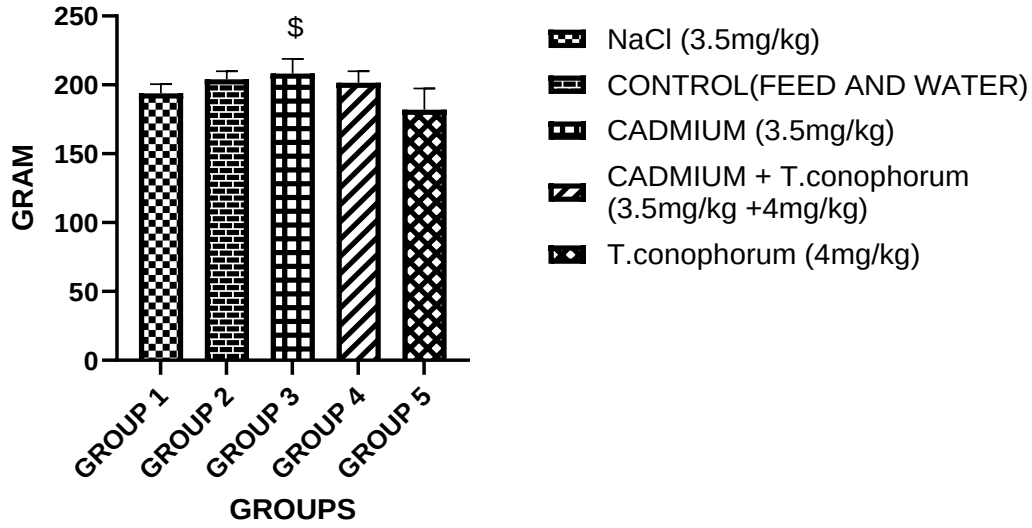

Figure 5 Bar chart showing the final animal body weight across all groups. Values are mean SEM of data obtained; p values ($p<0.05$). **$=** indicates an increase in group 3 when compared to the control group and the other groups.

Neurobehavioral Studies

Number of Turns (First Behavior)

The results of the beam walk test, as shown in Figure 6, indicate significant differences in the number of turns across all groups ($p = 0.0064$; $F = 4.909$). Group 1 (10.00 ± 1.049) and Group 5 (5.800 ± 0.5831) exhibited an increase in the number of turns, while Group 3 (4.600 ± 1.166) and Group 4 (4.200 ± 0.800) showed a decrease compared to the control group (5.400 ± 1.435).

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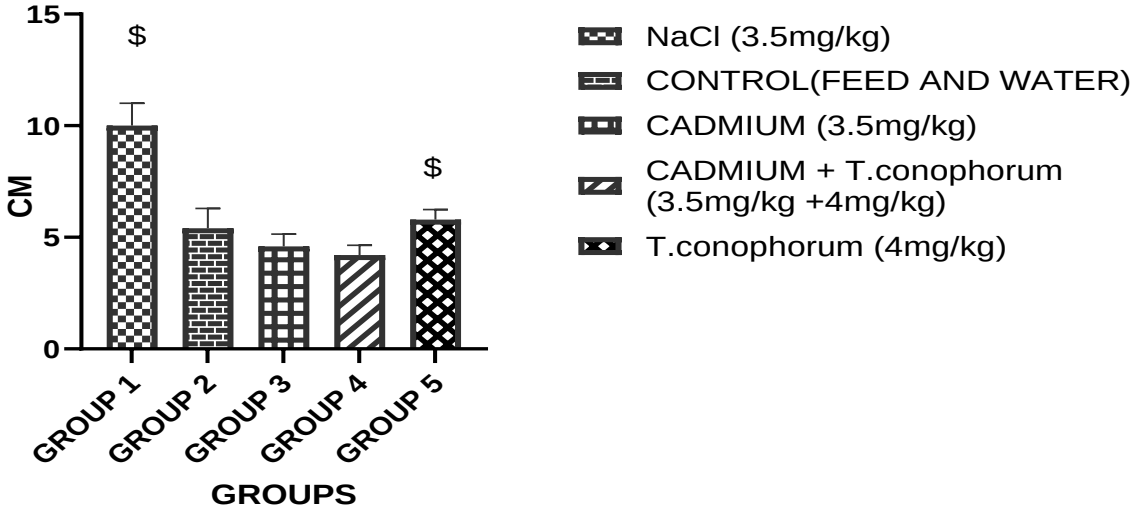

Figure 6: Bar chart showing the Number of turns across all groups. Values are mean SEM of data obtained. Significant difference was observed ($p<0.05$). **$=** indicates an increase in group 1 and group 5 when compared to the control group and the other groups.

Distance Traveled (First Behavior)

Figure 7 shows the distance traveled by the animals during the beam walk test. A significant difference was noted across all groups ($p = 2.922$; $F = 0.0470$). Group 1 (111.2 ± 28.27) traveled a greater distance, while Group 3 (47.00 ± 13.84), Group 4 (51.00 ± 13.08), and Group 5 (29.00 ± 5.788) traveled less compared to the control group (86.00 ± 26.14).

