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Effects of Virgin Coconut Oil on Aluminium Chloride-Induced Alzheimer-Like Dementia in the Prefrontal Cortex

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**Authors’ contributions**

This work was carried out in collaboration between all authors. Authors OBA and JAO designed the study. Authors JAO and UEU performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors JOO, SYO and OSF managed the analyses of the study. Author ABOD managed the literature searches. All authors read and approved the final manuscript.

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**ABSTRACT**

**Aims:** The aim of this investigation is to study the effect of virgin coconut oil on the prefrontal cortex upon aluminium chloride-induced Alzheimer-likes dementia.

**Study Design and Methodology:** Twenty-eight (28) adult male Wistar rats were used and were randomly assigned into four groups:

- **Group A:** Considered to be the control group which took water and food daily.
- **Group B:** Virgin coconut oil treated group, were administered virgin coconut oil orally with 1700 mg/kg BW for 42 days.
- **Group C:** Aluminium chloride treated group, were administered Aluminium chloride orally with 200 mg/kg BW for 42 days.
- **Group D:** Aluminium chloride + virgin coconut oil treated group, the Wistar rats were administered

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Aluminium chloride orally with 200 mg/BW for 21 days and then virgin coconut oil orally with 1700 mg/BW for the next 21 days. The neurobehavioural investigation was done after the experiment to evaluate learning and memory using Barnes Maze. Animals were sacrificed by cervical dislocation. The whole brain was carefully excised from the skull. Prefrontal cortex was removed and fixed in 10% formal saline for histological analysis which includes H&E stain for general histoarchitecture and Cresyl fast violet for Nissl substance.

**Place and Duration of Study:** The study was done in Babcock University and approved by the Babcock University Ethics commission (BUHREC) with the BUHREC number 672/16. The experiment took the total of 45 days.

**Results:** Neurobehavioral study revealed a significant high latency in the animals treated with Aluminium chloride when compared with control and other treated groups. H &E technique, as well as Cresyl fast violet, did not show any observable altered morphological presentation in the control and VCO treated groups. Also, a cellular density between the control and virgin coconut oil group appears normal across the cortical layers with appreciable spines and neuronal projections. The Aluminium chloride treated group induced degenerative changes in the cortex and was characterized by fragmented pyramidal and granule cell layer with observable pyknotic cells. Also, there was a comparatively increased cell density in the cortical layers of the VCO and Aluminium chloride + VCO treated groups.

**Conclusion:** Findings from this study support the view that, virgin coconut oil has ameliorative effects which neutralize the adverse effect of Aluminum chloride and tried to restore the integrity of the prefrontal cortex.

**Keywords:** Prefrontal cortex; Alzheimer; dementia; virgin coconut oil; aluminium chloride.

## 1. INTRODUCTION

### 1.1 Background of Study

Alzheimer’s disease (AD) is a slow, progressive and fatal neurodegenerative disease [1]. The neuropathological hallmarks of Alzheimer’s disease include deposition of extracellular senile plaques containing aggregates of the amyloid β protein (Aβ), intracellular neurofibrillary tangles (NFT) composed of hyper-phosphorylated tau protein, and a massive neuronal loss [2,3]. Although the cause of AD remains poorly understood multiple factors are reported to influence AD onset. The primary among these is the cause of mutations in the Aβ precursor (APP) and presenilins 1 and 2 (PS1 & PS2) that lead to increasing in the production of the 42-residue of Aβ (Aβ42). The E4 allele of apolipoprotein E is the most prevalent risk factor in addition to levels of cholesterol, homocysteine and several minor metal ions such as Al, Cu, Fe are linked to the AD. The contributions of neurotoxicity of Aluminium in experimental animals were first reported in 1897 by Dollken [4].

Alzheimer’s disease is recognized as the major cause of dementia in modern society and may result in significant limitations for sufferers through cognitive impairment. Dementia is a clinical syndrome that involves progressive deterioration of intellectual abilities. Various cognitive functions can be impaired with dementia, including memory, language, reasoning, decision making, visuospatial function, attention, and orientation [5]. There are several and irreversible causes of dementia [5,6]. Reversible dementias are relatively rare but potentially treatable and occur in secondary to another medical condition, including depression, nutritional deficiencies, metabolic and endocrine disorders, space-occupying lesions, normal pressure hydrocephalus, or substance abuse the most evident symptom in the early AD is the memory loss [7]. Alzheimer’s disease is characterized by atrophy of cerebral cortex and hippocampal brain tissue.

