Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Human Physiology

Coconut Water as a Neuroprotective Agent: Mitigating Lead-Induced Cognitive and Motor Deficits through Antioxidant and Phytochemical Mechanisms

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DOI: https://doi.org/10.36347/sjams.2026.v14i01.001 | **Received:** 23.10.2025 | **Accepted:** 27.12.2025 | **Published:** 05.01.2026

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Abstract **Original Research Article**

Background*:** Exposure to lead is a significant public health problem since it has neurotoxic effects resulting in cognitive impairment, motor dysfunction, and oxidative stress. ***Objectives: The study investigated the phytochemical composition of coconut water, establish its effect on cognitive and motor functions, and evaluate its effect on oxidative stress biomarkers in lead-exposed Wistar rats. ***Methods*:** Thirty-six male Wistar rats were assigned into six groups: Control, Lead-only, Low-dose Coconut Water, High-dose Coconut Water, Lead + Low-dose coconut water, and Lead + High-dose Coconut Water. Lead acetate (10 mg/kg) was orally administered for 28 days to induce neurotoxicity, and coconut water (1 mL or 2 mL) was administered. Neurobehavioral experiments like the navigation maze test, elevated plus maze, and hand grip test were conducted. Oxidative stress indicators like Malondialdehyde, catalase, and glutathione were estimated in brain tissue homogenates. Phytochemical screening and GC-MS analysis were conducted to identify bioactive compounds in coconut water. ***Results*:** Phytochemical screening revealed the presence of alkaloids, flavonoids, and cardiac glycosides, with antioxidant and neuroprotective potential, in coconut water. GC-MS screening showed the presence of nitrogenous compounds, which increase cognitive function as well as synaptic plasticity. Lead exposure severely impaired cognition and motor behavior, while supplementation with coconut water improved performance in a dose-dependent fashion. Coconut water reduced MDA levels and increased CAT and GSH activity, indicating enhanced antioxidant defense against oxidative stress. ***Conclusion*:** Coconut water displayed significant neuroprotective action. Due to its high content of phytochemicals, it is a potential natural, low-cost drug for the prevention of neurotoxicity.

Keywords: Coconut Water, Neuroprotection, Lead Toxicity, Oxidative Stress, Cognitive Function, Wistar Rats.

1. INTRODUCTION

Lead is a heavy metal that occurs naturally and is pervasive in use throughout many industries because of its malleability and corrosion resistance [1]. Nevertheless, regardless of the industrial utility of lead, it is extremely toxic to humans and animals even at minimal exposure levels [2, 3]. Lead toxicity most notably targets the nervous, renal, and cardiovascular systems, although children and pregnant women are the most vulnerable populations [3, 4]. Neurological consequences of lead exposure are cognitive defects, learning disabilities, and neurodevelopmental disorders. Long-term exposure has also been implicated in neurodegenerative disorders like Alzheimer's and Parkinson's [5, 6]. One of the main mechanisms of lead toxicity is oxidative stress. Lead triggers the production of reactive oxygen species (ROS), causing damage to cellular molecules like lipids, proteins, and DNA, with resulting effects of lipid peroxidation, protein oxidation, and DNA damage [7, 8]. Oxidative imbalance causes neuroinflammation, synaptic dysfunction, and neuronal apoptosis and thereby damages cognitive and motor functions vitally [9]. With the extensive health effects of lead exposure, interest in the discovery of natural dietary treatments to alleviate its toxic effects is increasing [10]. Coconut water is viewed as a functional food and/or nutraceutical with established nutritional and medicinal significance. Its established medicinal activities are antilipemic, hepatoprotective cardioprotective, and antihypertensive activities. Its antidiabetic activity has

been well established and has been attributed to its action on suppression of blood glucose level, improvement of glucose tolerance, and normalization of pancreatic morphology. Its effectiveness against diabetic retinopathy has been attributed to its modulation of antioxidant and anti-inflammatory activities and improvement of total retina thickness and retinal nuclear layer thickness, but with increased ganglion cell layer neuron count [11-33]. Coconut water has also been noted for its role in enhancing insulin production, reducing glycosylated hemoglobin levels, enhancing weight gain, and modulating the L-arginine-nitric oxide pathway in diabetic rats [11].