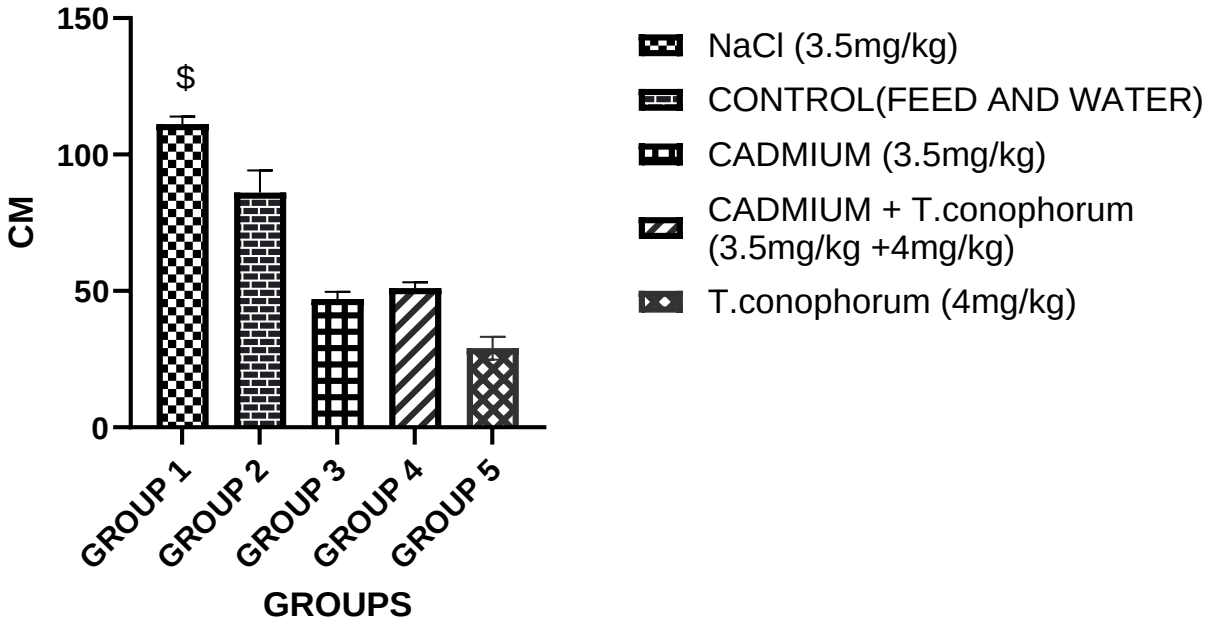

Figure 7: Bar chart showing the Distance travelled across all groups. Values are mean SEM of data obtained. No significant difference was observed ($p>0.05$). **$=** indicates an increase in group 1 when compared to the control group and the other group.

Foot Slip (First Behavior)

The results for foot slips are presented in Figure 8. No significant difference was observed across all groups ($p = 0.7380$; $F = 0.4972$). Group 1 (4.800 ± 1.114), Group 3 (3.800 ± 0.663), and Group 4 (4.200 ± 1.020) had

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observable increases in foot slips, while Group 5 (3.400 ± 0.510) showed no change compared to the control group (3.400 ± 0.748).

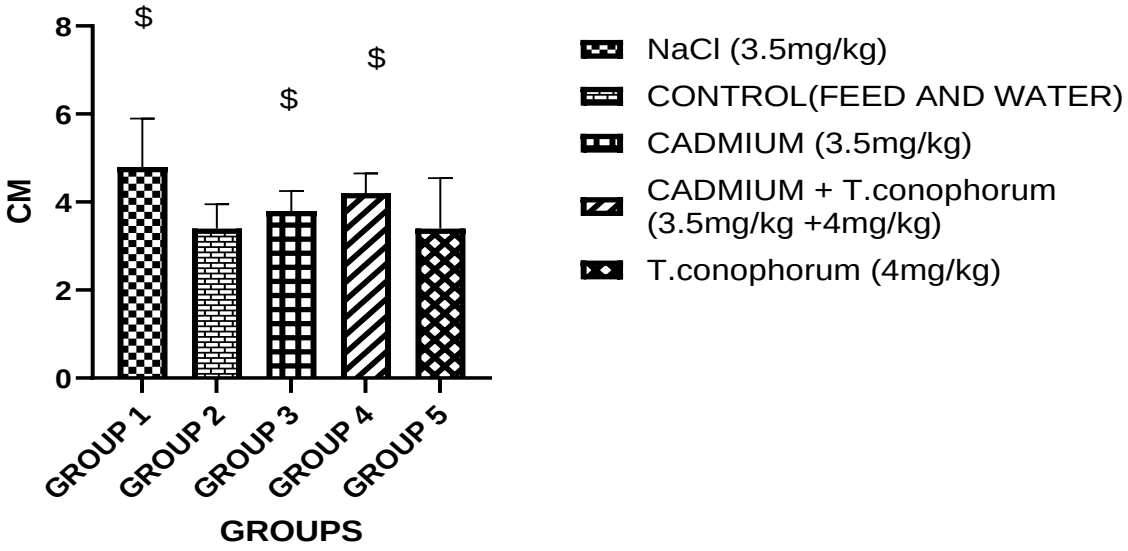

Figure 8: Bar chart showing the Foot slip across all groups. Values are mean SEM of data obtained. No significant difference was observed (p>0.05). **$=** indicates an increase in group 1, group 3 and group 4 when compared to the control group and the other group.

Number of Turns (Second Behavior)

Figure 9 illustrates the results of the second behavior assessment, showing significant differences in the number of turns across all groups (p = 0.0001; F = 11.51). Group 1 (5.800 ± 0.8602), Group 3 (2.400 ± 0.9274), Group 4 (0.800 ± 0.583), and Group 5 (2.800 ± 0.583) exhibited a decrease in the number of turns compared to the control group (7.400 ± 0.09274).

