

Original Research Article

EFFECTS OF VIRGIN COCONUT OIL ON ALUMINIUM CHLORIDE-INDUCED ALZHEIMER-LIKE DEMENTIA IN THE PREFRONTAL CORTEX

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: Control group 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 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.

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 45days.

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 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 by neutralizes the adverse of Aluminum chloride effect and tried to restore the integrity of the prefrontal cortex.

KEY WORDS: Prefrontal cortex, Alzheimer- likes dementia, Virgin Coconut Oil, Aluminium Chloride.

INTRODUCTION

BACKGROUND OF STUDY

Alzheimer's disease (AD) is a slow, progressive and fatal neurodegenerative disease⁽¹⁾. The neuropathological hallmarks of Alzheimer's disease include deposits of extracellular senile plaques containing aggregates of the amyloid β protein(A β), intracellular neurofibrillary tangles(NFT) composed of hyperphosphorylated 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, are mutations in the A β precursor(APP) and presenilins 1 and 2(PS1 &PS2) that lead to increase in the production of the 42-residue A β (A β 42). The E4 allele of apolipo-protein 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 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 functions. Various cognitive abilities 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 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 early AD is 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 aging population⁽⁸⁾. It is a deliberating neurodegenerative disorder characterized by the progressive loss of cholinergic neurons, leading to the onset of severe behavioral, motor and cognitive impairments⁽⁹⁾. In 2015, there were approximately 48 million people worldwide with AD⁽¹⁰⁾. It most often begins in people over 65 years of age, although 4% to 5% of cases are early-onset Alzheimer's which begins before this⁽¹¹⁾. Three genes causative for the early on-set familial AD (FAD) have been identified. These are the amyloid precursor protein (APP) gene on chromosome 21 and the presenilin 1(PS1) and presenilin 2 (PS2) genes on respectively chromosomes 14 and 1. Inheritance of the ϵ 4-allele of the apolipoprotein E (ApoE) gene has been recognized as a strong genetic risk factor for the late onset of the disease⁽²⁾.

ALUMINIUM CHLORIDE AND ALZHEIMER'S DISEASE

Aluminium is a well-established neurotoxin and is suspected to be linked with various neurodegenerative diseases including Alzheimer's disease⁽¹²⁾. The hypothesis that Aluminium is an environmental contributor to the pathogenesis of AD termed the "aluminium hypothesis", was proposed in the 1960s based on various neurotoxicological, analytical, and epidemiological findings^{(13),(14),(15)}.

The brain survives on glucose, a form of sugar which is the primary source of fuel for brain cells. In Alzheimer's disease, the brain starts to have problems absorbing glucose. Brain cells that don't get enough glucose wither and die, resulting in poor concentration and memory. Coconut oil contains a high percentage of a molecule called medium chain triglyceride (MCT). These MCT molecules are converted

to ketones in the liver. Ketones cross the blood-brain barrier and act as an alternative food for brain cells. Having sufficient fuel improves brain cell health, thereby reducing or even reversing the symptoms of Alzheimer's dementia ⁽¹⁶⁾.

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 behavior, personality expression, decision making, and moderating social behavior ⁽¹⁸⁾. The basic activity of this brain region is considered to be orchestration of thoughts and actions in accordance with internal goals ⁽¹⁹⁾. The most typical psychological term for functions carried out by the prefrontal cortex area is executive function. Executive function relates to 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⁾.

VIRGIN COCONUT OIL

Coconut oil typically comes from the coconut white "meat" called copra, coconut milk, or coconut milk residue. Processing by drying, wet-squeeze, or centrifuge separates 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 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 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⁾.

PHYTOCHEMICALS OF VIRGIN COCONUT OIL

Virgin coconut oil has a high percentage of phytochemicals sometimes also referred to as 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 toxicity of Alzheimer's amyloid beta (A β) peptide. In vitro studies which have investigated flavonoids indicate that the hydroxyl groups could trap hydrogen bonds of A β , which is important as this may reduce A β aggregation. Phenolic compounds can bind A β fibrils with their long axis parallel to the long axis of A β fibrils ⁽³⁰⁾.

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 convert readily in the liver into energy for the body ⁽²⁷⁾. Brain cells depend upon mitochondria which acts as 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.

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 other 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.

ALUMINIUM CHLORIDE AND ALZHEIMER'S DISEASE

Many scientific studies have brought to light the potential toxicity of Aluminium in experimental animals were 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 AD ^{(13),(36)}.

Al³⁺ has positive charges and a relatively small ionic radius in comparism 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 elf aggregation and accumulation of highly phosphorylated cytoskeleton proteins, including neurofilament and micro-tubule 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⁾.