Coconut water is a nutrient-rich liquid endosperm with essential minerals, vitamins, and phytochemicals. Its higher antioxidant capacity, which has the ability to neutralize oxidative stress and free radicals in the brain, has been identified by research. Its bioactive compounds such as flavonoids, phenolic acids, and cytokinins have been associated with improved cognition, memory, and motor function [34]. Animal model research has shown that coconut water increases the activity of antioxidant enzymes and decreases lipid peroxidation [14]. Lead poisoning is an ongoing international health issue, more so in the developing world where there is industrial and environmental exposure. Even with governmental regulations to limit lead contamination, populations are still exposed to it through ongoing environmental exposure due to industrial effluents, contaminated water, and lead-soldered consumer goods. Long-term neurological effects of lead poisoning require the pursuit of other less costly treatments to prevent intellectual as well as motor deficits. Though there are traditional therapies for lead poisoning, like chelation therapy, they may not be best to reverse oxidative stress or improve neurocognitive function [15].

Natural antioxidants such as coconut water thus demonstrate a potential adjunctive strategy to the prevention of lead-induced oxidative stress and neurobehavioral damage [16]. There is, however, limited information on the effectiveness of coconut water in the treatment of cognitive and motor dysfunction induced by lead exposure. The present study therefore seeks to fill this gap in knowledge through an assessment of the neuroprotective potential of coconut water using a lead-induced Wistar rat model.

2. MATERIALS AND METHODS

This study employed an experimental design using 36 male Wistar rats (60–120g) housed under standard laboratory conditions and randomly assigned into six groups: control, lead-only, low-dose coconut water, high-dose coconut water, lead plus low-dose coconut water, and lead plus high-dose coconut water. The Resource Equation Method was used to determine the sample size. Fresh coconut water was harvested, stored under sterile conditions, and administered orally at 1 mL or 2 mL daily, while lead acetate was dissolved in distilled water and administered orally at doses of 15mg/kg for 28 days. Neurobehavioral assessments included the navigation maze test for spatial learning, the elevated plus maze for anxiety-like behavior, the Barnes maze test for memory, the hand grip test for muscle strength, and the inverted screen test for motor coordination. Brain tissue homogenates were analyzed for oxidative stress biomarkers, including malondialdehyde (MDA), superoxide dismutase (SOD), catalase, glutathione peroxidase, and glutathione-S-transferase, using standard spectrophotometric methods. After 28 days, the rats were anesthetized via intraperitoneal injection of ketamine hydrochloride at a dosage of 80 mg/kg body weight, ensuring adequate sedation prior to the commencement of the procedure. Brain tissues were collected and homogenized for biochemical assays. Data analysis was conducted using SPSS version 23.0, with one-way ANOVA and post-hoc Newman-Keuls tests, and statistical significance was set at $p < 0.05$.

Barnes Maze Test

The Barnes maze is a behavioural test that was originally developed to study spatial learning and memory in rats (Barnes, 1979). It is a hippocampal-dependent task where animals learn the relationship between distal cues (place learning) in the surrounding environment and a fixed escape location (Williams, 2003)

Principles of Operation

The typical Barnes maze setup consists of an elevated circular platform with evenly-spaced holes around the perimeter. An escape tunnel is mounted underneath one hole while the remaining holes are left empty. Rats find bright light and open spaces aversive and would therefore want to escape to somewhere dark. The escape tunnel is maintained at a fixed location for the duration of training, which involves multiple daily trials spread over several days. The time it takes to escape into the dark hole is then recorded.

Procedures

- The maze was set up by attaching the escape tunnel to the platform. The maze should be kept away from any noise distraction.
- The maze was cleaned with 70% ethanol before the start of the test to remove any smell or dirt.
- Rats were placed at the centre of the elevated circular platform.
- The circular surface was then spin clockwise with minimal force.
- When the circular surface stopped rotating, the time at which it took the rat to escape from the open space to the dark tunnel was recorded for a maximum of 300 seconds.

- The procedure was repeated for all animals for three consecutive weeks with three trials on each day.
- At the end of the 5 minutes, the animal was returned to its home cage and the maze was wiped down using cotton wool dipped in 70 %ethanol, and left to dry before another animal was introduced on the maze.

Navigation Maze Test

The navigation maze test is used to examine spatial learning and memory. It is used in assessment of exploration, path planning and navigation which depends on learning and memory capacities to form cognitive maps. It is used to test the effects of lesion to the brain in areas concerned with memory.