Alzheimer’s disease (AD) is the most common form of dementia in the ageing population [8]. It is a deliberating neurodegenerative disorder characterized by the progressive loss of cholinergic neurons, leading to the onset of severe behavioural, motor and cognitive impairments [9]. In 2015, there were approximately 48 million people worldwide with the AD [10]. The most often begins in people over 65 years of age, although 4% to 5% of cases show early-onset Alzheimer’s which begins before this [11]. Three genes causative for the early on-set familial AD (FAD) has been identified. These are the amyloid precursor...
1.3 Prefrontal Cortex

The prefrontal cortex is the cerebral cortex which covers the front part of the frontal lobe. The prefrontal cortex contains Brodmann areas 9, 10,11,12,46,47 [17]. This brain region has been implicated in planning, complex cognitive behaviour, personality expression, decision making, and moderating social behaviour [18]. The basic activity of this brain region is considered to be an orchestration of thoughts and actions in accordance with internal goals [19]. The most typical psychological term for functions to carry out by the prefrontal cortex area is the executive function. Executive function relates abilities to differentiate among conflicting thoughts, determine good and bad, better and best, same and different, future consequences of current activities, working towards a defined goal, prediction of outcomes, expectation based on actions, and social control [20].

1.4 Virgin Coconut Oil

Coconut oil typically comes from the coconut white “meat” called copra, coconut milk, or coconut milk residue are processed by drying, wet-squeezing or centrifugation to separate out the oil [21,22,23,24]. When coconut oil is separated into different components, some of the main saturated fats are the medium-chain fatty acids, caprylic acid and lauric acid. Nonhydrogenated virgin coconut oil is considered healthier than the hydrogenated version which has the unhealthy trans-fatty acids [25,26,27]. Medium-chain fatty acids that move quickly to the liver with most medium-chain fatty acids quickly supplying energy to the body and a small amount of medium-chain fatty acids which are becoming stored fat in the body. Virgin coconut oil and extra virgin coconut oil use fresh coconut oil, which goes through a wet-squeeze process, that differs from heated drying process [28]. The wet-squeeze process maintains or increases the natural vitamin and polyphenol content available in the final product [29].

1.5 Phytochemicals in Virgin Coconut Oil

Virgin coconut oil has a high percentage of phytochemicals sometimes also referred to polyphenols. Phenolic acids are recognized for their antioxidant properties. P-coumaric acid, ferulic acid, caffeic acid and catechin acid are the major phenolic acids found in coconut oil. The hydroxyl group of phenolic acid compounds may be able to reduce the toxicity of Alzheimer’s amyloid beta (Aβ) peptide. In vitro studies which have investigated flavonoids that indicate the hydroxyl groups which could trap hydrogen bonds of Aβ, which is important as this may reduce Aβ aggregation. Phenolic compounds can bind Aβ fibrils with their long axis parallel to the long axis of Aβ fibrils [30].

1.6 Virgin Coconut Oil and Alzheimer’s Disease

The attention to coconut oil may stem initially from the budding interest in medium chain triglycerides (MCT) which break down into ketones “food” and appear to increase cognitive function [31,32,33]. Medium-chain triglycerides are medium chain fatty acids that are digested more easily than other fats. Found in coconut oil, MCT converts readily in the liver into energy for the body [27]. Brain cells depend upon mitochondria which acts tiny power plants to transform glucose into energy for cell activities. During the decline of Alzheimer’s disease,
cerebral glucose metabolism seems to suffer as mitochondria become less able to absorb and use glucose [34]. Acting as a glucose substitute, ketones from the breakdown of MCT [27] which contains medium chain fatty acids may appear to offer the necessary simple fuel for the dysfunctioning mitochondria in the brain cells to use for energy [21] in a carefully controlled study found that high levels of a ketone food such as beta-hydroxybutyrate helped people with probable Alzheimer's disease function significantly better on paragraph recall.