MECHANISM OF ACTION OF ALUMINIUM CHLORIDE

Aluminium exhibits only one oxidation state, Al³⁺. Al³⁺ has 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

164 self-aggregation and accumulation of highly cytoskeleton proteins, including neurofilamen and
165 microtubule- associated protein and so forth ⁽³⁷⁾.

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167 long-term potentiation (LTP), which is a form of synaptic information storage well known as a paradigm of
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169 synthesis and thus affects the neurotransmitter content. Al³⁺ also inhibits voltage-gated Ca²⁺ channels
170 and neurotransmitter receptors, and thus impairs various brain functions related to learning and memory.

171 MATERIAL AND METHOD

172 EXPERIMENTAL ANIMALS PROCUMENT, HOUSING AND HANDLING.

173 Twenty- eight (28) male adult wistar rats (*Rattus novegicus*) at the weight of 120-150g were procured
174 from the animal house, Babcock University were used to conduct this research work. The rats were aged
175 7 weeks at the time of purchase. The animals were kept in plastic cages with net covers for adequate
176 ventilation, under standard conditions (25-29⁰c, 12hours light and 12 hours' dark cycles). They were feed
177 with pelleted diet and water given *ad libitum*. The wistar rats were left to acclimatize in the environment
178 for seven days before the research commenced. The study was approved by the Babcock University
179 Ethics commission (BUHREC) with the BUHREC number 672/16.

180 PROCUREMENT OF ALUMINIUM CHLORIDE AND ADMINISTRATION

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

183 PROCUREMENT OF VIRGIN COCONUT OIL AND AMINISTRATION

184 The coconuts were bought from Ilishan market, Ogun State, Nigeria.

185 a) The VCO was extracted using a modified wet extraction method described by Nevin and
186 Rajamohan (2004,2009).^{(29),(23)}

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

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

190 d) The resultant coconut milk will be left for about 24 hours to facilitate the gravitational separation of
191 the emulsion as previously described by (Onsaard *et al.*, 2005) and (Nour *et al.*, 2009).

192 e) Demulsification produced layers of an aqueous phase (water) on the bottom, an emulsion phase
193 (cream) in the middle layer and an oil phase on top as described by (Nour *et al.*, 2009).

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

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

197 EXPERIMENTAL DESIGN

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

200 GROUP A: Serve as the control group, the wistar rats were given water and food daily.

201

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

204

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

207

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

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212 TABLE 1: ILLUSTRATING THE GROUPING OF ANIMALS

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

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215 NEUROBEHAVIOURAL ANALYSIS

216 Neurobehavioural investigation was done after the experiment to evaluate learning and memory using
217 Barnes Maze.

218 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⁽⁴¹⁾. 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 transgenic strain of rodents that model for the disease such as Alzheimer's disease .

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 Maize 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 cages together, the holding cages were cleaned.

HABITUATION DAY

Three animals were selected per group and they were each placed in a transparent 3,500ml beaker in the middle of the maze static for 15seconds after which the beaker was lifted and a buzzer was turned on to 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 escape cage. After habituation the animals returned to their group cages and the maze was cleaned with alcohol and cotton wool.

TRAINING DAY

In this phase of interaction, in the center 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.

262 **PROBE DAY**

263 Probe trial is done to determine the memory taught from the previous training days. On the 5th day, 24
 264 hours after the last training day, the maze was placed just as it was during the training phase and the
 265 probe trial was conducted. The animal was covered with the cylinder and after 10seconds the cylinder
 266 was lifted and the buzzer was switched allowing the animal to explore the maze. After a fixed interval of
 267 90seconds, the rat was removed. The latency was measured.

268 **SACRIFICE OF EXPERIMENTAL ANIMALS**

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

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

274 **RESULT AND DISCUSSION**

275 **BODY WEIGHT**

276 As shown in the figure 1 below, at the end of the experiment the final body weight of group A (control)
 277 was 152.4 ± 2.112 . There was no significant difference in final body weight of group B (VCO)
 278 (138.8 ± 4.652) when compared to group A (control) but group B was slightly lower than group A (control).
 279 The final body weight of group C (AlCl_3) (111.8 ± 6.822) was significantly ($P < 0.05$) lower than group A
 280 (control) (152.4 ± 2.112). The final body weight of group D ($\text{AlCl}_3 + \text{VCO}$) (132 ± 3.564) was significantly ($P <$
 281 0.05) higher when compared to group A (control) (152 ± 2.112).