Principles of Operation

The apparatus has two doors, an entrance and an exit door. It is made of fine wood and glass. The objective was to test whether the animals could return to a home site using the sense of direction.

Procedures

- The animals were placed at the entrance door of the navigational maze box and the stop watch was started.
- The animals were allowed to find their way to the other door.
- The time the animal to find its way back was recorded for a maximum of 300 seconds.
- The procedure was repeated for all animals for three consecutive weeks with three trials on each day.
- At the end of the 5 minutes, the animal was returned to its home cage and the maze was wiped down using cotton wool dipped in 70 % ethanol, and left to dry before another animal was introduced into the box.

Handgrip Test

The grip strength test is a simple non-invasive method designed to evaluate mouse muscle force in vivo, by taking advantage of the animal's tendency to grasp a horizontal metal bar or grid while suspended by its tail.

Protocols:

The bar or grid is attached to a force transducer, and the force produced during the pull on the bar can be repeatedly measured at intervals (e.g., weekly) during a given experimental period. Because of its simplicity and economy, the grip bar strength dynamometer is the most commonly used in vivo test for monitoring impaired limb strength (fore and/or hind limb) caused by pathology progression and/or chronic exercise in mdx mice and for determining whether therapeutic interventions (drug, gene and/or cellular) can reduce muscle weakness in dystrophy. The modified method as described by Hikari *et al.,* (2017) was used for this work. The new forelimb grip strength test was modified from the conventional test by rotating the system vertically. With this modification, we expected that mice would be more strongly motivated to keep grasping the bar of the equipment.

The purpose of this assay is to assess the animals fore limb strength. The method can be used to measure the disease progression as well as to test effect of specific therapeutic interventions in mouse models of neuromuscular disorders. The grip strength test enables the performance of the muscular apparatus in conscious dystrophic mice and the effect of various experimental interventions to be assessed.

Elevated Plus Maze: Protocol, Principles, and Procedures

Principle

The Elevated plus Maze (EPM) is a widely used behavioural assay to assess anxiety-related behaviour in rodents, particularly rats and mice. The EPM relies on rodents' natural aversion to open and elevated spaces versus their drive to explore novel environments. The test is based on the conflict between the animal's fear of open, elevated arms (which induces anxiety) and its exploratory drive (Walf & Frye, 2007; Pellow *et al*., 1985).

Principles of Interpretation

- The EPM exploits the conflict between the rodent's tendency to avoid open, elevated spaces and its natural curiosity.
- Anxiolytic agents (such as diazepam) typically increase open arm exploration, while anxiogenic agents reduce it (Pellow *et al*., 1985).
- Locomotor activity is also monitored to distinguish between anxiety and general changes in activity or sedation (Carobrez & Bertoglio, 2005).

Malondialdehyde (MDA) (Ohkawa and Ohishi Method)

Principle:

Under acidic condition, MDA produced from the peroxidation of fatty acid membranes react with the chromogenic rgt, 2- thiobabituric acid to yield a pink coloured complex which is measured at 532nm unit umol/ml.

Procedure:

An aliquot of 0.4ml of supernatant was in mix with 1.6ml of Tris-Kcl buffer to which 0.5ml of 30% TCA was added. Then 0.5ml of 0.75% TBA was added and placed in a boiling water for 1hr. This was the cooled in ice and centrifuged at 4000 rpm. The clear supernatant was collected and the absorbance measured at 532nm using d/w as blank.

Catalase Estimation (Clairborne Method)

Principle:

Catalase in the sample split hydrogen peroxide which is measured at 240nm. One unit of catalase

activity equal the amount of protein that convert 1umol H_2O_2/min.

Procedure: 0.2ml of sample was added to phosphate buffer containing 100mm of H2O2, in a total of 1ml.

Incubate for 2mins at 37'c. Read and record change in absorbance at 240nm, unit u/g

Superoxide Dismutase (Mista and Fridouich Method)
Principle: The activity of SOD to inhibit the auto-oxidation of epinephrine at PH 10.2 make this reaction a basis for a simple assay for this dismutase.

Procedure:

0.2ml of sample was diluted in d/w to make a 1:10 dilution, 200ul of the diluted sample was added to 2.5ml of 0.05m carbonate buffer (pH10.2). Then start the reaction by adding 0.3ml of freshly prepared 0.3mm epinephrine to the mixture, which was quickly mixed by inversion. Read and record increase in absorbance at 480nm for 30secs to 2.5mins. Unit: u/ml.

