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Effects of *Tetrapleura tetraptera* on aluminium-induced testicular damages in adult Wistar rats

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Abstract

Introduction: Aluminium (Al), the third most abundant metallic element in the Earth's crust, is widely distributed in the environment and present in water, soil, plants, and animal tissues. Excessive exposure is linked to reproductive toxicity. **Aim:** This study evaluated the protective effects of phenolic extracts of *Tetrapleura tetraptera* (Aidan fruit) on aluminium-induced testicular toxicity in adult male Wistar rats. **Methodology:** Thirty-five rats were randomly assigned into five groups (n = 7): Group 1 received distilled water (control), Group 2 was administered $AlCl_3$ (100 mg/kg), Group 3 received $AlCl_3$ + low-dose extract (250 mg/kg), Group 4 received $AlCl_3$ + high-dose extract (500 mg/kg), and Group 5 received $AlCl_3$ + donepezil (10 mg/kg). Toxicity were induceced for 28 days and treatments lasted 14 days, after which animals were sacrificed following IACUC guidelines. The right testes were homogenized for biochemical and hormonal assays, while the left testes were fixed in formalin for histological studies. **Results:** $AlCl_3$ exposure did not significantly alter body weight but markedly reduced hormonal levels (AMH, LH, FSH, testosterone) and impaired sperm quality and testicular architecture ($p \leq 0.05$). It also elevated oxidative stress markers, including malondialdehyde (MDA) and Nitric Oxide (NO). Treatment with *T. tetraptera*, especially at 500 mg/kg, significantly restored hormonal profiles, improved sperm count and motility, enhanced antioxidant enzymes (SOD, AChE), and reversed histological damage to the seminiferous tubules ($p \leq 0.05$). **Conclusion:** These findings suggest that phenolic extracts of *T. tetraptera* confer dose-dependent protection against aluminium-induced reproductive toxicity through antioxidant mechanisms, hormonal regulation, and testicular tissue restoration.

Keywords: *Tetrapleura tetraptera, Aluminum chloride, Wistar rats, Antioxidants, Spermatogenesis, Reproductive toxicity*

1. Introduction

Aluminium (Al) is the third most abundant metallic element in the Earth's crust, following oxygen and silicon

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(Joseph *et al.*, 2021). It is widely distributed in the environment and occurs in water, soil, plants, and animal tissues, including humans and laboratory animals such as Wistar rats. Despite its prevalence, aluminium has no known biological role and is increasingly recognized for its toxic effects, particularly on the central nervous and reproductive systems (Joseph *et al.*, 2021). In humans, aluminium accumulation has been linked to neurodegeneration, reproductive toxicity, and foetal teratogenicity, especially in regions with high environmental exposure (Liaquat *et al.*, 2021).

In developing countries such as Nigeria, daily aluminium exposure is a public health concern due to its use in cookware, food packaging, and drinking water contamination (Fellows *et al.*, 2024). In response to growing evidence of toxicity, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) revised the Provisional Tolerable Weekly Intake (PTWI) from 7.0 mg/kg body weight in 1989 to 1.0 mg/kg in 2007, citing reproductive and neurodevelopmental risks (Steffensen *et al.*, 2019).

Aluminium toxicity is mediated largely through oxidative stress, resulting in cellular damage and functional impairment (López-Botella *et al.*, 2021). Within the reproductive system, it disrupts the hypothalamic–pituitary–gonadal axis, impairs testosterone synthesis and spermatogenesis, and reduces sperm motility, morphology, and viability (Cheraghi *et al.*, 2017; López-Botella *et al.*, 2021). Animal studies, particularly in Wistar rats, have been instrumental in elucidating these mechanisms. Wistar rats are widely used because of their genetic uniformity, well-characterized reproductive physiology, and high sensitivity to heavy metals (Patel *et al.*, 2024). Aluminium exposure in both humans and rats leads to testicular degeneration, altered Leydig and Sertoli cell function, and reduced reproductive hormone levels (Adelakun *et al.*, 2021).

Although synthetic chelating agents exist, they often cause adverse effects and are unsuitable for chronic reproductive toxicity (Adelakun *et al.*, 2021). As a result, interest has shifted toward phytotherapeutics. *Tetrapleura tetraptera* (Aidan fruit), a West African medicinal plant, has shown promise due to its rich phytochemical composition, including flavonoids, tannins, and alkaloids (Mensah *et al.*, 2024; Adusei *et al.*, 2019). These compounds possess antioxidant, anti-inflammatory, and metal-chelating properties, making the fruit a potential candidate for mitigating aluminium-induced toxicity (Adelakun *et al.*, 2021).

However, limited studies have specifically examined the effects of *T. tetraptera* phenolic extracts on aluminium-induced testicular damage. Preliminary evidence suggests that aqueous extracts do not impair copulatory behaviour or testicular morphology, but their protective role under toxicological stress remains unclear (Adelakun *et al.*, 2021). Therefore, this study investigates the protective effects of *T. tetraptera* phenolic extracts on aluminium-induced testicular toxicity in adult male Wistar rats.

2. Materials and methods

2.1. Materials

2.1.1. Experimental chemicals

Aluminium Chloride ($AlCl_3$): 20 g $AlCl_3$ produced by Span Chemie company, India and was gotten from Dennis and Dennis Store Ilorin, Kwara State and authenticated in the Department of Chemistry, Faculty of Natural and Applied Sciences, Al-Hikmah University, Ilorin.

Donepezil: 10 gm Donepezil: was bought from One-Step Pharmacy Ilorin, Kwara State.

Experimental plant: Aidan fruit (*Tetrapleura tetraptera*): 1000 g of Aidan fruit was obtained from Mandate daily market Ilorin, Kwara State where it was powdered and the seed were removed and was taken to the Industrial Chemistry, Faculty of Physical Science, University of Ilorin, Kwara State where it was extracted.

Other materials: Syringes, gloves, sample bottles, distilled water and dissecting kits were of analytical value.

Experimental animals: Thirty five (35) apparently healthy Wistar rats were purchased from the Department of Human Anatomy, Faculty of Basic Medical Science, College of Health Science, Al-Hikmah University, Ilorin, Kwara State.To replicate their natural habitat, the Wistar rats were housed at ambient temperature with a typical light-dark cycle (12:12). To suit their nutritional needs, the Wistar rats were given purified water and grower pellet diet during the trial. Two weeks prior to the start of the experiment, the Wistar rats were acclimated.

2.2. Methods

2.2.1. Ethical approval

The ethical approval for the study was sought from the Health Science Ethical Review Committee for Animal Use and Care of the College of Health Sciences, Al-Hikmah University, Ilorin, Kwara State, Nigeria with the Approval Number:HUI-ERC/ASN/2018/2025/3214.

2.2.2. Experimental protocol

Group A which was the Negative Control group consisting of seven (7) Wistar rats, where administered distilled water for 28 days. Group B which was the Toxic group were given $AlCl_3$ for 28 days. Group C which was the Treated group c were administered slow dose $AlCl_3$ for 28 days and PT-T for 14 days. Group D which was the treated group consisting of seven (7) Wistar rats, were administered medium dose of $AlCl_3$ for 28 days and PT-T for 14 days. Group E which was the treated group consisting of seven (7) Wistar rats, were administered high dose of $AlCl_3$ for 28 days and PT-T for 14 days. Group F which was the Positive Control group consisting of seven (7) Wistar rats, were administered $AlCl_3$ for 28 days and PT-T for 14 days.

2.2.3. Histological examination

At the conclusion of the study (day 14 and day 28), the rats were humanely euthanized using cervical dislocation. The testes were quickly dissected and fixed in 10% formalin for 48 hours, after fixation, the testes tissues were embedded in paraffin and sectioned into thin slices (5 µm). These sections were then stained with Haematoxylin and Eosin (H&E) to examine any histological changes, including signs of testicular damage such as seminiferous tubule degeneration, germ cell loss, and the interstitial tissue alterations. Microscopic analysis was performed to identify and assess structural changes in the testes.

