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Anticonvulsant and Neuroprotective Effects of Virgin *Cocos nucifera* oil in PTZ and MES Seizure Models

Joffa, Price Paul Kwaku [2], Chukwuma, Samuel Anakwe [2,*], Ogu, Oyinbrakemi Collins [1] and Eferebo, Rebecca Ogoniba [1]

[1] *Department of Biochemistry, Faculty of Basic Medical Sciences, Niger Delta University, Wilberforce Island, Bayelsa State.*
[2] *Department of Human Physiology, Faculty of Basic Medical Sciences, Niger Delta University, Wilberforce Island, Bayelsa State.*

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Abstract

This study evaluated the anticonvulsant and neuroprotective effects of Virgin *Cocos nucifera* oil (VCNO) using PTZ and MES seizure models in Wistar rats. VCNO was prepared using cold extraction. For PTZ test, twenty-four Wistar rats were divided into control, test 1 (5 ml/kg VCNO), test 2 (10 ml/kg VCNO) and standard (5 mg/kg diazepam) groups. Similarly, twenty-four Swiss mice were used for MES test. Treatments were administered orally for 21 days period and then seizures were induced. Seizure parameters and brain oxidative stress markers (SOD, catalase, GPx, MDA) were evaluated. VCNO (10 ml/kg) significantly prolonged seizure onset (179.65 ± 5.76 s vs. 95.68 ± 3.85 s) and reduced seizure duration (720.65 ± 9.56 s vs. 2800.68 ± 36.06 s) in PTZ model, with reduced mortality. In MES model, VCNO reduced hindlimb tonic extension duration with no mortality. Both doses significantly increased antioxidant enzymes and decreased MDA levels ($p < 0.05$). VCNO exhibits dose-dependent anticonvulsant and neuroprotective effects in seizure models, supporting its traditional use and further investigation as a potential therapeutic agent for epilepsy.

Keywords: *Cocos nucifera*; Epilepsy; Anticonvulsant; Neuroprotection; PTZ; MES; Oxidative stress

1. Introduction

Epilepsy is a chronic neurological condition affecting approximately 50 million people worldwide and is characterized by recurrent unprovoked seizures (Prameswari, 2024; Joffa et al., 2026). Although there are many antiepileptic drugs available, about 30% of patients continue to experience seizures or suffer from significant adverse effects, therefore, it is necessary to find new therapeutic agents with better efficacy and safety profiles (Kanner & Bicchi, 2022).

The maximal electroshock (MES) and pentylenetetrazole (PTZ) seizure models remain the gold standard for preclinical anticonvulsant screening (D'Amora et al., 2023). The MES model identifies drugs effective against generalized tonic-clonic seizures, while the PTZ model detects compounds active against absence and myoclonic seizures (Löscher & White, 2023).

Cocos nucifera, commonly known as coconut, has a long history of use in traditional medicine in tropical areas to treat multiple conditions, including neurological disorders (Kotecha, 2025). Phytochemical studies have identified polyphenolic compounds, flavonoids, and novel sesquineolignans (nuciferols A and B) from *C. nucifera*, with nuciferol B demonstrating significant neuroprotective effects against oxidative stress-induced neuronal death (Sebghatollahi et al., 2025). Previous investigations have reported anticonvulsant activity of methanolic extracts from *C. nucifera* flowers

* Corresponding author: Chukwuma, Samuel Anakwe

through GABAergic mechanisms (Archana et al., 2021). However, the anticonvulsant potential of Virgin *Cocos nucifera* oil (VCNO) remains unexplored.

This study was designed to evaluate the anticonvulsant and neuroprotective effects of Virgin *Cocos nucifera* oil using PTZ and MES seizure models in Wistar rats.

2. Materials and Methods

2.1. Ethical Clearance

The Ethics Committee of the Faculty of Basic Medical Sciences, College of Health Sciences, Niger Delta University Wilberforce Island, Bayelsa State, Nigeria formally approved all experimental methods used in this study.