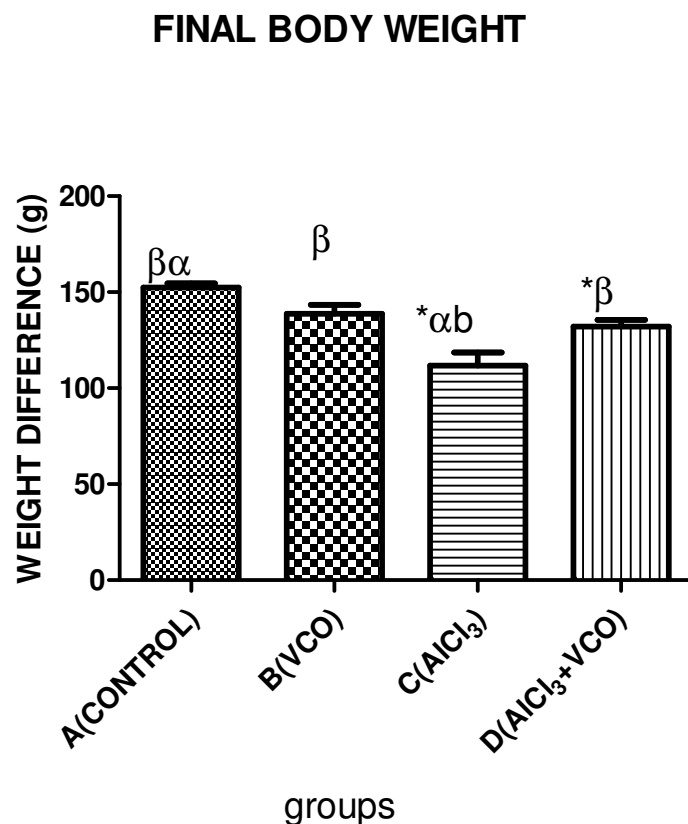


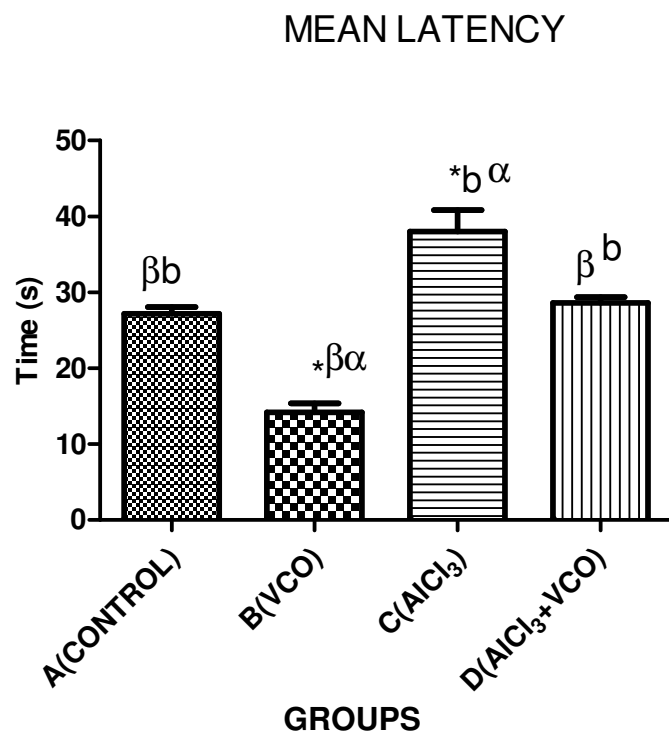
Fig. 1 Bar chart showing the final body weight of control and treated groups after 42 days

NEUROBEHAVIOURAL STUDY

BARNES MAZE

MEAN LATENCY

In the figure 2 below the mean latency for group A (control) was 27.20 ± 0.8602 . The mean latency for group B (VCO) ($14.20 \pm 1.1.58$) 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).



293

294 Figure 2 Values are mean $\pm$ SEM of data obtained; * = significantly different from group A (control), b =
 295 significantly different from group B (VCO), β = significantly different from group C (AlCl₃), α = significantly
 296 different from group D (AlCl₃ + VCO), $P < 0.05$.