2.3. Biochemical assays

Acetylcholinesterase (AChE): Acetylcholinesterase activity was markedly reduced in the testes of rats exposed to aluminium chloride, indicating impaired testicular innervation and disrupted spermatogenic regulation. AChE is essential for the cholinergic signalling that supports sperm maturation and motility; its suppression suggests neurochemical dysfunction within the testes.

Malondialdehyde (MDA): Malondialdehyde levels were significantly elevated in the testes of the denoting intense lipid peroxidation and oxidative damage to the seminiferous epithelium. Elevated MDA levels are indicative of disrupted membrane integrity in germ cells and Leydig cell degeneration, ultimately compromising testosterone synthesis and sperm development.

Superoxide Dismutase (SOD): Superoxide dismutase a critical antioxidant enzyme in testicular defense, suggesting an overwhelmed antioxidant system within the testes. A weakened SOD response implies the testes were unable to neutralize superoxide radicals, increasing susceptibility to oxidative injury and cell death. However, SOD activity was elevated in the treatment groups, especially with high-dose Aidan fruit, highlighting recovered antioxidative defense and improved testicular resilience.

Nitric Oxide (NO): Nitric oxide levels were increased in the testes following toxic exposure, indicating an inflammatory state and potential vascular dysfunction within the testicular microenvironment. Excess NO may interact with superoxide radicals to form harmful peroxynitrite, contributing to germ cell apoptosis and testicular tissue injury.

2.4. Hormonal assay

2.4.1. Testosterone levels

Testosterone levels were measured using a commercially available rat-specific ELISA kit, following the manufacturer's protocol. Testosterone is essential for spermatogenesis, libido, and maintenance of secondary sexual characteristics. A reduction in testosterone levels indicates impaired testicular endocrine function.

Luteinizing Hormone (LH): Luteinizing hormone is typically measured in serum using immunoassay techniques such as ELISA (enzyme-linked immunosorbent assay) or chemiluminescent immunoassays.

LH plays a pivotal role in testicular endocrine function by stimulating Leydig cells in the interstitial compartment of the testes to produce testosterone, which is crucial for spermatogenesis. A reduction in LH levels disrupts testosterone synthesis, leading to hypogonadism, reduced libido, infertility, and testicular atrophy.

Follicle-Stimulating Hormone (FSH): Follicle-stimulating hormone is measured using radioimmunoassay or automated ELISA methods and serves as a key marker of Sertoli cell function in the testes. FSH is essential for the initiation and maintenance of spermatogenesis by stimulating Sertoli cells to produce androgen-binding protein (ABP) and support the maturation of germ cells. When FSH levels fall below normal, the spermatogenic process is compromised, leading to oligospermia or azoospermia (low or absent sperm count), testicular dysfunction, and possible infertility.

Anti-Müllerian Hormone (AMH): Anti-Müllerian hormone is measured using high-sensitivity ELISA kits, especially in reproductive and developmental biology studies. In males, AMH is secreted primarily by immature Sertoli cells during early life and continues at a lower level into adulthood, serving as a marker of Sertoli cell presence and function. Low AMH levels in postnatal or adult testicular tissue are indicative of Sertoli cell dysfunction, depletion of the germinal epithelium, or testicular dysgenesis.

2.5. Statistical analysis

Data were expressed as mean ± Standard Error of the Mean (SEM). Statistical comparisons between groups were performed using one-way analysis of variance (ANOVA) followed by Tukey's post-hoc test to determine significant differences. A p-value of < 0.05 was considered statically significant. All statistically analysis was performed using GraphPad Prism 8 software.

3. Results

3.1. Physical observation

Physical observations were carried out daily throughout the experiment to monitor the health status, behavioral changes, and overall physical response of the rats in each group.

Group 1 (Control–distilled water): Rats in this group remained physically stable and active throughout the 28-Day period. They showed no signs of abnormal behavior, bleeding, or death. Feeding and grooming remained normal.

Group 2 (Toxic–aluminum chloride): From day 2, rats in this group began showing signs of distress such as excessive calmness and hiccups. By day 4, nasal bleeding was observed. Mortality began occurring by Day 16. Aggressiveness, reduced feed intake, and signs of meningitis were also noted. The group was sacrificed on Day 28.

Group 3 (Aluminum chloride + low dose PPT): Initial signs of distress (mucous secretion, spinning, walking sideways) were observed during the aluminum chloride phase. After the introduction of Aidan fruit extract on Day 29, gradual behavioral improvement and signs of healing were recorded. However, some rats died on Day 32, 33, and 35. By Day 41, the remaining rats were visibly active with reduced signs of complication.

Group 4 (Aluminum chloride + high dose PPT): This group exhibited significant distress, including nose bleeding and feed refusal, starting from the aluminum chloride phase. One death occurred early (Day 25), followed by progressive improvement after high-dose Aidan extract administration. However, one death was also recorded on Day 27. Signs of recovery (reduced swelling, improved feeding, and activity) became prominent by Day 30.

Group 5 (Aluminum chloride + donepezil): Early signs included mucous secretion, limb paralysis, and aggressiveness. Mortality occurred on Day 15. After donepezil administration began, improvement in motor function and healing of visible injuries (e.g., swollen limb, cleft palate) was recorded. By Day 30, healing was evident, and rats remained active up to Day 41.

3.2. Body weight of the animals

Although, there was no significant difference in the body weight across experimental groups. The Toxic group

showed increased weight gain when compared with the initials. Moreso, all treated groups, especially the Donepezil and High Dose Aidan groups demonstrated improved weight recovery when compared with the Toxic group. This suggests a potential protective or restorative effect of the treatments against Aluminium-induced systemic toxicity as shown in Figure 1.

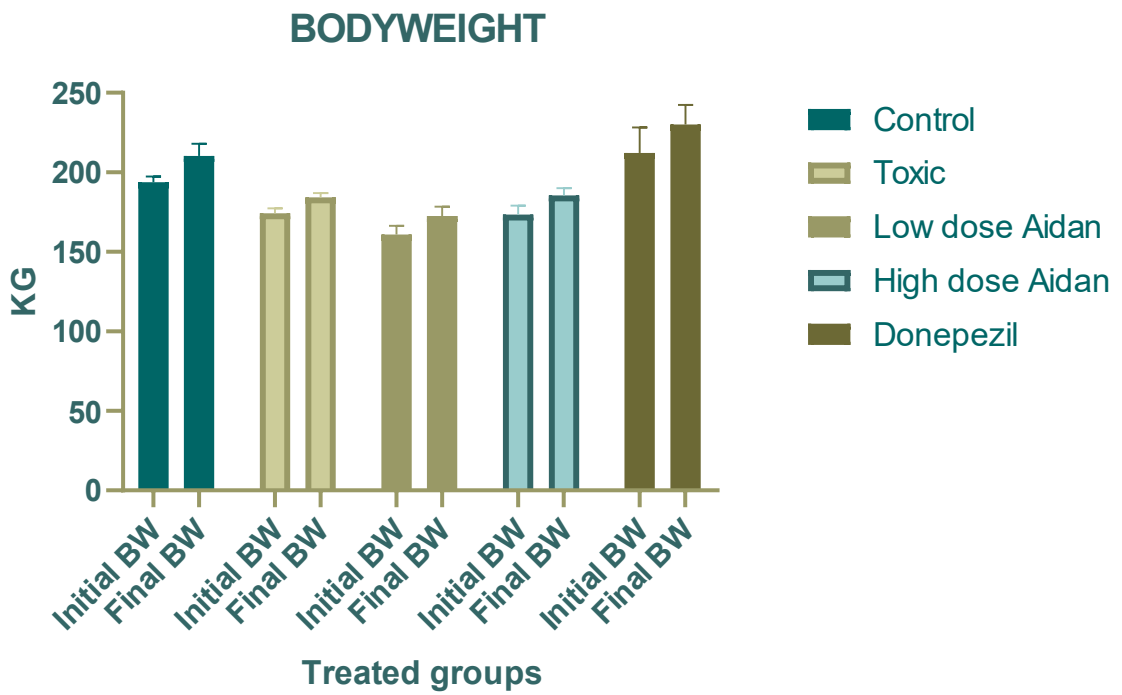

Figure 1: Chart showing the initial and final body weight across treated group

3.3. Hormonal assays

The toxic group showed a nonsignificant reduction in FSH compared with the control. Both low- and high-