1.7 Aluminium Chloride (ALCL₃)

Aluminium chloride is one of the most abundant metals in the earth's crust. Human exposure to Al has been increasing over the decades. This element appears mainly in food products and in drinking water derived from both food products and in drinking water derived from both natural sources and treatment methods. Aluminium (Al) is a trivalent cation found in its ionic form in most kinds of animal and plant tissue and in natural waters everywhere. The element aluminium does not occur in its pure state but is always combined with another element such as chloride, hydroxide, silicate, sulphate and phosphate. The wide distribution of this element ensures the potential for causing human exposure and harm [35]. Aluminium is a neurotoxicant without any useful biological function that more readily enters the brain as the age rises.

1.8 Aluminium Chloride and Alzheimer's Disease

Many scientific studies have brought to light the potential toxicity of Aluminium in experimental animals was initially and in humans under different clinical conditions. The pioneering studies on Al neurotoxicity in experimental animals were initially described [4]. Al as a neurotoxin metal was initially established in the early 1970s after years of uncertainty. The Al hypothesis in AD came to light following the extraordinary discovery of Klatzo et al. [13], who showed that injections of Al salts into rabbit brain led to the formation of Neurofibrillary tangles of the AD [13,36].

Al³⁺ has positive charges and a relatively small ionic radius in comparison to other metal ions such as Ca²⁺, Zn²⁺, Na⁺. Thus, Al³⁺ firmly binds to metal-binding amino acids or phosphorylated amino acids and acts as a cross linker. Al can cause oligomerization of proteins, inducing conformational changes that can inhibit their degradation by proteases. Strong binding of Al³⁺ to phosphorylated amino acids promotes self-aggregation and accumulation of highly phosphorylated cytoskeleton proteins, including neurofilament and microtubule associated proteins (MAPs). Consequently, Al causes apoptotic death of neurons and glial cell. Chronic administration of Al impairs long-term potentiation, which is a form of synaptic information storage well known as a paradigm of memory mechanisms. Al also impairs various enzymes including those related to neurotransmitter synthesis and thus affects neurotransmitter content. Al³⁺ also inhibits voltage-gated ions Ca²⁺ channels and neurotransmitters receptor, and impairs synaptic transmission. Al causes spatial memory deficit, influences emotional reactivity, and impairs various brain function related to learning and memory [37].

1.9 Mechanism of Action of Aluminium Chloride

Aluminium exhibits only one oxidation state, Al³⁺. Al³⁺ has an affinity for negatively charged, oxygen-donor ligands. Inorganic and organic phosphates, carboxylate and deprotonated hydroxyl groups form strong bonds with Al³⁺. Owing to these chemical characteristics, Al³⁺ binds to the phosphate groups of DNA topology and influencing the expression of various genes essential for brain functions. Lukiw et al. reported that nanomolar levels of Al³⁺ were sufficient to influence neuronal expressions [38].

Al³⁺ also binds to the phosphate groups of nucleoside di- and triphosphates, such as ATP and can thus influence energy metabolism. Furthermore, Al exhibits the functions of various protein kinases and phosphatases. Al can cause the oligomerization of proteins, including conformational changes that can inhibit their degradation by proteases. Strong binding of Al³⁺ to phosphorylated amino acids promotes the self-aggregation and accumulation of highly cytoskeleton proteins, including neuro-filament and microtubule-associated protein and so forth [37].

Consequently, Al causes apoptotic death of neurons and glial cells. Chronic administration of Al impairs long-term potentiation (LTP), which is a form of synaptic information storage well known as a paradigm of memory mechanisms. Al also impairs various enzymes including those related to neurotransmitter synthesis and thus
affects the neurotransmitter content. \( \text{Al}^{3+} \) also inhibits voltage-gated \( \text{Ca}^{2+} \) channels and neurotransmitter receptors, and thus impairs various brain functions related to learning and memory [39].

2. MATERIALS AND METHODS

2.1 Experimental Animals Procurement, Housing and Handling

Twenty-eight (28) male adult Wistar rats (\textit{Rattus norvegicus}) at the weight of 120-150 g were procured from the animal house, Babcock University were used to conduct this research work. The rats were aged 7 weeks at the time of purchase. The animals were kept in plastic cages with net covers for adequate ventilation, under standard conditions (25-29°C, 12 hours light and 12 hours’ dark cycles). They were fed with pelleted diet and water were given \textit{ad libitum}. The Wistar rats were left to acclimatize in the environment for seven days before the research commenced. The study was approved by the Babcock University Ethics commission (BUHREC) with the BUHREC number 672/16.