297 HISTOLOGICAL RESULTS

298 PLATE 1: Photomicrographs of different groups stained with H&E

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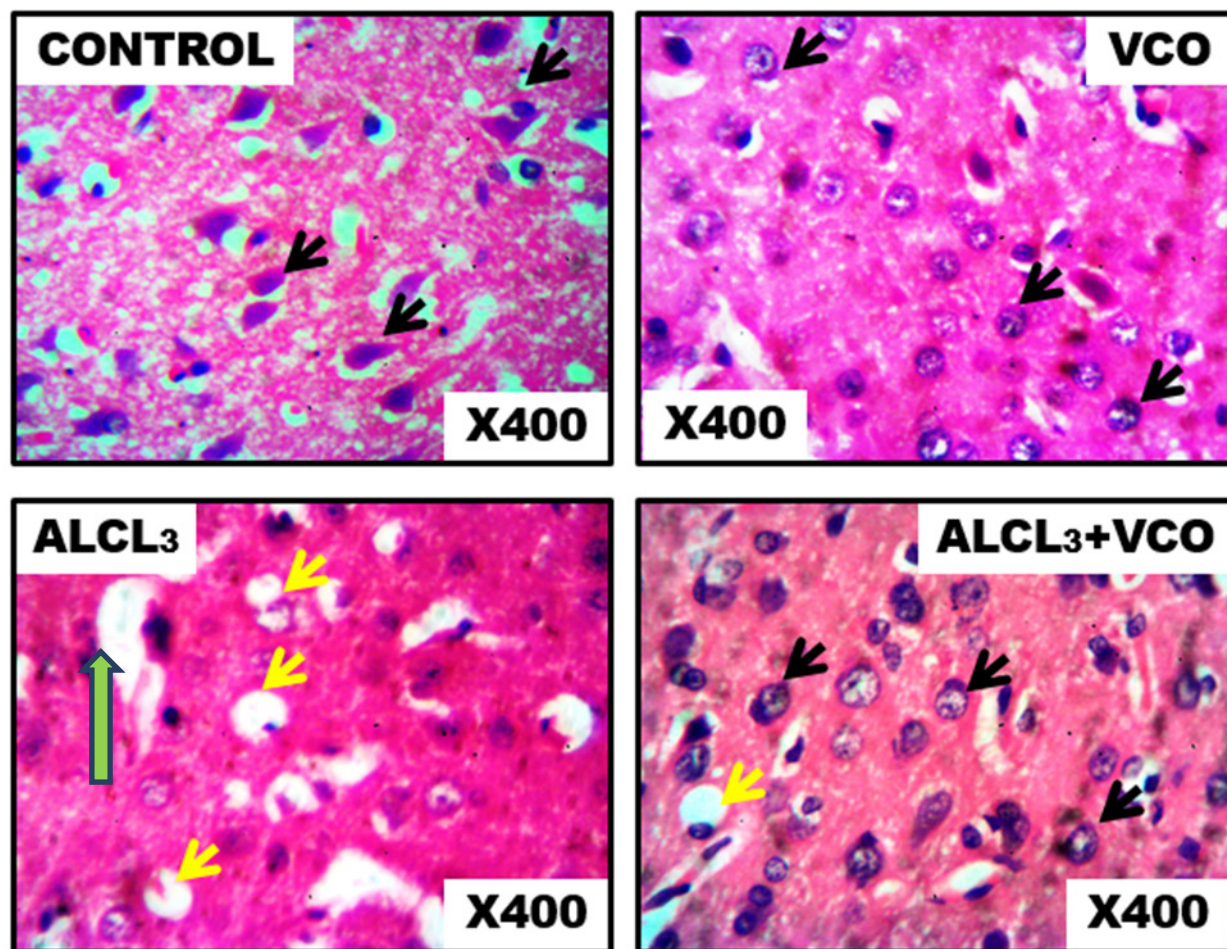
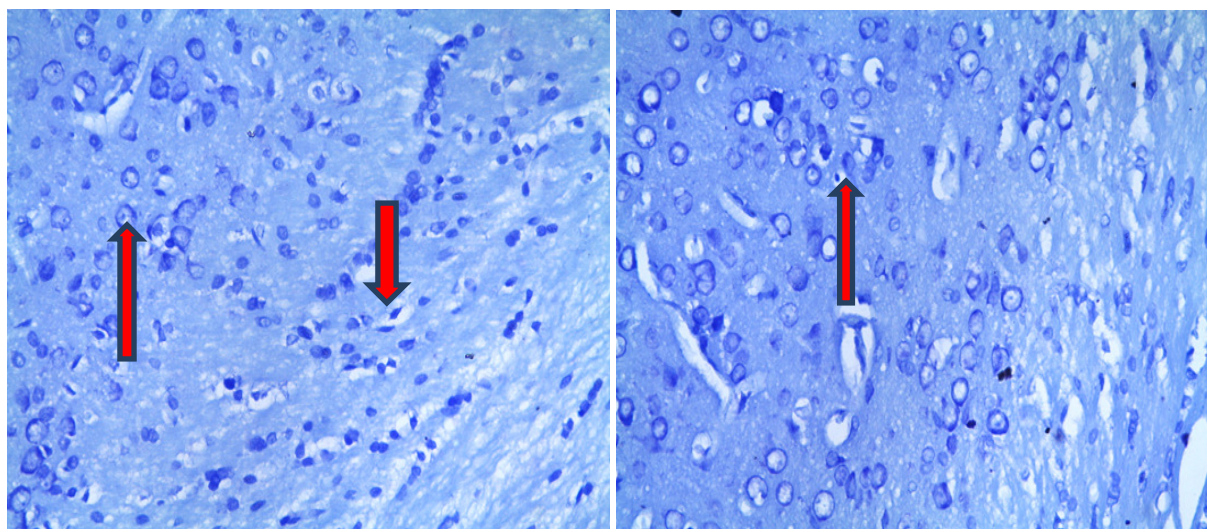