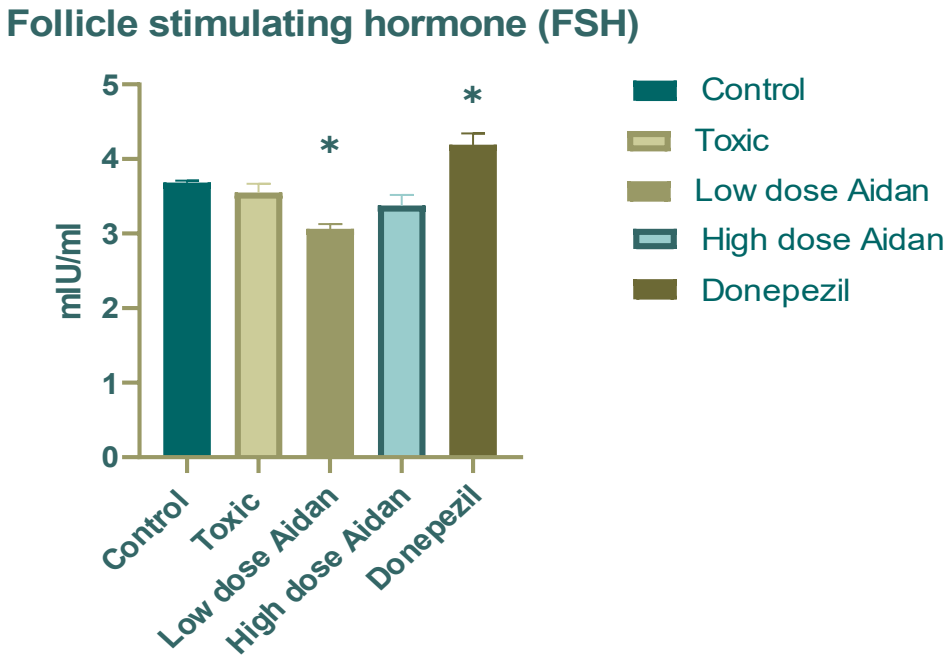

Figure 2: Bar chart showing mean ± SEM of FSH level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

dose groups also showed nonsignificant decreases relative to the toxic group, whereas Donepezil produced a significant increase ($p < 0.05$). Luteinizing hormone levels were slightly reduced in the toxic group compared with control, but the difference was not significant. Low- and high-dose groups exhibited nonsignificant increases, while Donepezil also increased LH, though not significantly. Anti-Müllerian hormone was nonsignificantly elevated in the toxic and low-dose groups compared with control; however, both the high-dose and Donepezil groups showed significant increases relative to the toxic group ($p < 0.05$). Testosterone was significantly reduced in the toxic group compared with control ($p < 0.05$). The low-dose group showed a significant increase relative to the toxic group, while the high-dose group demonstrated a further significant reduction. Donepezil treatment produced a nonsignificant decrease compared with the toxic group.as shown in the Figures 2, 3, 4 and 5.

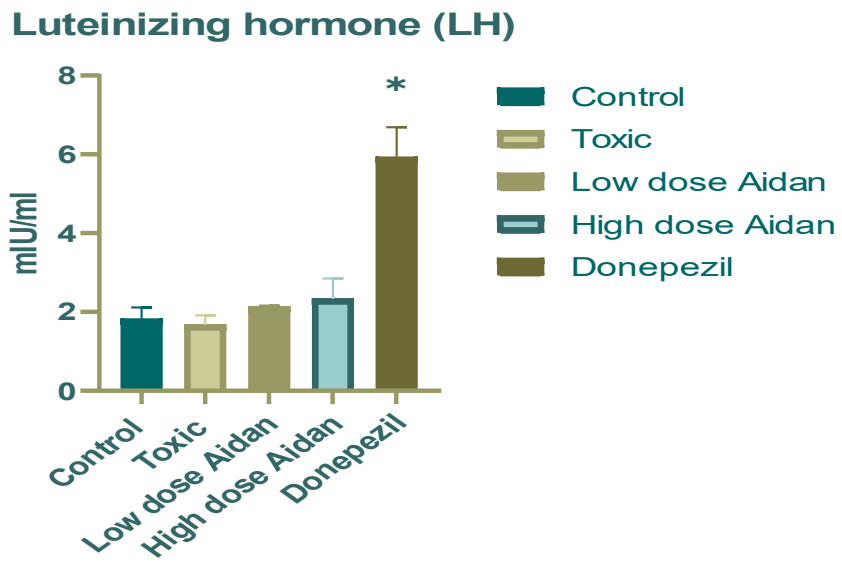

Figure 3: Bar chart showing mean ± SEM of LH level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

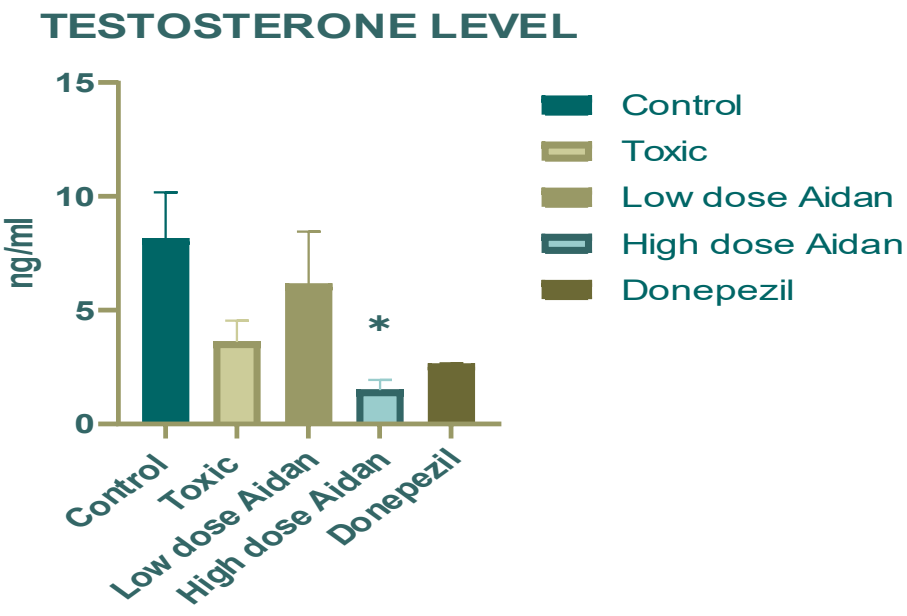

Figure 4: Bar chart showing mean ± SEM of Testosterone level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