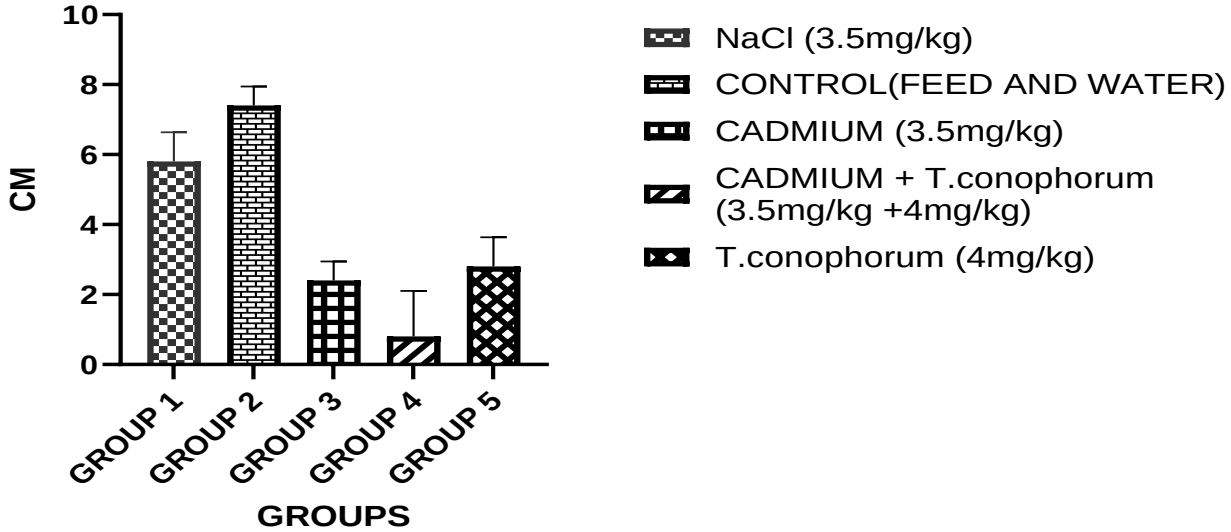

Figure 9: Bar chart showing the Number of turns across all groups. Values are mean SEM of data obtained. Significant difference was observed (p<0.05).

Distance Traveled (Second Behavior)

The results for the second behavior of distance traveled are shown in Figure 10. No significant difference was observed across all groups (p = 0.0770; F = 2.479). Group 1 (76.00 ± 29.89), Group 3 (102.0 ± 45.87), Group 4

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(16.00 ± 10.30), and Group 5 (42.00 ± 28.92) showed a decrease in distance traveled compared to the control group (162.4 ± 50.30).

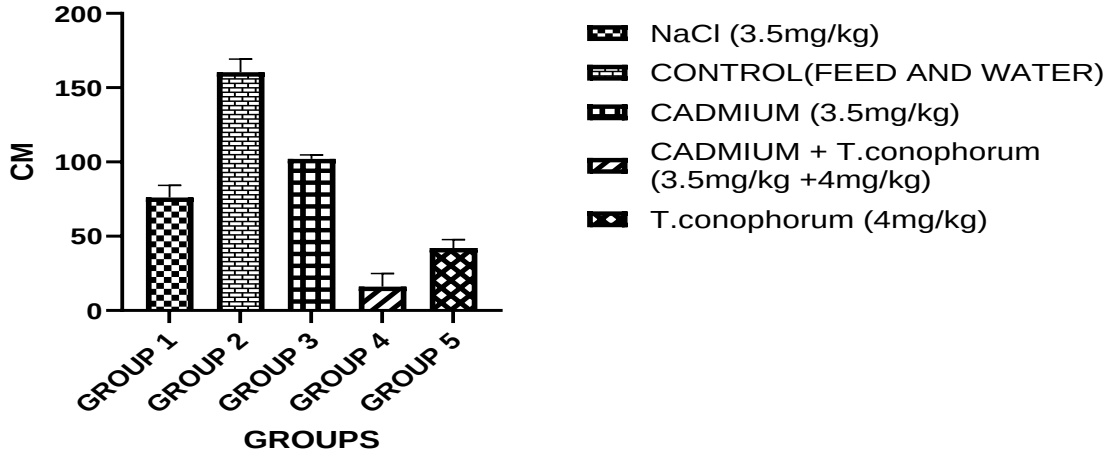

Figure 10: Bar chart showing the Distance travelled across all groups. Values are mean SEM of data obtained. No significant difference was observed (p>0.05).

Foot Slip (Second Behavior)

Figure 11 presents the results for foot slips during the second behavior assessment. No significant difference was observed across all groups (p = 0.7204; F = 0.5224). Group 3 (3.600 ± 1.749) showed an increase in foot slips, while Group 1 (2.800 ± 1.319), Group 4 (1.400 ± 0.600), and Group 5 (2.200 ± 1.114) exhibited decreases compared to the control group (3.000 ± 0.548).