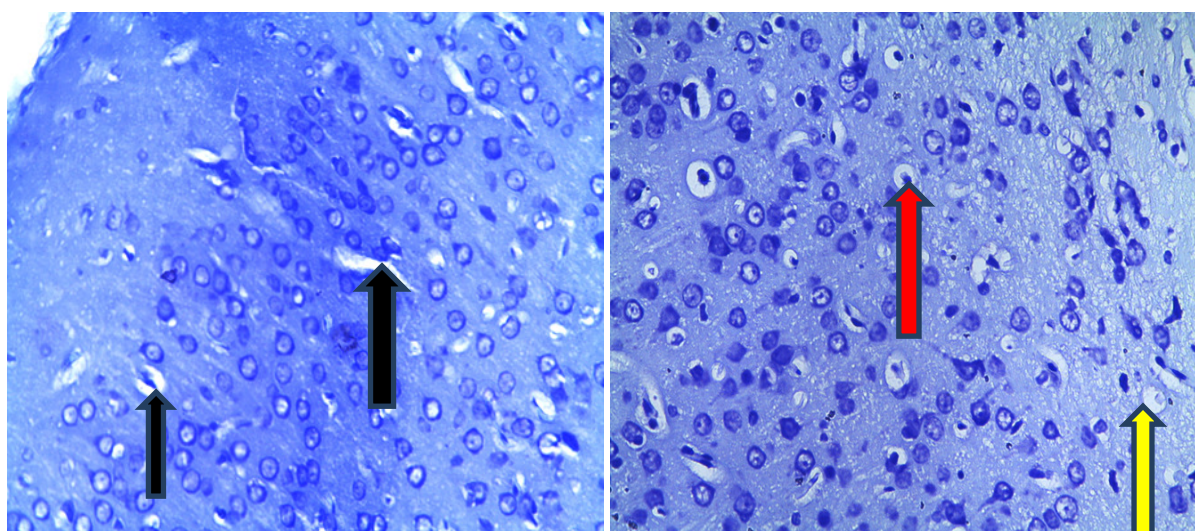
Plate 1 Photomicrographs showing panoramic views of prefrontal cortex general histomorphological presentations in Wistar rats across the study groups. Hematoxylin and Eosin stain (X400). The Control and VCO groups (Group A & B) present normal neurons and glia cell without any mechanical assault (Black 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).

315 PLATE 2: Photomicrographs of all the groups stained with Cresyl fast violet

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319 Plate 2: Photomicrographs showing Nissl profiles of prefrontal cortex general histomorphological
 320 presentations in Wistar rats across the study groups. Cresyl fast violet stains (X100). The Control and
 321 VCO group (Group A&B) presented normal nissl profiles (Red arrows). Group C (group ALCL₃) showed
 322 serious loss of nissl substance (Black arrows) while Group D (ALCL₃ + VCO) showed moderate or fewer
 323 loss of nissl substance (Yellow arrow) as well as normal nissl substance (Red arrow).

324 DISCUSSION AND CONCLUSION

325 Alzheimer's disease is a neurodegenerative disorder characterized by a progressive loss of memory and
 326 cognition⁽²⁾. Despite more than a century of research, and the massive information on AD, the causes
 327 and an early and accurate diagnosis and treatment remain elusive⁽⁴⁴⁾. In AD there are widespread
 328 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.*,2008 stated that the saturated fatty acids in VCO consists of medium chain fatty acids (MCFA) that are made up 8-10 carbons. MCFA is easily digested and absorbed and is directly absorbed into 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, 2013 in which there was a reported decrease in weight of the wistar rats administered aluminum 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 $AlCl_3$ treated animals.

The general cyto-architecture 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 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.

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 is 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 protect and ameliorative effects that is 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:

ETHICAL APPROVAL:

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