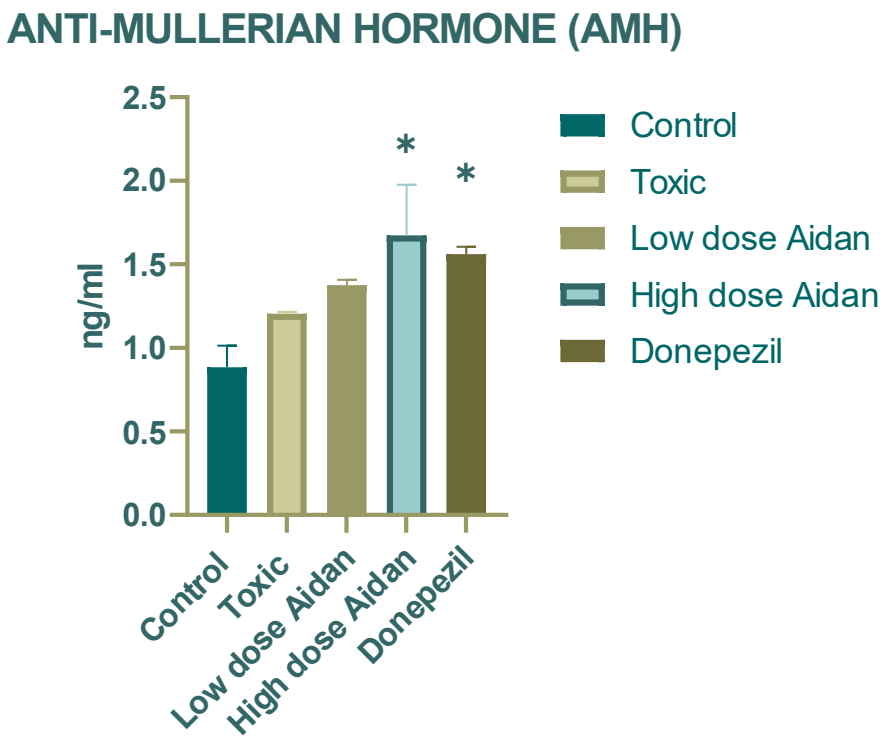

Figure 5: Bar chart showing mean ± SEM of AMH level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

3.4. Oxidative stress markers

The toxic group exhibited a significant increase in MDA compared with the control ($p < 0.05$), indicating aluminum chloride–induced oxidative stress. Treatment with low dose, high dose, and Donepezil

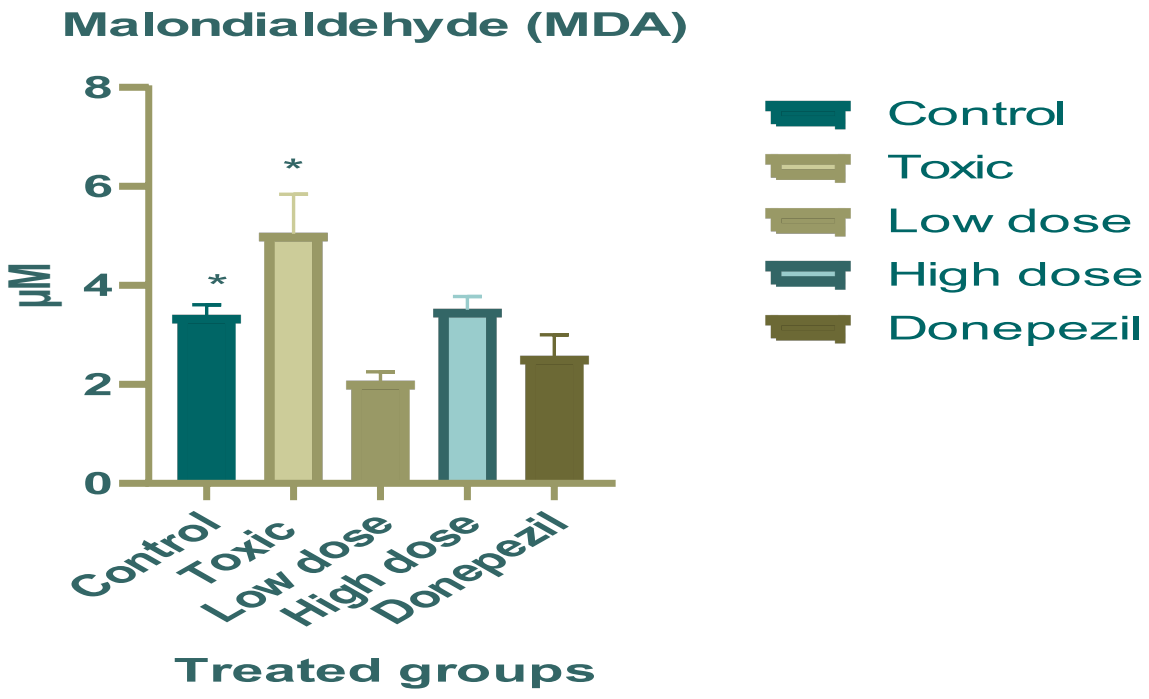

Figure 6: Bar chart showing mean ± SEM of MDA level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

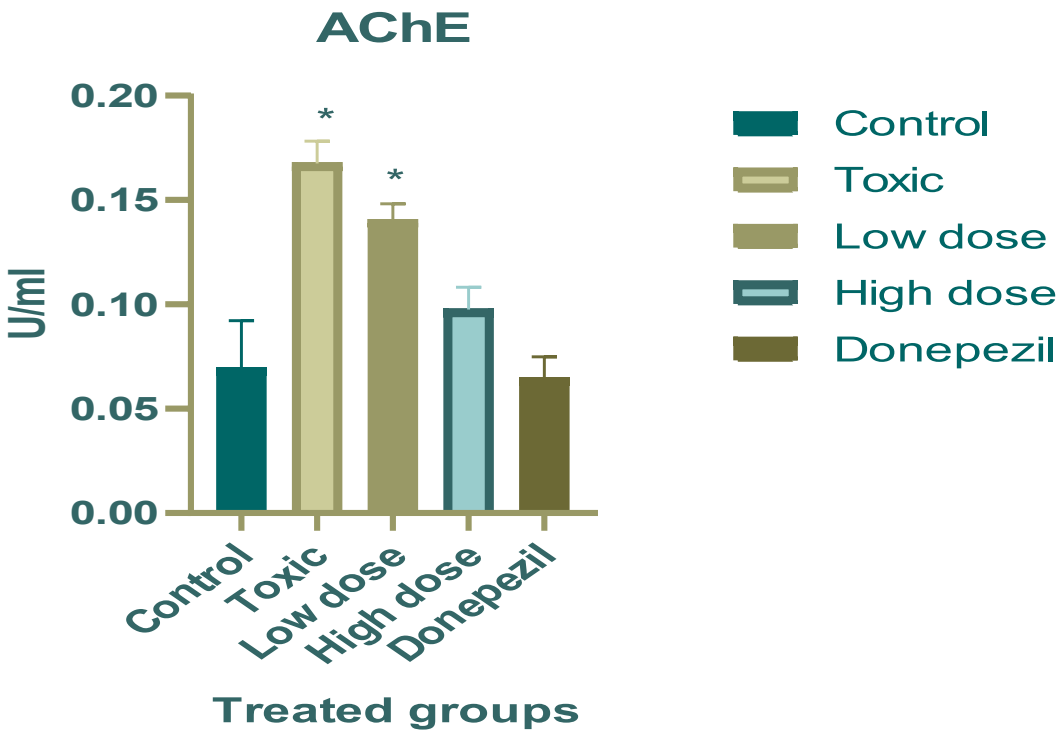

Figure 7: Bar chart showing mean ± SEM of AChE across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