2.2. Plant Material Collection and Extract Preparation

Matured coconut fruits were obtained from Amassoma market, Southern Ijaw, Bayelsa Nigeria. Virgin coconut oil was prepared using the cold extraction method. Mature coconuts were de-shelled, and the fresh coconut flesh was washed thoroughly with clean water and cut into smaller pieces. The coconut pieces were blended with a measured quantity of distilled water to obtain a smooth paste. The paste was filtered through a clean muslin cloth to extract the coconut milk, and the solid residue was discarded. The extracted coconut milk was transferred into a clean glass container and allowed to stand briefly before being placed in a refrigerator at approximately 4 °C for 30-60 minutes to facilitate phase separation. During refrigeration, the fat-rich cream layer solidified and separated from the aqueous layer beneath it. The hardened cream layer was carefully scooped off and transferred into another sterile container. The cream was gently strained to remove residual curd and moisture, and the separated oil was collected as virgin coconut oil. The oil was stored in airtight amber bottles at room temperature until further use.

2.3. Drug and Chemicals

Pentylenetetrazole (PTZ) (SIGMA-ALDRICH, Inc, 3050, Spruce Street, St. Louis, MO 63103, USA), the agent used to induce seizures and diazepam (Juhel Pharmaceuticals Enugu, Nigeria), the standard anti-seizure drug, were both administered orally. All other chemicals used in the research were of analytical grade.

2.4. Experimental Animals

This study utilized a total of forty-eight animals, comprising twenty-four Swiss mice and twenty-four Wistar male rats. They were obtained from the animal house of Pharmacology Department at Niger Delta University. Before the experiment began, the animals underwent a two-week acclimatization period in the departmental research laboratory. They were housed in standard plastic cages under controlled environmental conditions, which included a 12-hour light/dark cycle, sufficient ventilation, and a temperature range of 20-25 °C. During this acclimatization period, they had free access to standard laboratory chow and clean drinking water.

2.5. PTZ-Induced Seizures Test

Twenty-four healthy male Wistar rats were divided into four separate groups (n=6 per group), each containing six rats. These groups received different treatments over a 21-day period:

- Group 1 (Control): 10 ml/kg distilled water + 70 mg/kg PTZ
- Group 2 (Test 1): 5 ml/kg Virgin *Cocos nucifera* oil + 70 mg/kg PTZ
- Group 3 (Test 2): 10 ml/kg Virgin *Cocos nucifera* oil + 70 mg/kg PTZ
- Group 4 (Standard): 5 mg/kg diazepam + 70 mg/kg PTZ.

On the 21st day, thirty minutes after these treatments, seizures were triggered by administering pentylenetetrazol (PTZ) at 70 mg/kg. The rats were then monitored for the first 30 minutes to record the time until the first convulsion, signs of Straub's tail, the start of myoclonic jerks and whether any deaths occurred.

2.6. MES-Induced Seizure Test

Swiss mice were divided into four groups (n=6 per group) and treated for 21 days:

- Group 1 (Control): 10 ml/kg distilled water
- Group 2 (Test 1): 5 ml/kg Virgin *Cocos nucifera* oil

- Group 3 (Test 2): 10 ml/kg Virgin *Cocos nucifera* oil
- Group 4 (Standard): 5 mg/kg diazepam

On the 21st day, thirty minutes after treatment, seizures were induced using an electroconvulsion therapy unit (Electroconvulsive Therapy Unit, Ugobasile Biological Apparatus, 21025, Comeria, VA, Italy. Model No.: 57800-001) that delivered a brief electrical stimulus (50 mA, 80 Hz for 0.2 seconds) through ear electrodes. To ensure good contact, the electrodes were moistened with saline before being placed on the mice's ears. The current intensity was predetermined in pilot tests to be the level that causes hindlimb tonic extension (HLTE) where the hind legs fully extend without causing death in all control animals. The duration of HLTE was recorded, and full protection was defined as the total absence of this hindlimb extension.