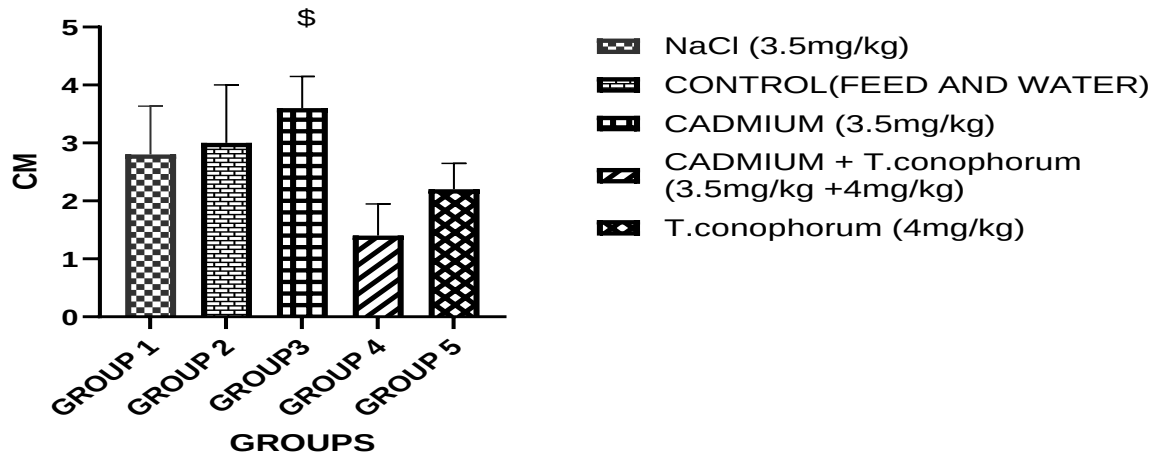

Figure 11: Bar chart showing the Foot slip across all groups. Values are mean SEM of data obtained. No significant difference was observed (p>0.05). **$=** indicates an increase in group 3 when compared to the control group and the other group.

Histological Findings

Histological examination of the medulla cells of the brain was performed using H&E and Luxol Fast Blue staining techniques. The results are presented in Plates 1-10, which illustrate the histological changes observed in the different treatment groups.

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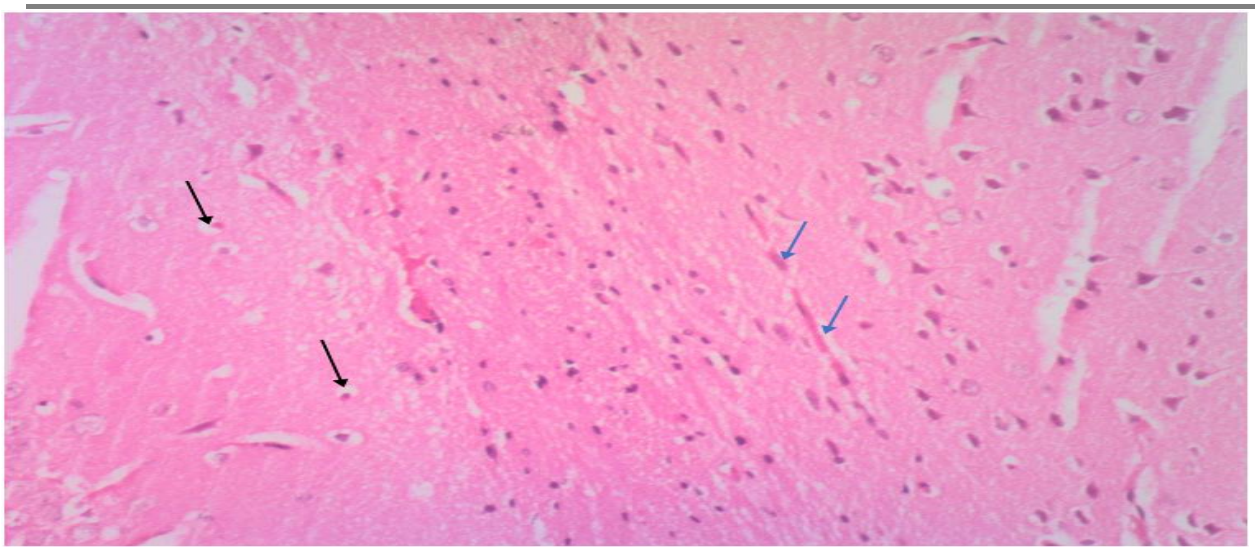

Plate 1: Rat Medulla cells of brain treated with 3.5mg/kg normal saline showing numerous schwann cells (blue), pyramis (black), all appearing normal. Magnification x100 H&E Stain