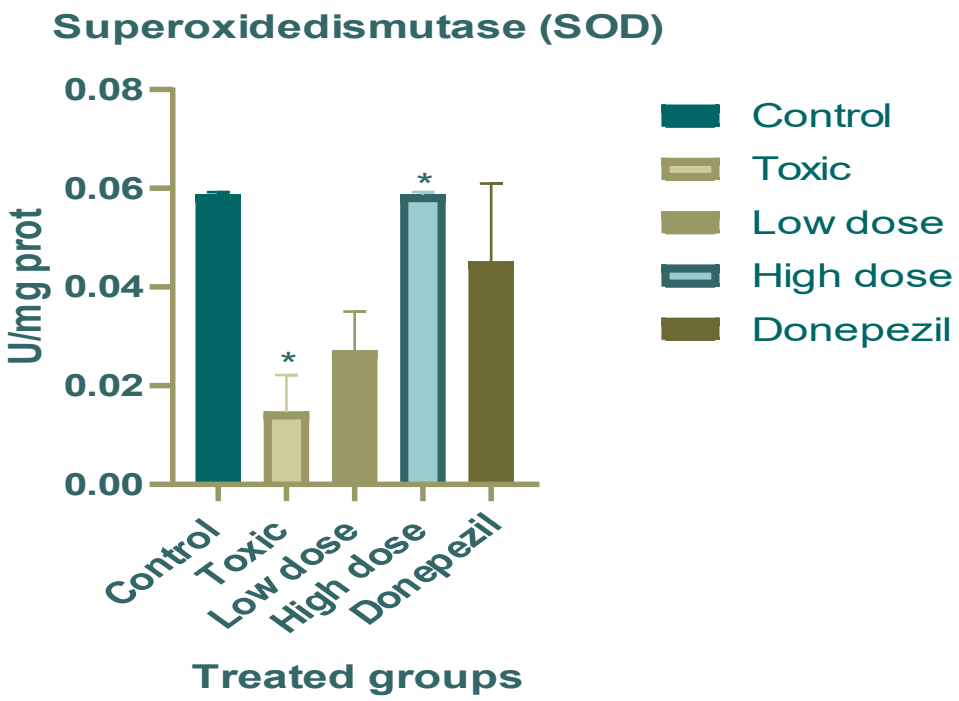

Figure 8: Bar char showing mean ± SEM of SOD level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

significantly reduced MDA relative to the toxic group, with values comparable to the control. Acetylcholinesterase activity was significantly elevated in the toxic group compared with control, while both high dose and Donepezil significantly reduced AChE; the reduction in the low-dose group was not

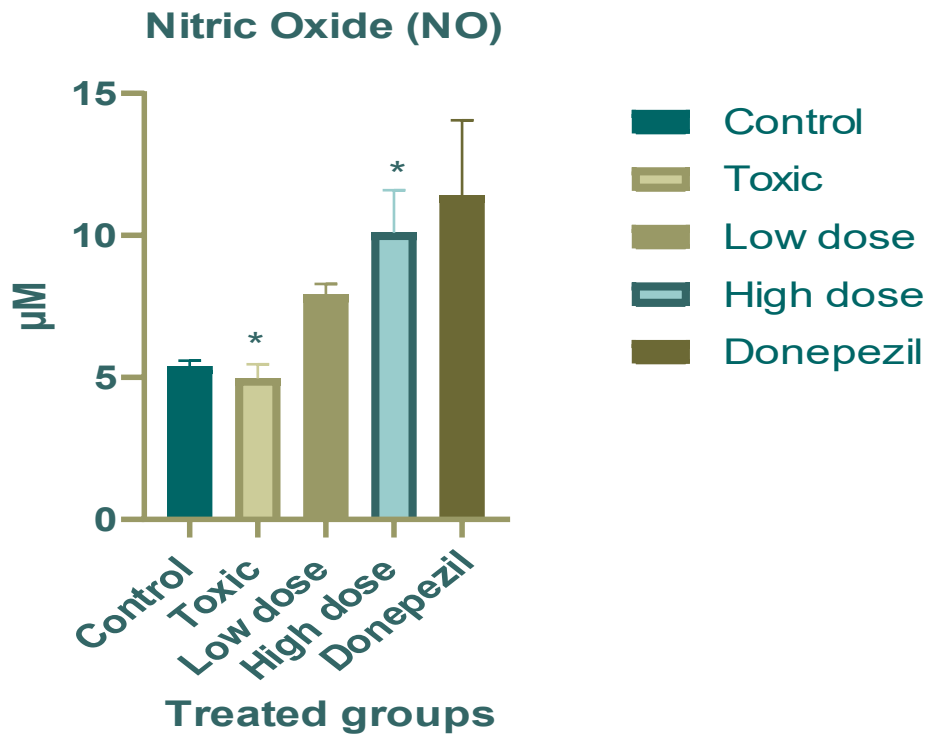

Figure 9: Chart showing mean ± SEM of NO level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

significant. Superoxide dismutase was markedly reduced in the toxic group compared with control ($p < 0.05$). In contrast, all treatment groups (low dose, high dose, and Donepezil) significantly increased SOD relative to the toxic group. Nitric oxide levels were nonsignificantly reduced in the toxic group compared with control, but significantly increased in the low-dose, high-dose, and Donepezil groups relative to the toxic group. as shown in the Figures 6, 7, 8 and 9.

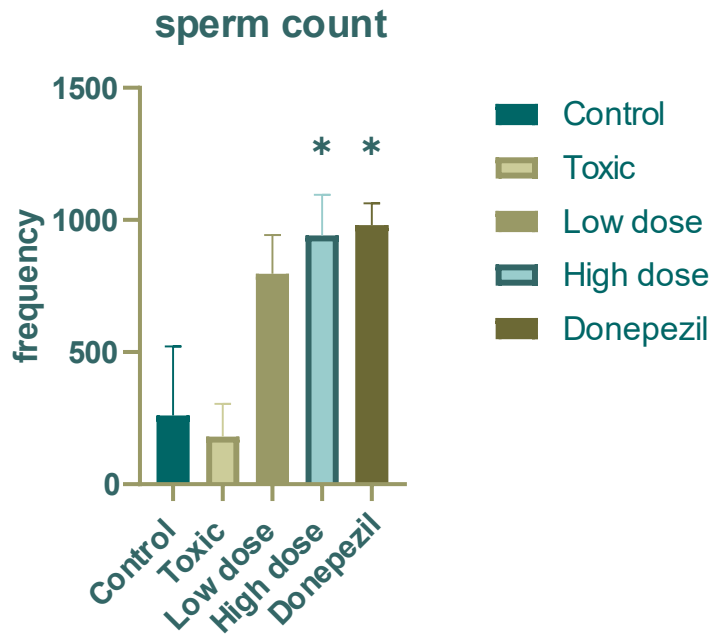

Figure 10: Bar chart showing mean ± SEM of Sperm count level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

3.5. Semen analysis

Sperm count, morphology, and motility were reduced in the toxic group compared with control, though the differences were not statistically significant. Treatment with low dose, high dose, and Donepezil

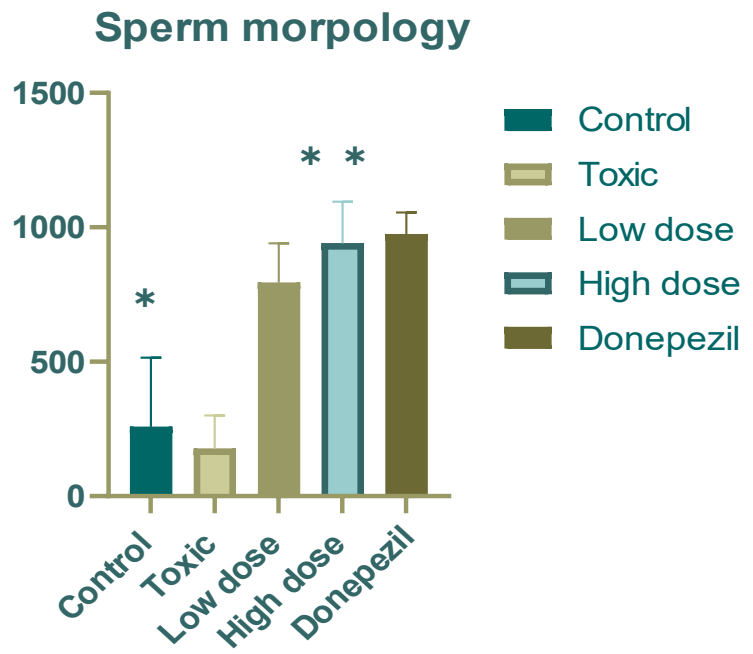

Figure 11: Bar chart showing mean ± SEM of Normal sperm morphology level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