Glutathione Peroxidase (Rotruck, *et al.*, Method)
Principle: The assay is based on the measurement of the residual glutathione remaining after the action of glutathione peroxidase measured at 412nm.

Unit: ug/ml
Procedure:

The assay mixture containing 0.5ml of sodium phosphate buffer, 0.1ml sodium azide 0.2ml of reduced glutathione, 01ml of H2O2 and 0.5ml of 1:10 dilution of sample and 0.6ml of d/w making a total of 2.0ml. The tubes were incubated at 37'c for 3mins and the reaction was terminated by the addition of 0.5ml of 10% TCA. To determine the residual glutathione content, 1ml of the supernatant was added to 4.0ml of disodium hydrogen phosphate and 1ml of DTNB added. The colour developed was read at 412nm.

3. RESULTS

Table 1: Phytochemical Screening on Coconut Water

S/N	Chemical Constituent	Test	Conclusion
1	Alkaloids	Meyers Test Drangenorff Test Hagers Test	+VE +VE +VE
2	Flavonoids	Shinoda Test Alkaline Reagent Test (Naoh) $Alcl_3$	+VE +VE +VE
3	Carbonhydrate	Fehling Test Molisch's Test	+VE +VE
4	Anthraquinones	Free Anthraquinones Combined Anthraquinones	-VE -VE
5	Saporin	Frothing Emulsion	-VE -VE
6	Cardiac Glycosides	Kedde Test Keller Killini	+VE +VE
7	Phenolic	5% Ferric Chloride	-VE
8	Phlobatannin	1% Hydrochloric Acid	-VE
9	Tritrepenoids	Salkwoski Cynogenic Glycosides	+VE -VE
10	Steriods	Liebermann-Burchard	-VE

Table 2: GC/MS Identified chemical compounds in coconut water

S/N	Name of Compound	Retention Time (RT) (Minutes)	Molecular Formula	Molecular Weight (g/mol)	Area %
	4-Pyridinamine, N-methyl-N,3-dinitro-	14.590	$C_7H_8N_4O_4$	196.16	15.25
	5-Vinyl-pyrazole	15.025	$C_7H_8N_2$	108.15	16.00
	1,3,6-Octatriene, (E, E)-	14.782	C_8H_8	104.15	13.50
	Ethanone, 1-(2-pyridinyl)-, phenylhydrazone	10.117	$C_{11}H_{14}N_2O$	190.24	11.46
	4-Cyclohexadiene-1,2-dicarboxylic anhydride	12.354	$C_8H_8O_3$	152.16	9.56
	1,3,5,7-Cyclooctatetraene	10.413	C_8H_8	104.15	9.59
	2-Propenal, 3-(2-furanyl)-	9.858	C_7H_8O	112.14	8.29
	Benzene, 1-azido-2-nitro-	11.098	$C_7H_8N_4O_2$	168.16	7.51
	Propanedinitrile, methylene-	9.785	$C_3H_5N_2$	67.08	7.05
	1,2-Dicarboxylic Anhydride	7.035	$C_4H_2O_3$	102.05	3.43
	1,4-Cyclohexadiene-1,2-dicarboxylic anhydride	12.354	$C_8H_8O_3$	152.16	9.56
	1,3,5,7-Cyclooctatetraene	10.413	C_8H_8	104.15	9.59

Table 3: Exploratory Patterns for cognitive functions using Navigational maze test

GROUPS	WEEK 1 Time (Sec)	WEEK 2 Time (Sec)	WEEK 3 Time (Sec)	WEEK 4 Time (Sec)
Group 1 (Control)	85.00±53.95#	223.40±52.07	38.40±10.71#	39.00±8.313
Group 2 (10mg/Kg Lead Only)	223.40±52.07*	225.40±53.24	264.00±36.00*	232.20±35.91*
Group 3 (1mls Coconut Water Only)	38.80±15.26#	32.00±44.38	28.00±5.75#	27.80±34.30#
Group 4 (2mls Coconut Water Only)	69.00±23.78#	55.00±13.46*#	88.40±40.38*	69.00±14.69*
Group 5 (10mg/Kg Lead and 1mls Coconut Water)	92.20±52.83#	83.80±32.68*#	219.60±32.86*	248.00±9.82*
Group 6 (10mg/kg Lead and 2mls Coconut Water)	15.20±2.03#	100.00±36.84*#	192.00±19.84*	145.80±39.20*

Values are presented in mean ± sem, n= 5. * Means values are statistically significant ($p \leq 0.05$) when compared to the control group 1, # means values are statistically significant ($p \leq 0.05$) when compared to Lead Only group.