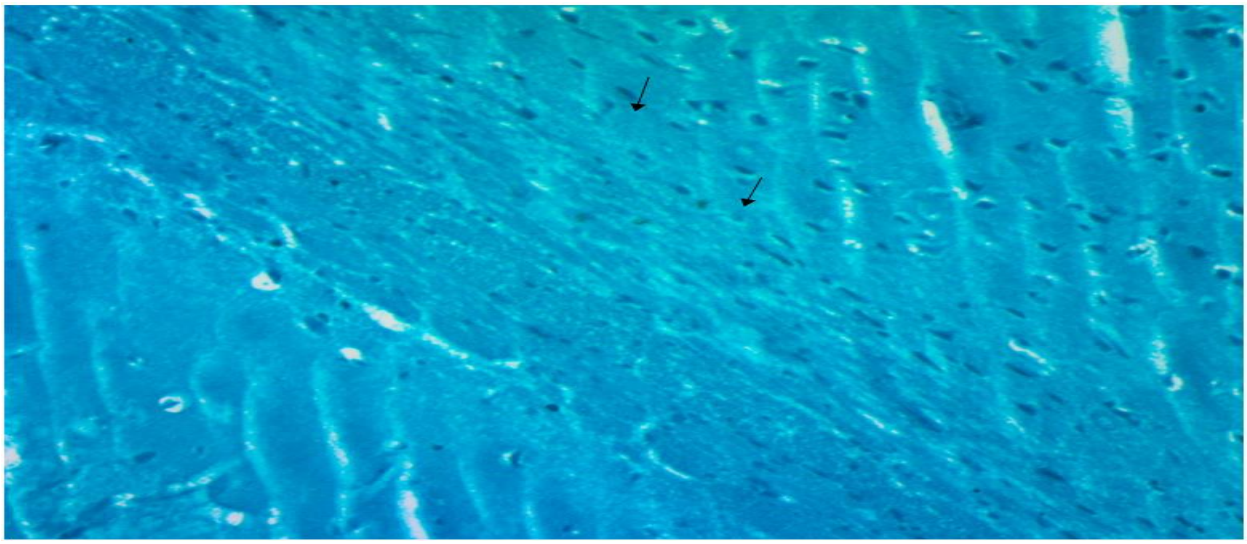

Plate 2: Rat medulla cells of the brain treated with 3.5mg/kg normal saline showing numerous deep blue stained schwaan cells (black). Magnification X100. Luxol Fast Blue Stain

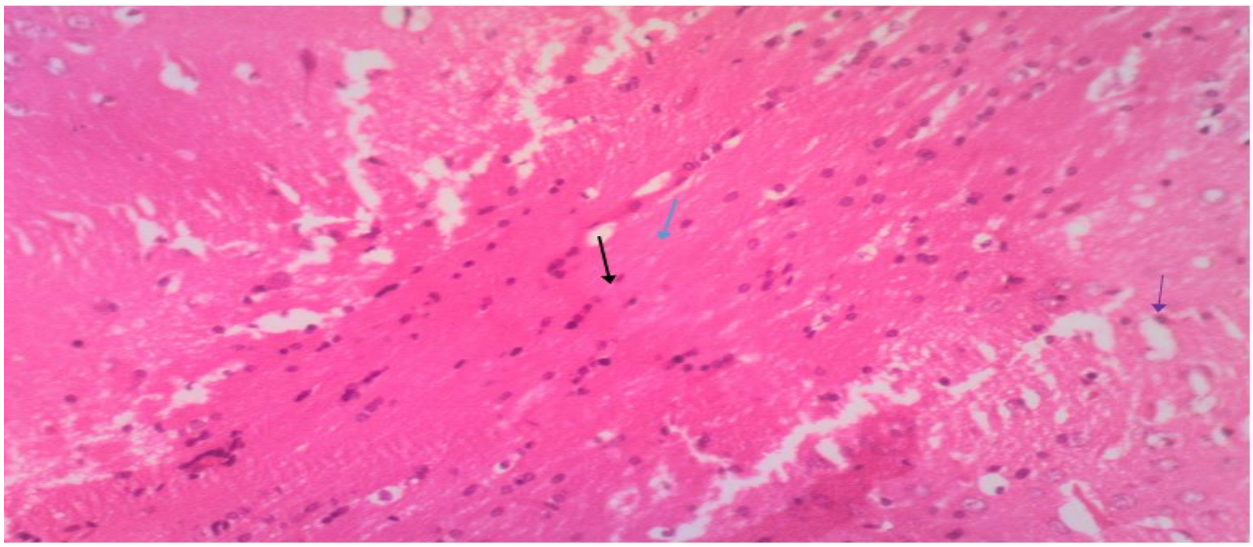

Plate 3: Rat medulla cells of brain fed on rat pellet and water only. Shows normal histoarchitecture of unmyelinated nerves (purple), myelin sheath (blue) and axon (black). Magnification X 100 H&E Stain

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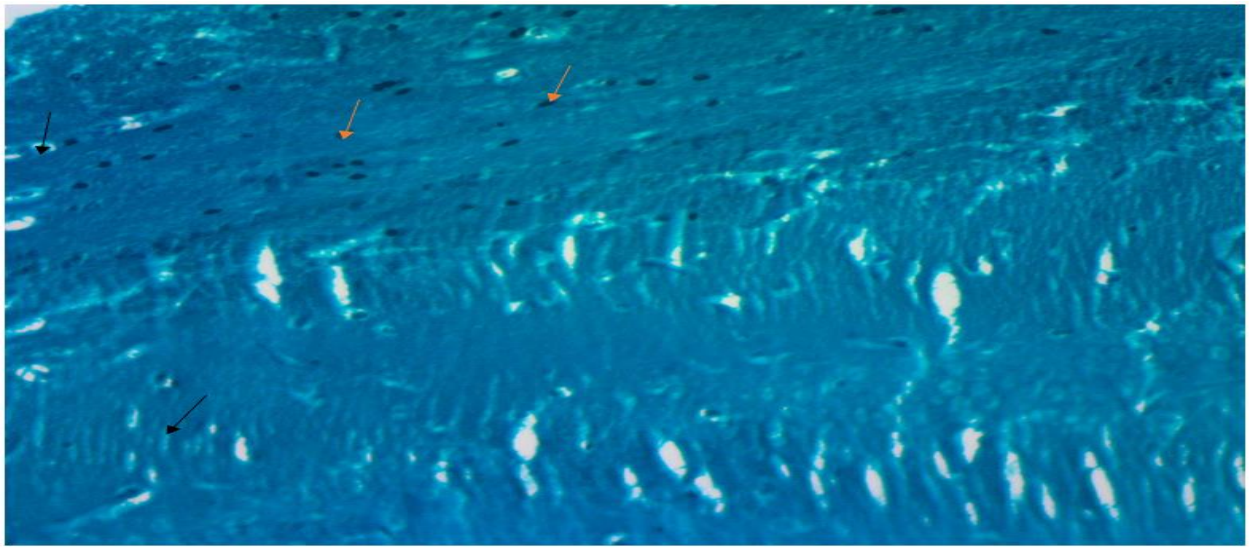