2.2 Procurement of Aluminium Chloride and Administration

The aluminium chloride was procured from a chemical store in Ogun State, Nigeria. 17 g of aluminium chloride was dissolved in 450 ml of distilled water.

2.3 Procurement of Virgin Coconut Oil and Administration

The coconuts were bought from Ilishan market, Ogun State, Nigeria. The VCO was extracted using a modified wet extraction method described by Nevin and Rajamohan [29,23].

a) The VCO was extracted using a modified wet extraction method described by Nevin and Rajamohan [29,23].

b) The solid endosperm of mature coconut was crushed and made into a viscous slurry.

c) About 500 ml of water was added to the slurry obtained and squeezed through a fine sieve to obtain coconut milk.

d) The resultant coconut milk will be left for about 24 hours to facilitate the gravitational separation of the emulsion as previously described by Onsaard et al. [40] and Nour et al. [41].

e) Demulsification produced layers of an aqueous phase (water) on the bottom, an emulsion phase (cream) in the middle layer and an oil phase on top is described by Nour et al. [41].

f) The oil on top will be viscous and heated for about 5 minutes to remove moisture.

The obtained VCO was then filtered through a fine sieve, stored at room temperature and used for the experiment.

2.4 Experimental Design

Twenty-eight (28) adult male Wistar rats were used for this experiment University. They were randomly assigned into five groups: A, B, C, D of 7 rats in each group to avoid overcrowding.

Group A: Serve as the control group, the Wistar rats were given water and food daily.

Group B: Virgin coconut oil treated group, the Wistar rats in this group were fed daily and administered virgin coconut oil orally with 200 mg/kg BW for 42 days.

Group C: Aluminium chloride treated group, the Wistar rats were fed daily and given water and administered aluminium chloride orally with 200 mg/kg BW for 42 days.

Group D: Aluminium chloride + virgin coconut oil treated group, the Wistar rats were fed daily and given water daily and administered aluminium chloride orally with 200 mg/kg BW for 21 days and then virgin coconut oil orally with 200 mg/BW for the next 21 days.

2.5 Neurobehavioural Analysis

The neurobehavioural investigation was done after the experiment to evaluate learning and memory using Barnes Maze.

2.6 Barnes Maze

The Barnes maze is a tool used in psychological laboratory experiments to measure spatial learning and memory. The test was first developed by Dr Carol Barnes in 1979 [42]. The basic function of Barnes Maze is to measure the ability of a rat to learn and remember the location of a target zone using a configuration of distal visual cues located around the testing area [42]. This noninvasive task is useful for evaluating novel chemical entities for their effects on cognition as well as identifying cognitive deficits in a transgenic strain of rodents that model for the disease such as Alzheimer’s disease.
Table 1. Illustrating the grouping of animals

<table>
<thead>
<tr>
<th>S/N</th>
<th>Group</th>
<th>Treatments</th>
<th>Dosages [mg/kg]</th>
<th>Rationale</th>
<th>Duration [days]</th>
<th>No. of animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>A Control [CO]</td>
<td>Distilled Water and pelleted feed</td>
<td>Ad libitum</td>
<td>Placebo</td>
<td>42</td>
<td>7</td>
</tr>
<tr>
<td>2.</td>
<td>Experimental B [VCO]</td>
<td>Virgin coconut oil</td>
<td>1700 (5000g/Kg BW)</td>
<td>Positive Control</td>
<td>42</td>
<td>7</td>
</tr>
<tr>
<td>3.</td>
<td>Experimental C [AlCl₃]</td>
<td>Aluminium Chloride only</td>
<td>200 (LD50 3.32g/kg BW)</td>
<td>Negative Control-to induce damage</td>
<td>42</td>
<td>7</td>
</tr>
<tr>
<td>4.</td>
<td>Experimental D [AlCl₃+VCO]</td>
<td>Aluminium chloride then virgin coconut oil</td>
<td>200 1700</td>
<td>Therapeutic effect</td>
<td>21 then 21</td>
<td>7</td>
</tr>
</tbody>
</table>

The Barnes maze consists of a circular surface with up to 20 circular holes around its circumference. Visual cues, such as coloured shapes or patterns, are placed around the table in plain sight of the animal. The table surface is brightly lit by overhead lighting under one of the holes is an escape box which can be reached by the rodent through the corresponding hole on the table top. The model is based on the rodents’ aversion of open spaces, which motivates the test subject to seek shelter in the escape box. A normal rodent will learn to find the escape box within four to five trials and will head directly towards the escape box without attempting to escape via incorrect holes. Various parameters are measured including latency to escape, path length, number of errors, and velocity [42]. The procedure for Barnes Maze is as follows:

The animals were made to interact with the Barnes maze in three phases: the habituation phase, training phase and probe day trial.