2.7. Antioxidant Assays

The brain tissue samples were sliced and rinsed with a cold 0.1 mMol Sodium Phosphate buffer, Ph 7.4. Following a standard method, a 10% (w/v) homogenate was prepared. This mixture was then centrifuged using a refrigerated centrifuge (4 °C) at 1000x g for 10 minutes to remove cell fragments and nuclei. The clear liquid (supernatant) was used immediately to determine oxidative stress parameters (SOD as described by Misra and Fridovich, 1967; CAT as described by Beutler, 1989; GPx as described by Paglia and Valentine, 1962; MDA as described by Armstrong & Al-Awadi, 1991).

2.8. Statistical Analysis

The data were statistically analysed using one-way ANOVA with Tukey-Kramer post-hoc testing. Results are presented as mean ± SD, with statistical significance set at $p < 0.05$.

3. Results

Table 1 Mean onsets of action and durations of action of PTZ-induced seizures in Wistar rats pretreated with Virgin *Cocos nucifera* oil (VCNO)

Groups	Onset of action(s)	Duration of action (s)	Number of deaths
Control (10 ml/kg Distilled water + 70 mg/kg PTZ)	95.68 ± 3.85^{a}	2800.68 ± 36.06^{a}	6
Test group 1 (5 ml /kg VCNO + 70 mg/kg PTZ)	108.85 ± 4.96^{b}	1780.92 ± 28.44^{b}	3
Test group 2 (10 ml/kg VCNO + 70 mg/kg PTZ)	179.65 ± 5.76^{c}	720.65 ± 9.56^{c}	2
Standard Group (5 mg/kg Diazepam + 70 mg/kg PTZ)	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0

The data are presented as the mean ± standard deviation for a sample size of 6. Different superscripts (a, b, c, d) indicate statistical significance ($p < 0.05$) among means within the same column.

Table 2 Mean duration of hindlimb tonic extension (HLTE) of MES-induced seizures in Swiss mice pretreated with Virgin *Cocos nucifera* oil (VCNO)

Groups	30 min	90 min	120 min	Number of deaths
Control (10 ml/kg Distilled water)	44.03 ± 3.83^{a}	46.31 ± 2.74^{a}	48.58 ± 4.02^{a}	5
Test group 1 (5 ml/kg VCNO)	25.38 ± 3.18^{b}	22.97 ± 1.85^{b}	36.39 ± 4.12^{b}	2
Test group 2 (10 ml/kg VCNO)	14.66 ± 2.14^{c}	10.01 ± 0.54^{c}	10.25 ± 1.20^{c}	0
Standard Group (5 mg/kg Diazepam)	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0.00 ± 0.00^{d}	0

The data are presented as the mean ± standard deviation for a sample size of 6. Different superscripts (a, b, c, d) indicate statistical significance ($p < 0.05$) among means within the same column (for each parameter).

Table 3 Mean levels of oxidative stress biomarkers in PTZ-induced seizure Wistar rats pretreated with Virgin *Cocos nucifera* oil (VCNO)

Groups	SOD (U/mg Protein)	Catalase (U/mg Protein)	GPx (U/mg protein)	MDA (U/mg Protein)
Control (10 ml/kg Distilled water)	3.28 ± 0.43[a]	2.11 ± 0.56[a]	2.10 ± 0.43[a]	6.22 ± 0.65[a]
Test group 1 (5 ml/kg VCNO)	8.44 ± 1.74[b]	5.79 ± 0.38[b]	6.94 ± 0.72[b]	3.03 ± 0.46[b]
Test group 2 (10 ml/kg VCNO)	12.72 ± 1.58[c]	8.11 ± 0.79[c]	9.44± 0.75[c]	1.58 ± 0.21[c]
Standard Group (5mg/kg Diazepam)	12.96 ± 2.0[c]	9.10± 0.86[c]	10.86 ± 1.11[c]	1.12 ± 0.26[d]

The data are presented as the mean ± standard deviation for a sample size of 6. Different superscripts (a, b, c, d) indicate statistical significance ($p < 0.05$) among means within the same column (for each parameter).