Plate 4: Rat medulla cells of brain fed on rat pellet and water only. Shows dark blue stained hypoglossal nucleus (red) containing numerous motor neurons. Pyramis (black) also seen stained lightly appearing pale blue but deep blue nucleus. Magnification X100 Luxol Fast Blue Stain

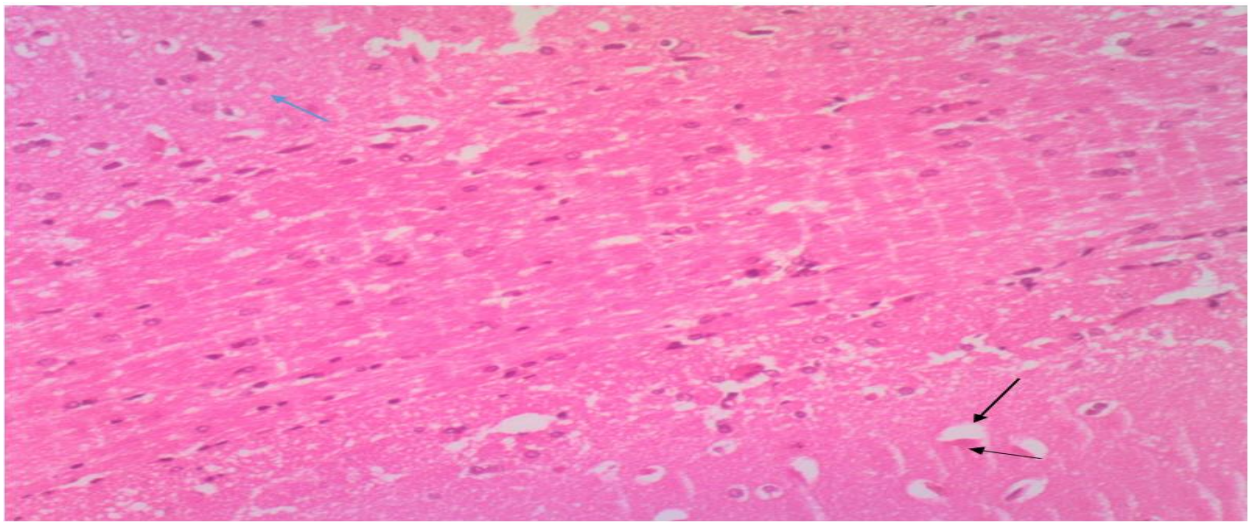

Plate 5: Rat medulla cells of brain treated with 3.5mg/kg cadmium acetate only. Shows distorted unmyelinated nerves (blue), enlarged and vacuolated pyramis (black). Magnification X100 H&E Stain

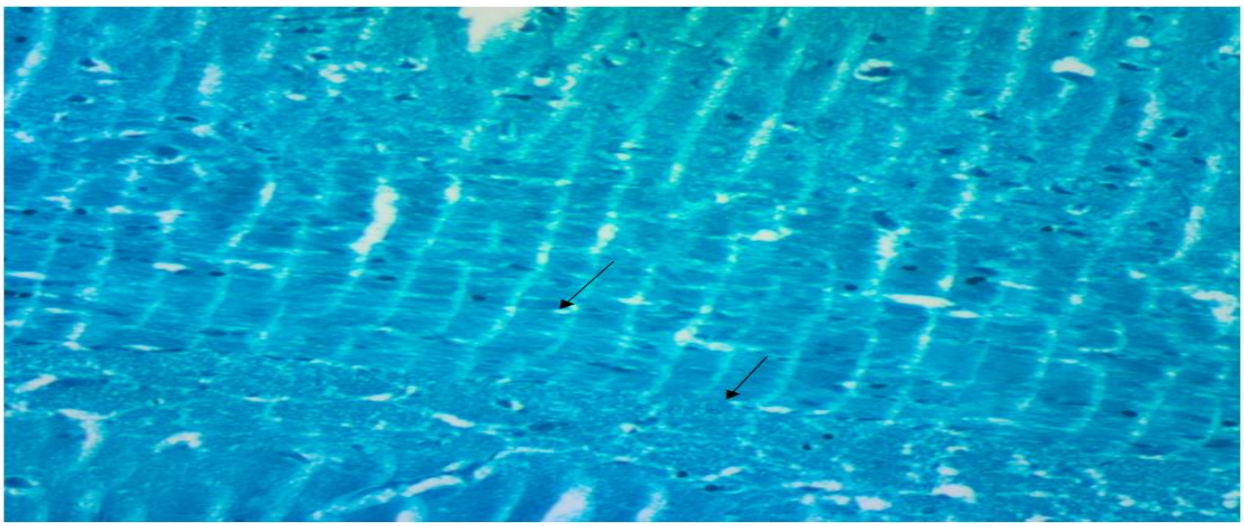

Plate 6: Rat medulla cells of brain treated with 3.5mg/kg cadmium acetate only. Shows disoriented myelin sheath that appears as light blue medullary rays (black). Magnification X100 Luxol fast blue Stain