Before the experiment began, the animals were made to acclimatize to the room where they were being tested for an hour. The maze was cleaned using 70% ethanol to avoid olfactory cues. Then all the rats (n = 3) from a cage were placed in holding cages (to control for potential artefacts that could occur as a result of housing some animals only two per cage) where they were till the end of the testing sessions.

When testing sessions were completed, the animals were returned to their group cages, the holding cages were cleaned.

2.7 Habituation Day

Three animals were selected per group and they were each placed in a transparent 3,500 ml beaker in the middle of the maze static for 15 seconds after which the beaker was lifted and a buzzer was turned on the scare the rat and it was cautiously led into the target hole. The rat was not forced into the target hole so as to avoid stress but they were tugged in lightly. When it got into the target hole, the hole was covered with the rat inside and then the rat was allowed to settle for about 2 minutes before repeating the procedure and turning off the buzzer. This procedure was repeated 4 times and then the rats were allowed to enter into the target hole into the escape cage independently and if they did not, they were nudged in with the beaker. Making the rats enter the escape cage was important so the animals were made to be aware that there is an escaped cage. After habituation, the animals returned to their group cages and the maze was cleaned with alcohol and cotton wool.

2.8 Training Day

In this phase of interaction, in the centre of the maze, the animals were placed in a 10 “tall opaque cylinder which was 7” in diameter for a period of 15seconds which allowed the animals face random directions when the cylinder was removed and then the buzzer was turned on and examination began. The animals were allowed to explore the maze within 3 minutes and if the animals did not enter into the escape cage it was nudged until it entered. After nudging for one minute, and the rat did not enter the escape cage, it was manually put on the platform in the escape cage and then it was allowed to be inside the escape cage for one minute and then it was returned to the holding cage. During the training phase, the total latency was recorded.

2.9 Probe Day

Probe trial is done to determine the memory taught from the previous training days. On the 5th day, 24 hours after the last training day, the maze was placed just as it was during the training phase and the probe trial was conducted. The animal was covered with the cylinder and after 10seconds the cylinder was lifted and the buzzer was switched allowing the animal to
explore the maze. After a fixed interval of 90 seconds, the rat was removed. The latency was measured.

2.10 Sacrifice of Experimental Animals

Immediately after behavioural analysis, animals were sacrificed by cervical dislocation. The whole brain was carefully excised from the skull. Prefrontal cortex was removed and fixed in 10% formal saline for histological activities. This procedure for histological demonstration are:

1. Haematoxylin and Eosin for cyto-architecture organization.
2. Cresyl Fast Violet for Nissl substance.

3. RESULTS

3.1 Body Weight

As shown in Fig. 1, at the end of the experiment the final body weight of group A (control) was 152.4±2.112. There was no significant difference in final body weight of group B (VCO) (138.8±4.652) when compared to group A (control) but group B was slightly lower than group A (control). The final body weight of group C (AlCl₃) (111.8±6.822) was significantly (P < 0.05) lower than group A (control) (152.4±2.112). The final body weight of group D (AlCl₃+VCO) (132±3.564) was significantly (P < 0.05) higher when compared to group A (control) (152±2.112).

![Fig. 1. Bar chart showing the final body weight of control and treated groups after 42 days](image1)

![Fig. 2. Values are mean ± SEM of data obtained; * = significantly different from group A (control), b = significantly different from group B (VCO), β= significantly different from group C (AlCl₃), α = significantly different from group D (AlCl₃ + VCO), P< 0.05](image2)
3.2 Neurobehavioural Study

3.2.1 Barnes maze

3.2.1.1 Mean latency

In Fig. 2 the mean latency for group A (control) was 27.20±0.8602. The mean latency for group B (VCO) (14.20 ±1.1.58) was significantly (P<0.05) lower than group A (control). The mean latency Group C (AlCl₃) was significantly higher than group A (control) (27.20±0.8602). There was no significant difference when group D (AlCl₃+VCO) (28.60±0.7483) was compared to group A (control) (27.20±0.8602).