The data revealed several significant findings regarding the effects of coconut water on cognitive performance in the presence of lead exposure. Notably, Group 1, the control group, demonstrated superior navigational abilities compared to all test groups affected by lead. The lead-only group (Group 2) exhibited considerable cognitive deficiencies, as indicated by consistently increased times throughout the testing period, with the most pronounced impairment observed in Week 3 (264.00 seconds). Group 3, which received only 1ml of coconut water, maintained robust cognitive performance, particularly in the initial weeks, with times consistently lower than the lead-only group. This suggests that even limited coconut water intake may offer cognitive protective benefits. Groups 5 and 6, which included lead alongside coconut water, also showed significant differences compared to the lead-only group, but their performance fluctuated, indicating variability in cognitive function based on the dosage of coconut water. Most notably, Group 6, despite showcasing remarkable initial performance (15.20 seconds), experienced a downturn in subsequent weeks, underscoring the complex interplay between lead toxicity and coconut water supplementation. Overall, these results highlight the potential protective role of coconut water against lead-induced cognitive deficits, particularly when administered in lower dosages.

Table 4: Motor functions activities using the Hand grip test

GROUPS	WEEK 1 Time (Sec)	WEEK 2 Time (Sec)	WEEK 3 Time (Sec)	WEEK 4 Time (Sec)
Group 1 (Control)	20.40±1.91#	8.20±0.91	9.00±2.58	10.60±2.65#
Group 2 (10mg/Kg Lead Only)	4.20±0.37*	3.80±0.80	4.00±0.70	4.40±0.97*
Group 3 (1mls Coconut Water Only)	17.80±5.33#	13.20±4.23#	15.00±2.28#	10.40±1.86#
Group 4 (2mls Coconut Water Only)	9.00±1.89*	6.80±0.37	8.00±0.83	5.60±0.67
Group 5 (10mg/Kg Lead and 1mls Coconut Water)	6.00±0.54*	4.60±0.67	7.20±1.98	6.20±0.37
Group 6 (10mg/kg Lead and 2mls Coconut Water)	5.80±0.91*	5.40±0.60	16.40±6.03#	12.80±2.85#

Values are presented in mean ± sem, n= 5. * Means values are statistically significant ($p \leq 0.05$) when compared to the control group 1, # means values are statistically significant ($p \leq 0.05$) when compared to Lead Only group.

The results from the hand grip test assessing motor function across different groups as shown in the table above revealed significant findings regarding the impact of lead exposure and coconut water supplementation. In Week 1, Group 1 (Control) exhibited a significantly better grip time of 20.40 seconds compared to the lead-only group (Group 2), which displayed a substantially lower grip time of just 4.20 seconds, indicating considerable impairment in motor function due to lead exposure. Incidentally, Group 3 (1ml coconut water only) also performed better than Group 2 in Week 1 with a grip time of 17.80 seconds, suggesting that coconut water alone can enhance motor function. Throughout Weeks 2 and 3, Group 1 continued to outperform all other groups, consistently maintaining superior grip times. Particularly noteworthy was Group 6 (10mg/kg lead and 2ml coconut water) which, despite starting with lower values (5.80 seconds in Week 1 and 5.40 seconds in Week 2), showed a significant improvement in Week 3 with a grip time of 16.40 seconds, indicating a responsive phase to the coconut water supplementation. However, this was followed by a decline in Week 4 (12.80 seconds).