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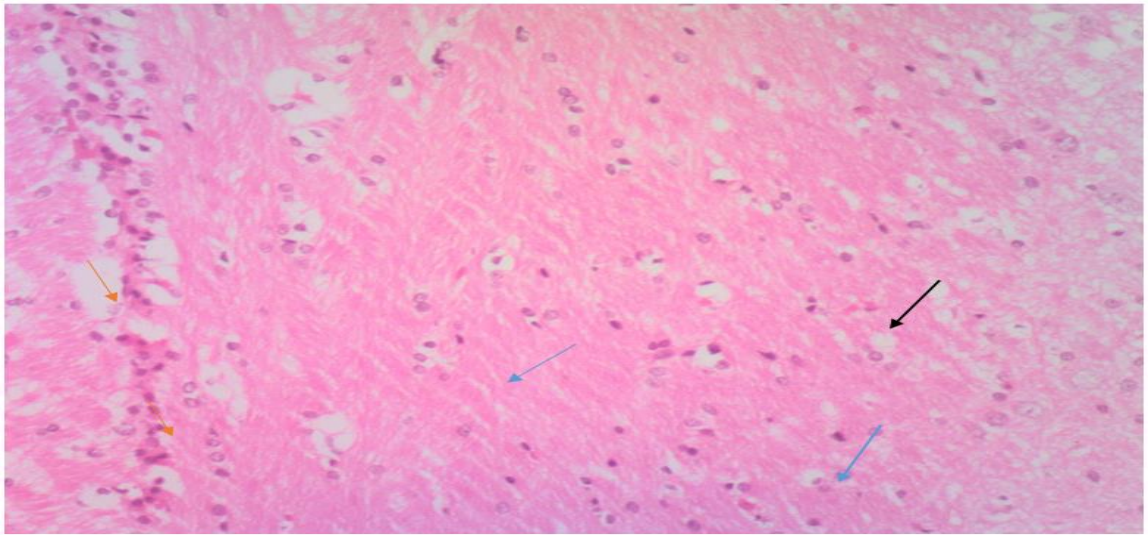

Plate 7: Rat medulla cells treated with 3.5mg/kg cadmium acetate and 0.5ml/kg oil seed of *T.conophorum* showing numerous schwaan cells (black), increased pyramis (blue) and numerous myelin sheath (red). Magnification X100 H&E Stain

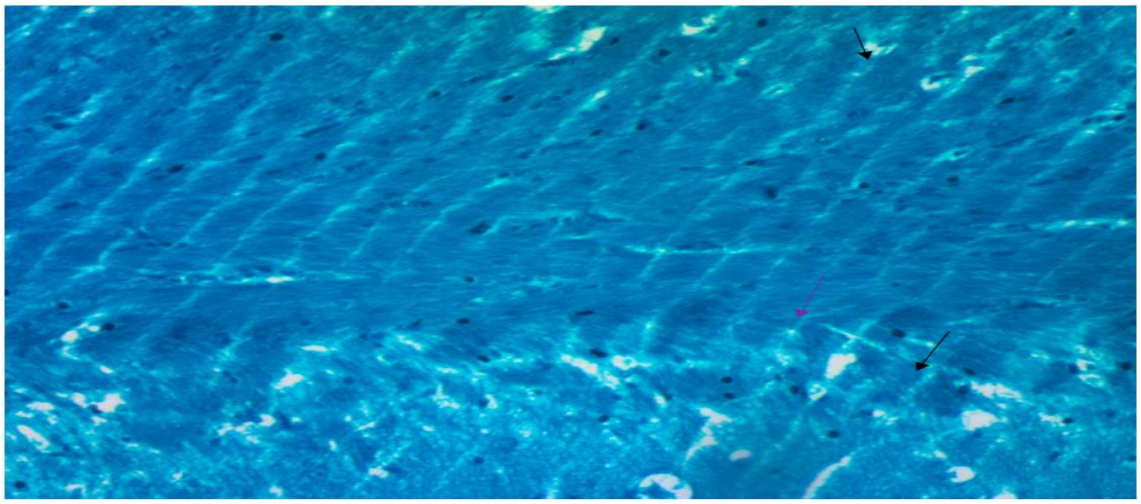

Plate 8: Rat medulla cells treated with 3.5mg/kg cadmium acetate and 0.5ml/kg oil seed of *T.conophorum* showing numerous schwaan cells (red), increased pyramis (black) and numerous myelin sheath (purple). Magnification X100 Luxol fast blue Stain