3.3 Histological Results

Plate 1: Photomicrographs of different groups stained with H&E.

Plate 2: Photomicrographs of all the groups stained with Cresyl fast violet.

Plate 1. Photomicrographs showing panoramic views of prefrontal cortex general histomorphological presentations in Wistar rats across the study groups. Hematoxylin and Eosin stain (X400). The Control and VCO groups (Group A & B) present normal neurons and glia cell without any mechanical assault (Black arrows). Group C (AlCl₃) present neuronal degeneration or assault as represented by Pyknosis (Yellow arrow) and Vacuolation (Green arrow) Group D (AlCl₃ + VCO) present some normal neuron and glia cells (Black arrows) as well as abnormal or assaulted neurons and glia cells (Red yellow arrows).
Plate 2. Photomicrographs showing Nissl profiles of prefrontal cortex general histomorphological presentations in Wistar rats across the study groups. Cresyl fast violet stains (X100). The Control and VCO group (Group A&B) presented normal nissl profiles (Red arrows). Group C (group ALCL₃) showed a serious loss of nissl substance (Black arrows) while Group D (ALCL₃ + VCO) showed moderate or fewer loss of nissl substance (Yellow arrow) as well as normal nissl substance (Red arrow).

4. DISCUSSION AND CONCLUSION

Alzheimer's disease is a neurodegenerative disorder characterized by a progressive loss of memory and cognition [43]. Despite more than a century of research and the massive information on the AD, the causes and an early and accurate diagnosis and treatment remain elusive [44]. In AD there is widespread degeneration of neurons in many brain regions including frontal cortices.

The results obtained from the morphological analysis revealed that there was no significant difference in the final body weight of the control group when compared to the VCO group although there was a slight increase in the final body weight in the control group when compared to the VCO group this finding is in accordance with research of Mohammed et al. 2013, they stated that consumption of virgin coconut oil did not lead to abnormal weight despite high saturated fatty acid content also Takeuchi et al. [45] stated that the saturated fatty acids in VCO consist of medium chain fatty acids (MCFA) that are made up 8-10 carbons. MCFA is easily digested and absorbed and is directly absorbed into the circulation and delivered to the liver, where it is metabolized faster into energy, thus it prevents body-fat accumulation. There was a significant decrease in weight of aluminium chloride treated group when compared to all other groups the decrease in weight is in accordance with the research carried out by Adekunle [46] in which there was a reported decrease in weight of the Wistar rats administered aluminium chloride when compared to the control group.

The result from the Barnes maze study revealed a significant high latency in the animals treated with aluminium chloride when compared with control and other treated groups this finding supports a study done by Ouafa et al., 2008 which demonstrated a significant impairment in spatial learning and memory in AlCl₃ treated animals.

The general cytoarchitecture of the prefrontal cortex in the Wistar rats as demonstrated using H &E technique did not show any observable altered morphological presentation in the control and VCO treated groups as shown in Plate 2. Also, a cellular density between the control and virgin coconut oil group appears normal across the cortical layers with appreciable spines and neuronal projections. The aluminium chloride treated group induced degenerative changes in the cortex and was characterized by fragmentation pyramidal and granule cell layer with observable pyknotic cells. Also, there was a comparatively increased cell density in the cortical layers of the VCO and Aluminium chloride + VCO treated groups.

The Nissl profile demonstration by CFV stain (X100) across PFC sections within the study groups shows normal morphological presentations in the control and VCO treatment groups that are characterized with normal and densely populated Nissl proteins, well stained
and outlined neurons. Aluminium chloride treated groups showed caused severe chromatolytic changes as well as some pyknotic changes in both the pyramidal and granule cell layers with a gross reduction in the cytoplasmic Nissl proteins.

It could, therefore, be concluded that virgin coconut oil has protected and ameliorative effects that are capable of preventing or neutralizes the adverse effects of Aluminium chloride effect on prefrontal cortex histoarchitecture as well as able to restore the integrity of the prefrontal cortex after it has been assaulted.

CONSENT

It is not applicable.

ETHICAL APPROVAL

As per international standard or university standard, written approval of Ethics committee has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES


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