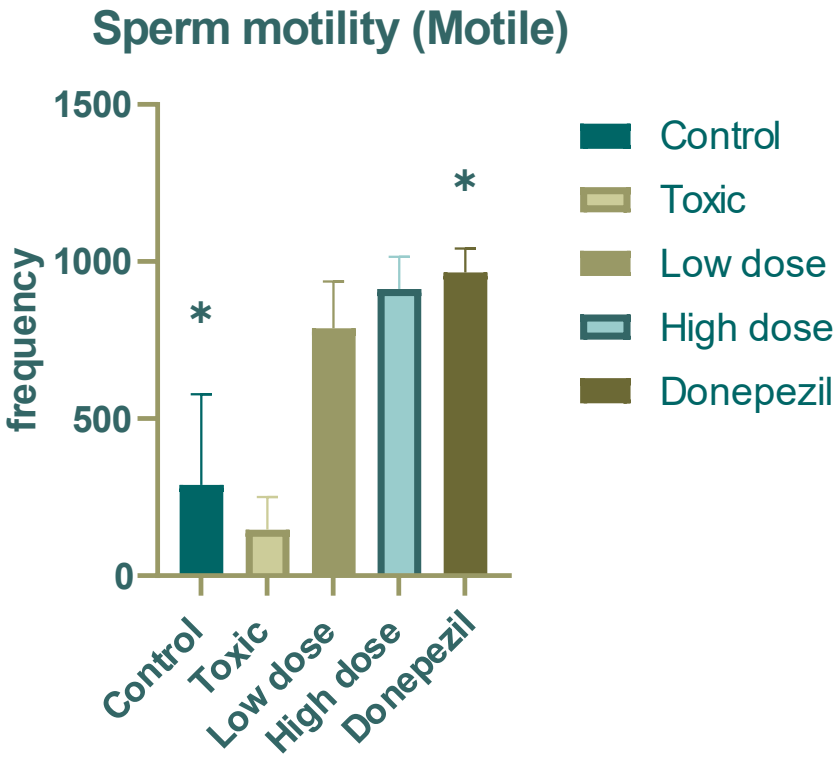

Figure 12: Bar chart showing mean ± SEM of Motile sperm level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

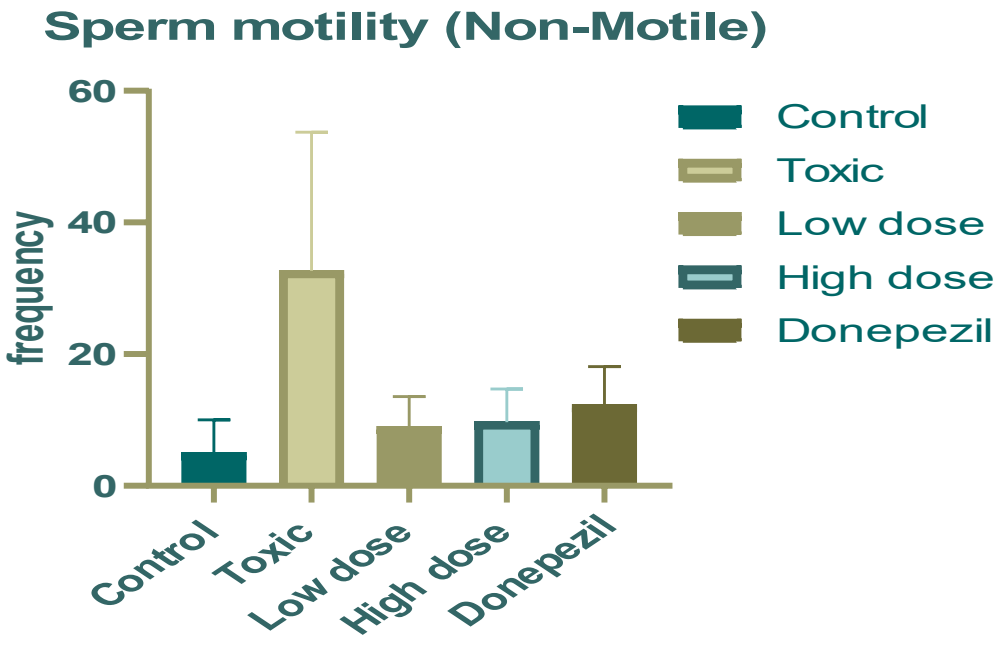

Figure 13: Bar chart showing mean ± SEM of Non motile sperm level across treatment group

Note: Asterisks (*) indicate significant differences compared to the control.

significantly improved sperm count, normal morphology, and motility relative to the toxic group ($p < 0.05$). Conversely, non-motile sperm were significantly increased in the toxic group compared with control, while all treatment groups significantly reduced non-motile sperm relative to the toxic group as shown in Figures 10, 11, 12 and 13.

3.6. Histology

The Photomicrograph of the control group showed normal testicular histology with intact basement membrane,

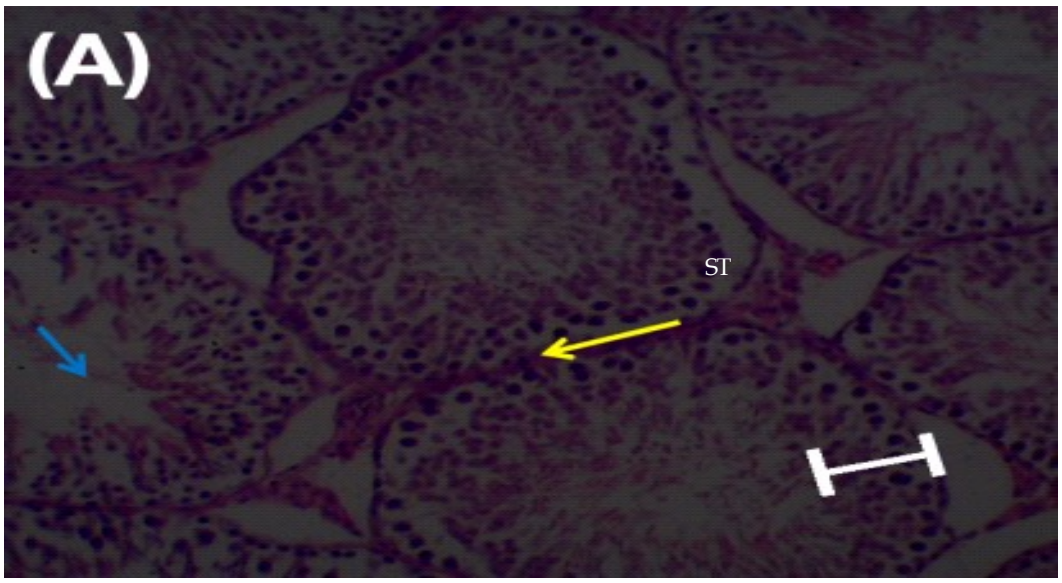

Plate 1: Photomicrograph of effect of Aidan fruit, Aluminium chloride and Donepezil. on the testes across experimental group. A. Control (2 ml/kg of distilled water), Lumen (blue arrow), Myoid cell (yellow arrow) and Germinal epithelium (white double arrow). IS (Interstitial Space). ST (Seminiferous Tubule), H&E 100x