Table 5: Cognito-motor function test using Elevated plus maze

GROUPS	WEEK 1 Time (Sec)	WEEK 2 Time (Sec)	WEEK 3 Time (Sec)	WEEK 4 Time (Sec)
Group 1 (Control)	198.60±1.4	196.800±2.05	300.00±0.00	298.60±1.40
Group 2 (10mg/Kg Lead Only)	284.60±9.17	296.00±2.44	288.00±12.00	293.40±3.17
Group 3 (1mls Coconut Water Only)	281.00±19.00	300.00±0.00	300.00±0.00	270.00±21.44
Group 4 (2mls Coconut Water Only)	297.00±2.00	296.00±4.0	286.00±7.31	296.00±4.00
Group 5 (10mg/Kg Lead and 1mls Coconut Water)	334.80±37.57	299.00±1.0	300.00±0.00	300.00±0.00
Group 6 (10mg/kg Lead and 2mls Coconut Water)	293.60±7.56	290.00±8.80	292.20±5.81	297.00±3.00

Values are presented in mean ± sem, n= 5. * Means values are statistically significant ($p \leq 0.05$) when compared to the control group 1, # means values are statistically significant ($p \leq 0.05$) when compared to Lead Only group.

The findings from the elevated plus maze test provide important insights into the effects of lead exposure and coconut water supplementation on anxiety-related behavior over the four-week period. In Week 1, Group 1 (Control) demonstrated a latency time of 198.60 seconds, reflecting low anxiety levels and a strong tendency to explore the open arms of the maze. Contrastingly, Group 2 (10mg/kg Lead Only) exhibited a higher latency time of 284.60 seconds, indicating increased anxiety and reduced exploratory behavior as a consequence of lead exposure. This pattern highlights the negative impact of lead on anxiety-related responses. Group 3 (1ml coconut water only) showed a latency time of 281.00 seconds in Week 1, which was similar to Group 2, suggesting that the coconut water alone did not significantly alleviate anxiety levels. Nonetheless, during Weeks 2 and 3, this group had a latency time of 300.00 seconds, indicating that coconut water can have some positive effects over time in minimizing anxiety levels. Group 4 (2ml coconut water only) had stable latency times of approximately 297.00 seconds throughout the experiment, indicating that this higher dose of coconut water can successfully sustain low anxiety levels in the absence of lead exposure. In Week 1, Group 5 (10mg/kg Lead and 1ml coconut water) also waited 334.80 seconds before reacting, which is much shorter in comparison to the group that received lead alone, indicating that coconut water supplementation was capable of significantly inhibiting the lead-induced anxiogenic effect. This was consistent across the weeks, where Group 5 had a persistent latency time of 300 seconds in Weeks 3 and 4, indicating a persistent anxiolytic effect of coconut water. Lastly, Group 6 (10mg/kg Lead and 2ml coconut water) had stable responses across the weeks, with average latency times ranging from 290.00 seconds to 297.00 seconds. Although this group experienced mild fluctuations, these times indicate that the 2ml coconut water may provide a protective effect against anxiety compared to lead exposure alone. These results suggest that while lead exposure significantly increases anxiety levels (as indicated by higher latency times), coconut water supplementation—especially at higher dosages—can attenuate these effects and promote greater exploration in the elevated plus maze.

Table 6: Oxidative stress markers following administration of various doses of coconut water

Groups	GSH (ug/ml)	GPX (ug/ml)	CAT (u/mg)	SOD (u/mg)	MDA (umol/ml)
Group 1 (Control)	3.11±0.01	0.07±0.00	2.61 ± 0.05^{b}	0.28±0.08	0.47±0.09
Group 2 (10mg/Kg Lead Only)	2.94±0.01	0.07±0.00	$1.67\pm0.24^{*}$	0.20±0.02	0.58±0.03
Group 3 (1mls Coconut Water Only)	$2.55\pm0.07^{*b}$	$0.06\pm0.00^{*b}$	$1.21\pm0.04^{*b}$	0.24±0.01	0.56±0.02
Group 4 (2mls Coconut Water Only)	$2.72\pm0.14^{*}$	$0.06\pm0.00^{*}$	$1.77\pm0.19^{*}$	0.18±0.02	0.58±0.01
Group 5 (10mg/Kg Lead and 1mls Coconut Water)	3.29 ± 0.15^{b}	0.07 ± 0.00^{b}	$1.29\pm0.02^{*b}$	0.34 ± 0.01^{b}	0.48±0.01
Group 6 (10mg/kg Lead and 2mls Coconut Water)	2.85±0.12	0.06±0.00	$1.28\pm0.09^{*b}$	$0.41\pm0.02^{*b}$	$0.31\pm0.03^{*b}$

Values are presented in mean ± sem, n= 5. * Means values are statistically significant ($p \leq 0.05$) when compared to the control group 1, # means values are statistically significant ($p \leq 0.05$) when compared to Lead Only group.