Table 4 Mean levels of oxidative stress biomarkers in MES-induced seizure Swiss mice pretreated with Virgin *Cocos nucifera* oil (VCNO)

Groups	SOD (U/mg Protein)	Catalase (U/mg Protein)	GPx (U/mg Protein)	MDA (U/mg Protein)
Control (10 ml/kg Distilled water)	3.11 ± 0.38[a]	2.22 ± 0.45[a]	1.98 ± 0.08[a]	5.94 ± 0.05[a]
Test group 1 (5 ml/kg VCNO)	5.88 ± 0.64[b]	4.65 ± 0.17[b]	5.12 ± 0.12[b]	2.18 ± 0.37[b]
Test group 2 (10 ml/kg VCNO)	9.45 ± 1.16[c]	7.34 ± 1.24[c]	8.06 ± 1.05[c]	1.06 ± 0.02[c]
Standard Group (5 mg/kg Diazepam)	11.11 ± 1.23[d]	9.45 ± 1.58[d]	12.18 ± 2.10[d]	0.54 ± 0.04[d]

The data are presented as the mean ± standard deviation for a sample size of 6. Different superscripts (a, b, c, d) indicate statistical significance ($p < 0.05$) among means within the same column (for each parameter).

4. Discussion

The present study shows that Virgin *Cocos nucifera* oil (VCNO) has a significant, dose-dependent anticonvulsant activity in both PTZ and MES seizure models. In the PTZ model, VCNO (10 ml/kg) prolonged seizure latency from 95.68 ± 3.85 seconds to 179.65 ± 5.76 seconds and reduced seizure duration from 2800.68 ± 36.06 seconds to 720.65 ± 9.56 seconds, with decreased mortality. In the MES model, the same dose (10 ml/kg) reduced hindlimb tonic extension duration across all time points evaluated, with no mortality recorded.

The anticonvulsant effects observed in the PTZ model suggest GABAergic action, where PTZ is a *GABA_A* receptor antagonist (Joffa et al., 2026). This aligns with previous reports of GABA-activated anticonvulsant effects of *C. nucifera* flower extracts (Grace & Monisha, 2022). Efficacy in the MES model further suggests that it could be useful in generalized tonic-clonic seizures perhaps by modulating sodium channels.

It is worth noting that VCNO significantly improved brain antioxidant enzymes (SOD, catalase, GPx) and decreased MDA levels in both models. At 10 ml/kg, SOD activity increased approximately 4-fold (from 3.28 ± 0.43 to 12.72 ± 1.58 U/mg protein) compared to controls in the PTZ model, with similar effects observed in the MES model (from 3.11 ± 0.38 to 9.45 ± 1.16 U/mg protein). These antioxidant properties likely play a role in neuroprotection against seizure-induced oxidative damage and are consistent with the presence of polyphenolic compounds and novel sesquineolignans recently identified in C. nucifera (Firdous et al., 2025; Joffa et al., 2026).

The dose-dependent nature of these effects and their comparability to diazepam-treated groups suggest that VCNO bioactive constituents have therapeutic potential with possible benefits over synthetic anticonvulsants in terms of adverse effect profiles.

5. Conclusion

Virgin *Cocos nucifera* oil has considerable dose-dependent anticonvulsant and neuroprotective properties in PTZ and MES seizure models, indicated by increased delay in seizure manifestation, shorter seizure, lowered mortality and improved antioxidant activities of brain enzymes with lower lipid peroxidation.

These findings support its traditional use in neurological conditions and the need to explore further the bioactive components and potential of the compound in treatment.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

The Research and Ethics committee of the Faculty of Basic Medical Sciences, College of Health Sciences, Niger Delta University gave approval for this study.

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