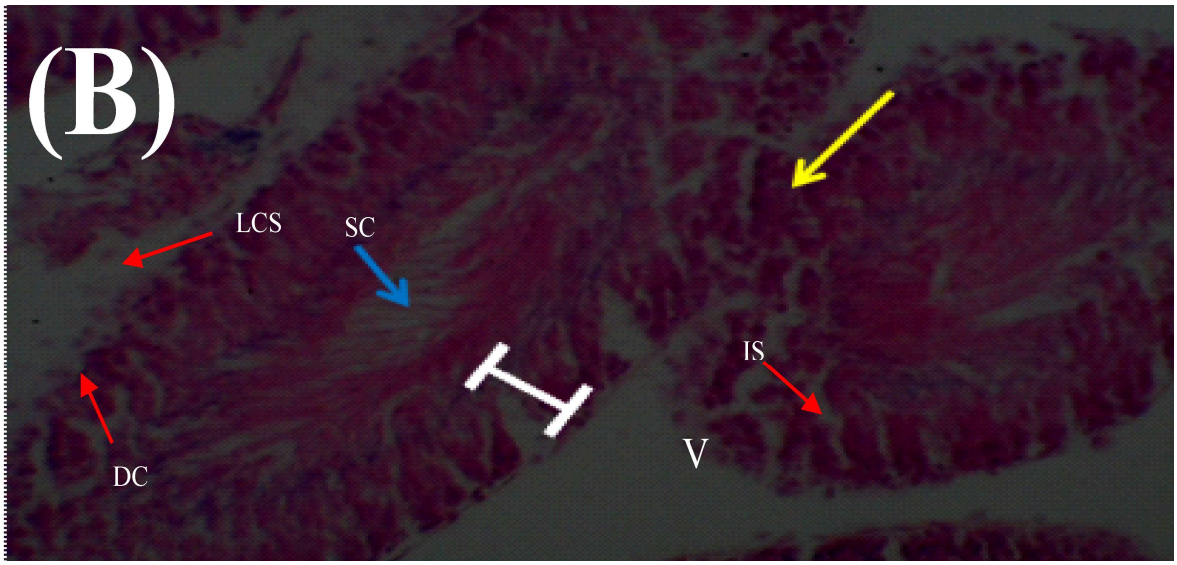

Plate 2: Photomicrograph of effect of Aidan fruit, Aluminium chloride and Donepezil. on the testes across experimental group. A. Control (2 ml/kg of distilled water), B. Toxic (100 mg/kg of AlCl3) Lumen (blue arrow), Myoid cell (yellow arrow) and Germinal epithelium (white double arrow). V (Vacuolation), T (Tubules), LCS (Leydig Cell Shrinkage), DC (Degenerating Cell). SC (Sertoli Cell). H&E x100

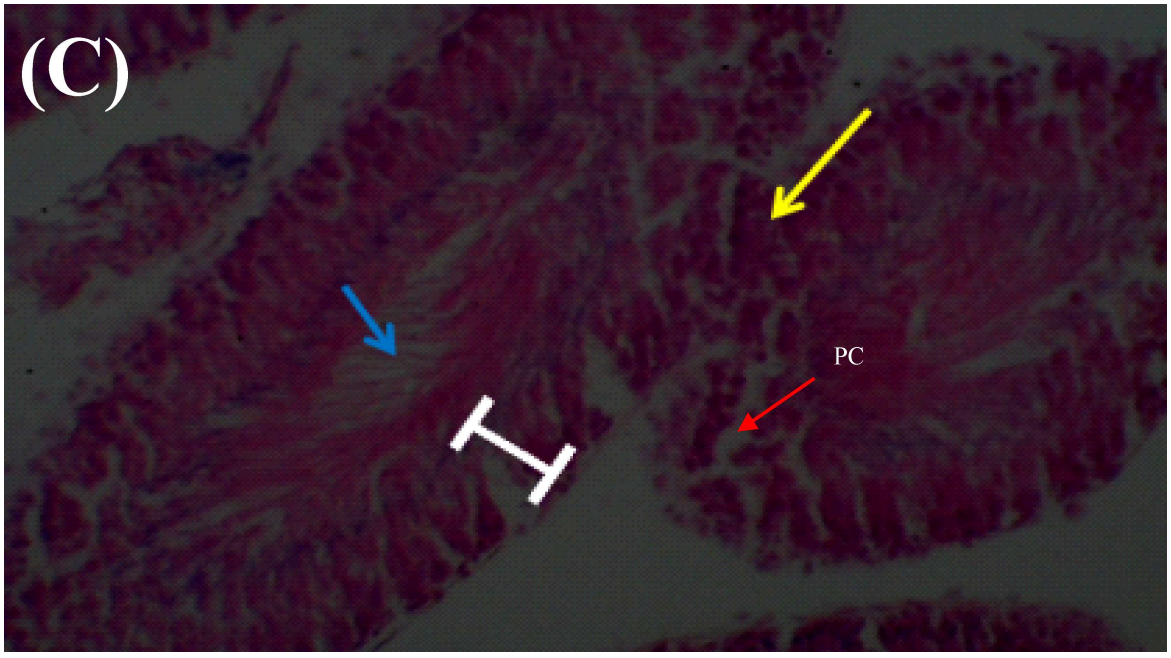

Plate 3: Photomicrograph of effect of Aidan fruit, Aluminium chloride and Donepezil. on the testes across experimental group. C. Lumen (blue arrow), Myoid cell (yellow arrow) and Germinal epithelium (white double arrow). PC (Partial Chromatolysis). H&E 100x

organized spermatogenic layers (Pate 1). However, the Toxic shows a Sparse or missing spermatogenic layers, unclear lumen boundaries, and reduced cellular density, showing impaired spermatogenesis when compared to the control (Plate 2). Meanwhile, the low dose group showed a moderate regeneration of testicular tissue. Spermatogenesis appears to be reinitiating, though not fully normalized (Plate 3). Moreover, the high dose PPT treated group showed well-defined and multilayered with active germ cells and visible spermatids showing

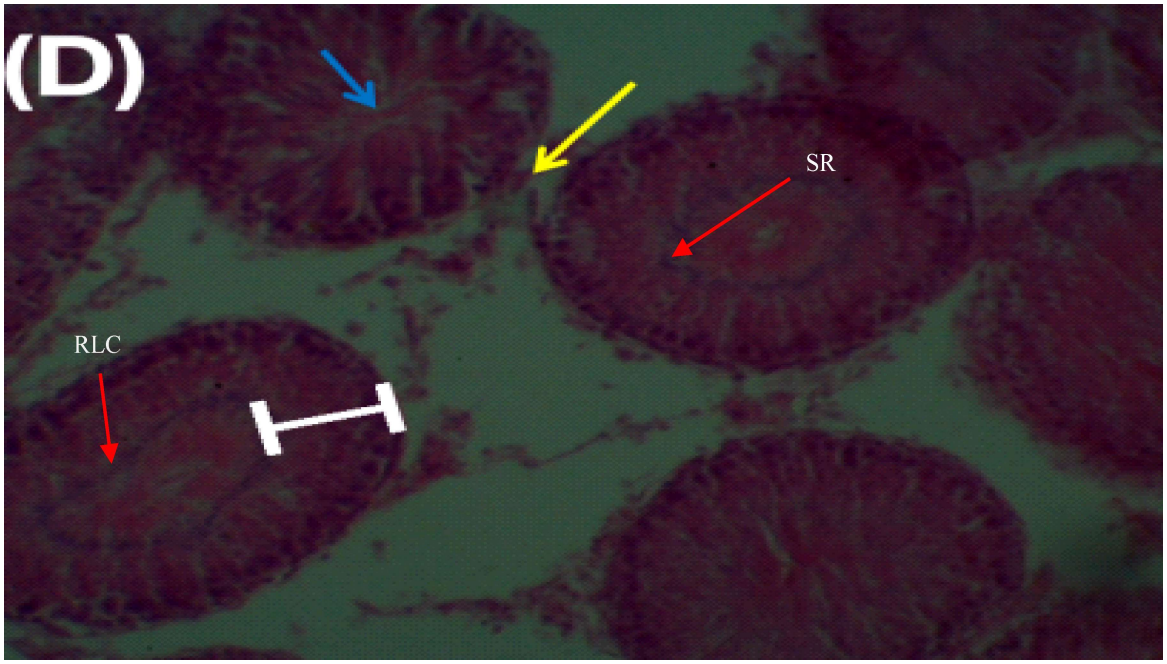

Plate 4: Photomicrograph of effect of Aidan fruit, Aluminium chloride and Donepezil. on the testes across experimental group. D. High Dose Group (100 mg/kg of AlCl3 and 500 mg/kg of PT-T). Lumen (blue arrow), Myoid cell (yellow arrow) and Germinal epithelium (white double arrow). SR (Shrinkage Restoration), RLC (Restored Leydig Cell). H&E 100x