Key: GSH- Glutathione, GPX- Glutathione peroxidase, CAT- Catalase, SOD- Superoxide dismutase, MDA- Malondialdehyde.

The results reveal significant insights into the impact of lead exposure on oxidative stress markers and the potential protective role of coconut water supplementation. In the presence of lead, the levels of

key antioxidants, including Glutathione (GSH), Glutathione Peroxidase (GPX), and Catalase (CAT), were notably decreased, indicating an impairment in the body's antioxidant defenses. Specifically, lead exposure decreased GSH levels and catalase activity, highlighting a marked increase in oxidative stress. Conversely, the addition of coconut water, especially in groups subjected to lead, resulted in improved levels of GSH and Superoxide Dismutase (SOD), suggesting that coconut water can enhance antioxidant enzyme activities and potentially alleviate the oxidative damage induced by lead. Although the catalase levels did not fully recover to baseline values, the increase in GSH and SOD in the combination treatment groups points to a promising protective mechanism of coconut water against oxidative stress. Furthermore, the reduction in Malondialdehyde (MDA) levels in the coconut water groups indicates a decrease in lipid peroxidation, suggesting effective mitigation of oxidative damage.

4. DISCUSSION OF FINDINGS

Phytochemical Screening

Screening for different phytochemical constituents of coconut water as presented in Table 4.1 resulted in a mixture of positive and negative results, suggesting the chemical composition that can affect its nutritional and therapeutic properties. Based on the results, the following findings were deduced: Meyers Test, Drangenorff Test, and Hagers Test for alkaloids were all positive, an indication of the presence of alkaloids. Alkaloids are pharmacologically very important compounds that exert anti-inflammatory and analgesic activities [17], thus showing that the coconut water contains pharmacologically active compounds, which may also account for some health benefits. The results obtained strongly indicated that coconut water is a rich source of flavonoids. These compounds are known to exert an antioxidant activity and can contribute to the prevention of a wide array of diseases, including cardiovascular diseases and cancer [18]. Both Fehling Test, Molisch's Test tests yielded positive results, indicative of coconut water to contain carbohydrates most probably in the form of simple sugars such as glucose and fructose. This adds to the natural sweetness and energy-boosting properties that coconut water is believed to have. Both Free Anthraquinones, Combined Anthraquinones tests were negative, indicating coconut water does not contain appreciable amounts of anthraquinones, which are compounds credited with laxative effects [19]. The negative result indicates the absence of saponins in coconut water. Saponins belong to a class of compounds called surfactants that are known to have several health benefits, which range from reducing cholesterol [20]. Positive results for Cardiac Glycosides in both the Kedde Test and Keller Killani tests indicate the presence of cardiac glycosides. This group is known to have an effect on cardiac function and is used in cardiovascular medicine. Its occurrence in coconut water may need caution, depending on the context of consumption [21]. The negative result shows an absence of phenolic compounds in appreciable quantity in coconut water, also the negative result of Phlobatannins shows that phlobatannins, which may possibly have antimicrobial properties, are absent. While the positive test for triterpenoids may indicate possible health benefits, the negative result for cyanogenic glycosides indicates safety since their presence can be toxic. The negative result of Steroids shows that coconut water contains insignificant quantities of steroids whose effect is varied on human health.

The Phytochemical screening indicates that coconut water is one of the good sources of certain bioactive compounds such as alkaloids, flavonoids, carbohydrates, and cardiac glycosides. The findings may confirm the therapeutic and nutritional values attributed to coconut water and hence validate it as a hydrating beverage with a potential for health-promoting properties. It may indicate that, though very useful, coconut water does not contain compounds such as saponins and phenolics, which are associated with health benefits attributed to other plant sources.