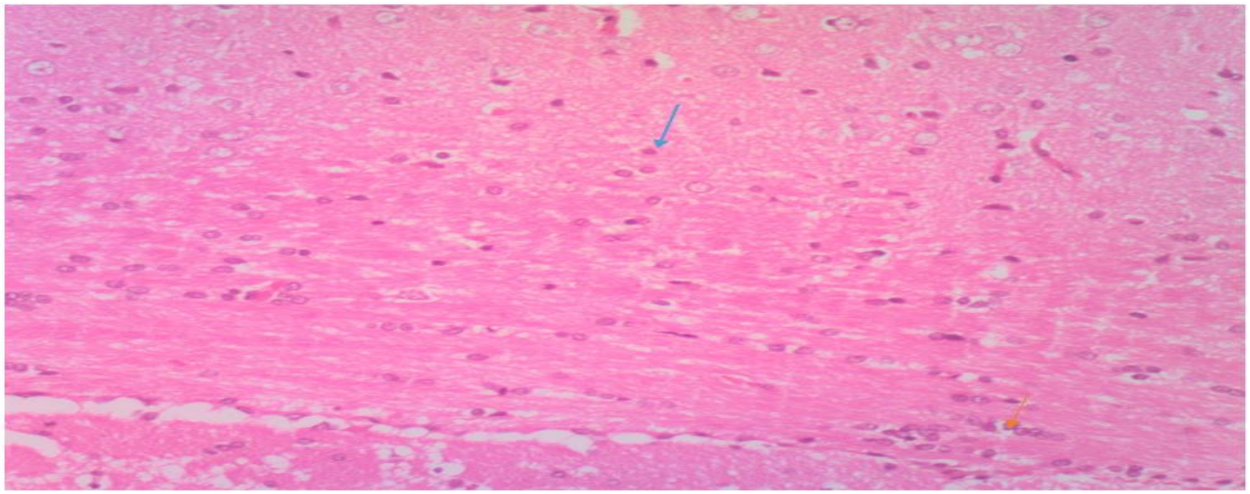

Plate 9: Rat medulla cells treated with 0.5ml/kg oil seed of *T. conophorum* only. Shows normal histology of medulla cells. There is an increase in neuronal cell body and other features of the medulla such as schwaan

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cells (black), pyramis (blue) and hypoglassal nuclei (red). Magnification X100 H&E Stain

Plate 10: Rat medulla cells treated with 0.5ml/kg oil seed of *T.conophorum* only shows distinct pyramis (black) using special stain. Magnification X100 Luxol fast blue Stain

DISCUSSION

Summary of findings

This study shows that exposure to cadmium acetate causes damage to the medulla oblongata seen under the microscope and leads to noticeable behavioral problems on the beam walk test. When *T. conophorum* oil seed is given orally at the same time, it partly protects the tissue structure and helps reduce behavioral issues. These results support the idea that natural products rich in antioxidants can help lessen neurotoxicity caused by metals.

Comparison with previous studies

Our results align with reports that cadmium causes neuronal vacuolation, myelin damage, and axonal abnormalities (Afifi & Embaby, 2016; Wang & Du, 2013). Cadmium's neurotoxicity is mainly linked to oxidative stress, which involves increased production of reactive oxygen species, lipid damage, and a decrease in antioxidants (Bauer & Bauer, 1999; Wang & Du, 2013).

The protective effects of *T. conophorum* oil seed observed here match previous studies showing that extracts from this plant have anti-peroxidative and antioxidant effects in animal tissues (Akomolafe et al., 2015; Amaeze et al., n.d.). Tokunbo et al. (2023) described neuroprotective actions of *T. conophorum* in hippocampal models, involving increased heat shock proteins and preserved neuronal markers; our findings in the medulla support those neuroprotective trends.

Position within the broader context of natural neuroprotectants

Many dietary plants and seed oils, like curcumin, tea polyphenols, and omega-rich oils, show neuroprotective effects against heavy metals and toxins through antioxidant, anti-inflammatory, and metal-binding activities. *T. conophorum* is part of this group, and its common use as food in West Africa suggests it could be promising for translation into health strategies. Still, its strength, how well it's absorbed, and safety compared to other agents need direct comparison studies.

Mechanistic considerations: need for biochemical biomarkers

While histology and behavioral tests indicate protection, confirming how *T. conophorum* works requires

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biochemical and molecular data. Including markers of oxidative stress is especially important. Future research should at least measure:

- Lipid peroxidation: malondialdehyde (MDA)

- Antioxidant enzymes: superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx)

- Non-enzymatic antioxidants: reduced glutathione (GSH)

- Inflammatory cytokines: TNF-α, IL-1β

- Apoptosis markers: caspase 3 activity, TUNEL staining

- Tissue cadmium levels (e.g., by ICP-MS) to relate metal load with injury and recovery.

These tests will help determine if *T. conophorum* mainly acts as a scavenger of ROS, promotes natural antioxidants, chelates metals, reduces inflammation, or works through other pathways.

Limitations

- The study used only one cadmium dose and one oil dose; dose–response effects were not explored.

- Histological assessment was qualitative; quantitative measurements and blinded scoring would improve reliability.

- No biochemical or molecular markers were measured, so mechanistic explanations are still speculative.

- Only female rats were used; potential sex differences were not examined.

Recommendations for future research

Future studies should include quantitative histomorphometry, blinded scoring, varied doses, and tests of oxidative stress and inflammation markers. Isolating and characterizing active compounds from *T. conophorum* oil, along with pharmacokinetic, toxicological, and metal load assessments, is necessary. If results are promising, clinical studies, like dietary supplement trials in populations at risk of cadmium exposure, could be pursued.

CONCLUSION

Tetracarpidium conophorum oil seed helped reduce cadmium acetate-induced tissue damage and behavioral problems in the medulla oblongata of Wistar rats, indicating its potential as a natural neuroprotectant. Because of its nutritional use and antioxidant properties, *T. conophorum* deserves further study as a supplement or therapy to lessen heavy metal neurotoxicity. Moving to human applications will need thorough preclinical research, including mechanistic and safety studies (such as measuring oxidative stress and tissue cadmium), dose optimization, and clinical trials to prove its safety and effectiveness.

Data Availability

All data from this study are included in this publication and its supplementary files. Raw data can be requested from the corresponding author.

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Ethics declarations

Approval for animal use was obtained from the Babcock University Health Research Ethics Committee (Approval number 428/23).

Conflict of interest

The authors have no conflicts of interest.

Authorship

All authors contributed to the study design, experiments, data analysis, and writing.

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