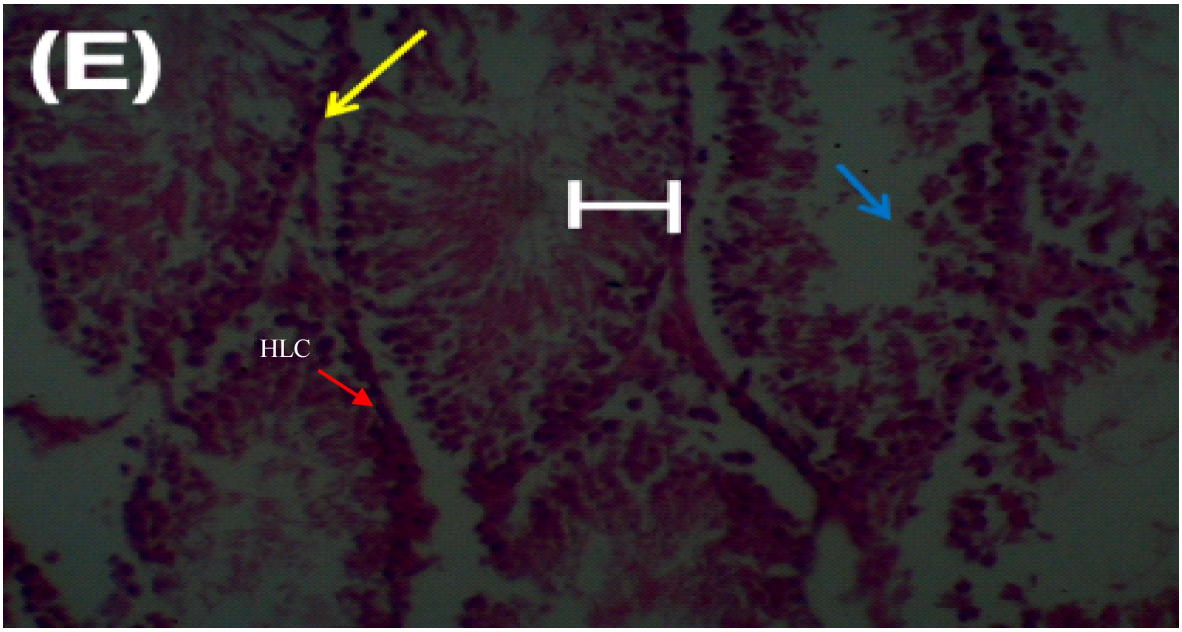

Plate 5: Photomicrograph of effect of Aidan fruit, Aluminium chloride and Donepezil. on the testes across experimental group. E. Positive Control (100 mg/kg of AlCl3 and 10 mg/kg of Donepezil). Lumen (blue arrow), Myoid cell (yellow arrow) and Germinal epithelium (white double arrow). HLC (Healing of Leydig Cell). H&E 100x

high therapeutic efficacy when compared with the low dose (Plate 4). Moreso, the Donepezil treated group showed slightly less defined cytoarchitecture when compared with the high dose PPT treated group as seen in (Plate 5).

4. Discussion

The effect of aluminum on general health and physical appearance observations recorded throughout the study provide early indicators of aluminum-induced toxicity. Rats exposed to $AlCl_3$ displayed signs such as mucous secretion, limb paralysis, feeding reduction, and ultimately, death. Behavioral distress like aggression and inactivity further affirms the neurotoxic and stress-inducing effects of aluminum. This is consistent with earlier studies (Lakshmi *et al*., 2015; Hanitrinirina, 2024). Where aluminum exposure led to systemic oxidative stress and poor survival rate in rodents.

The body weight did not show significant change ($p \leq 0.05$) across the groups. However, there was increase in the mean body weight in the toxic group. This increase in the results may depend on factors like dosage, duration of exposure, and individual animal responses. This agree with Mouro *et al.* (2018), who reported that during the experiment using male Wistar rats and body weight was monitored weekly and no body weight loss was reported and the animal maintain stable growth, despite histological changes seen. However, administration of *Tetrapleura tetraptera* (particularly at 500 mg/kg) and donepezil in Groups D and E improved body weight, suggesting a reversal of the metabolic disturbances. These results align with the findings of Adelakun *et al*. (2021), who documented that *Tetrapleura tetraptera* improves overall energy metabolism and appetite by mitigating oxidative stress.

Moreover, Aluminum exposure also led to a significant decrease ($p \leq 0.05$) in Testosterone, LH, and FSH, signifying disruption of the Hypothalamic-Pituitary-Gonadal (HPG) axis. Low levels of these hormones indicate impaired Leydig and Sertoli cell function, resulting in poor spermatogenesis. This observation correlates with findings of Lokman *et al.* (2022). Who reported that aluminum suppresses pituitary gonadotropins and testicular steroidogenesis. Treatment with *Tetrapleura tetraptera*, especially at 250 mg/kg, effectively restored testosterone and gonadotropin levels. Interestingly, the high dose (500 mg/kg) elevated FSH and AMH but reduced testosterone, possibly due to hormonal feedback inhibition or delayed Leydig cell recovery. This dose-specific hormonal effect indicates the need to carefully balance phytochemical dosing to avoid overstimulation of the endocrine axis.

Biomarkers such as MDA, SOD, AChE, and NO confirmed the role of oxidative stress in aluminum-induced testicular damage (Sabir *et al*., 2024). Elevated MDA and NO levels in the toxic group reflect intense lipid peroxidation and inflammation. Concurrent reductions in SOD and AChE indicate diminished antioxidative capacity and impaired testicular nerve signaling (Afolabi *et al*., 2018). The administration of *Tetrapleura tetraptera* at both doses reduced MDA and NO levels while improving SOD and AChE activity. This is attributed to the fruit's rich content of flavonoids (e.g., quercetin, catechin) and tannins, known for neutralizing free radicals and stabilizing cell membranes. These findings agree with those of Adusei *et al*. (2019), who demonstrated the strong In-vivo antioxidant activity of *Tetrapleura tetraptera* extracts.

Exposure to aluminum chloride drastically reduced sperm count, motility, and morphology, confirming spermatogenic arrest and germ cell apoptosis. The improvement in semen parameters in *Tetrapleura tetraptera*-treated groups, particularly the high-dose group (Group D), underscores its efficacy in restoring spermatogenesis. Although donepezil showed significant improvement, but *Tetrapleura tetraptera* proved comparable and natural.

Significantly, high-dose Aidan fruit reduced abnormal morphology and increased viable, motile sperm, demonstrating restored Sertoli cell and germinal epithelial functions. These results are in line with Adelakun *et al*. (2021), who confirmed increased sperm viability and count in male rats treated with *Tetrapleura tetraptera*.

Histological sections supported the biochemical findings. Exposure to aluminum chloride led to degeneration of seminiferous tubules, sloughed germinal epithelium, and vacuolization of interstitial tissue, the hallmarks of testicular atrophy. However, the architecture of the testis improved significantly in rats treated with *Tetrapleura tetraptera*, particularly in Group D, which exhibited reappearance of the lumen, intact germinal layers, and myoid cell continuity. This confirms the fruit's tissue regenerative and anti-inflammatory capabilities as shown in Ezeocha and Urenwoke (2022).

5. Conclusion

This study confirms that aluminum chloride induces severe testicular toxicity in Wistar rats through oxidative

stress, hormonal suppression, and direct cytological damage. These effects manifest as weight loss, poor semen quality, and histological degeneration of testicular tissue.

The Administration of phenolic extracts of *Tetrapleura tetraptera* significantly reversed these toxic effects. The low dose (250 mg/kg) showed better hormonal modulation, while the high dose (500 mg/kg) demonstrated superior restoration of sperm parameters and tissue structure. These findings support the potential use of *T. tetraptera* as a safe, cost-effective, plant-based therapy for managing male reproductive toxicity due to environmental contaminants like aluminium.

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