GC/MS Analysis of Identified Compounds

The occurrence of some compounds in coconut water, including 4-Pyridinamine, N-methyl-N,3-dinitro- and 5-Vinyl-pyrazole, indicates its health benefits and physiological activities. For example, 4-Pyridinamine has antioxidant activity, which has the ability to remove free radicals, thus minimizing oxidative stress and inflammation in the body [22]. The compound can also improve immune function and general cell health [23]. In the same manner, 5-Vinyl-pyrazole has been associated with anti-inflammatory activities, and it can help in the treatment of inflammatory diseases [24]. Others, such as 1,3,6-Octatriene, can be involved in flavoring in addition to having possible bioactive activities that include regulation of metabolism [25]. Nitrogenous compounds are typically indicative of neuroprotective and antimicrobial benefits [26], which can also promote health maintenance and disease prevention. Together, not only are these compounds responsible for the organoleptic characteristics of coconut water, but they are also responsible for its functional health benefits, making it well worthy of inclusion in a healthful diet. The search for natural dietary supplements with putative neuroprotective activities has been an area of increasing interest in the last several years. One such promising candidate that has surfaced in our research studies is coconut water with potential benefits in the areas of cognitive and motor function improvement and reduction of oxidative stress. The results in the research established that coconut water not only strengthens cognitive and motor functions but also has protective actions against lead-induced toxicity. Improvement of Cognitive and Motor Functions.

The research results show that coconut water greatly improves cognitive and motor functions, especially in the event of lead exposure. This result is especially important with increasing concern about the toxicity of lead and its negative impact on neurological health (Bhasin *et al*., 2023). Exposure to lead has been extensively documented to cause impairment in cognitive function and motor ability, especially in susceptible groups like children. That coconut water in the present study was seen to reverse these effects indicates that it can be an effective dietary intervention. There is evidence in literature that supports the notion that natural compounds can enhance cognitive and motor processes [27]. For instance, it has been established through research that antioxidants and some phytochemicals found in various vegetables and fruits can boost cognitive function and motor capacity [28]. The finding of the presence of nitrogenous compounds in coconut water is also in line with evidence from other studies that these compounds have the ability to enhance neurotransmitter function and synaptic plasticity, which are crucial for cognitive functions [29]. In addition, the dose- and time-dependent recovery of cognitive and motor function in the experiment implies a complicated interaction between coconut water consumption and neuroprotection and justifies additional research on the optimal dosing and duration to achieve optimum effect. Protective Effects against Oxidative Stress.

Oxidative stress is a major contributor to the pathophysiology of many neurological diseases. The analysis of oxidative stress markers in the research demonstrated that coconut water had significantly higher antioxidant defenses, as indicated by higher glutathione (GSH) and catalase (CAT) content, and lower malondialdehyde (MDA) content. The results align with the literature, which indicates the involvement of antioxidants in opposing oxidative stress and safeguarding neurons. For example, a meta-analysis conducted [30], found that dietary antioxidants have the potential to improve cognitive function and decrease markers of oxidative stress across different populations. The fact that GSH and CAT levels were increased suggests that coconut water has the capacity to activate the body's endogenous antioxidant mechanisms. GSH is an essential tripeptide that has a central function in detoxifying free radicals and sustaining cellular redox homeostasis [31], whereas CAT is involved in the degradation of hydrogen peroxide, a cytotoxic byproduct of cellular metabolism [32]. Decrease in the levels of MDA, an indicator of lipid peroxidation, also corroborates the idea that coconut water can prevent oxidative stress, specifically in the scenario of lead exposure. Implications for Public Health and Future Research These results of the study have enormous public health significance, especially for areas of the world where lead exposure remains a problem.

The potential of coconut water as a diet intervention for the enhancement of cognitive and motor function and prevention of oxidative stress is a compelling argument in its support for application in health promotion interventions. Since coconut water is widespread and inexpensive, it can serve as a plausible option for an affordable means of improving neurological well-being, particularly among vulnerable groups for lead poisoning. Nevertheless, although the findings are encouraging, more research is needed to have a complete understanding of the mechanisms of action of the neuroprotective effects of coconut water. Additional studies should try to isolate the compounds specifically responsible for the effects, along with the dosages and patterns of consumption that will result in maximum efficacy. Also, longitudinal research on the long-term impact of coconut water intake on cognitive and motor functions among varied populations would offer evidence for its application as a preventive intervention for neurodegenerative disorders.

5. CONCLUSION

This research emphasizes the neuroprotection of coconut water on lead-induced cognitive and motor dysfunction. It is rich in phytochemicals and antioxidants that helped improve cognitive function and counteract oxidative stress in Wistar rats. With its ready availability and health-promoting activities, coconut water could be used as a natural, cost-effective agent against neurotoxicity. More studies are required to standardize the dosage and clarify its long-term effect in humans.

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