A comparative Study of the Effects of Virgin Coconut Oil and Vitamin D on Alzheimer’s Disease Induced by Aluminium Chloride in Male Albino Rats Hippocampus

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Abstract

ALZHEIMER’S DISEASE (AD) is a neurological disorder that is leading to dementia worldwide. This study aims to determine the effects of virgin coconut oil and vitamin D on AD induced by aluminum chloride in rats. Thirty adult male Albino Wister rats were divided into five groups (six rats for each group). The first group served as a control group; the second group was given an oral dose of 200 mg/kg b.w. AlCl₃ every day for 6 weeks; and the third group was supplemented with VD orally daily at a dose of 500 IU/kg b.w. for 6 weeks and an oral dose of 200 mg/kg b.w. AlCl₃ after one hour. The fourth group was given an oral dose of 5 ml of VCO and a dose of 200 mg/kg b.w. after one hour of AlCl₃ daily. The last group received an oral dose of 1.5 mg/kg b.w. of Rivastigmine (RT) daily and an oral dose of 200 mg/kg b.w. of AlCl₃ after one hour. After six weeks, the behavioral test was performed in rats to assess learning and memory, and physiological and histological studies was made in the hippocampus and blood plasma to determine the effect of VCO and VD on AlCl₃-induced AD in rats. AlCl₃ led to a significant decrease in the memory and learning maze test. Further, a significant increase in AchE, LPO, and NFL, along with a significant decrease in DA, reduced GSH and SOD. In the hippocampus histological examination, we observed an increase in neuronal loss in AlCl₃-treated rats. While VCO and VD-treated rats showed a reduction in the damage caused by AlCl₃ associated with improvements in behavioral and physiological alterations. According to this study, a daily oral dosage of VCO and VD improves learning and memory in AD rats.

Keywords: Alzheimer’s disease, Aluminum chloride, Vitamin D, Virgin coconut oil, Hippocampus

Introduction

Alzheimer’s disease AD is a neurological disorder that is the leading cause of dementia worldwide [1] AD is a neurodegenerative condition characterised by the gradual degradation of hippocampal and cortical neurons that leads to impairment of memory and cognitive capacity. It is one of society’s most financially demanding diseases. Although short-term memory loss is typically the first clinical sign, distant memory retrieval remains quite intact throughout the disease [2]. Low acetylcholine levels, increased oxidative stress, and antioxidant enzyme disruption produce learning and memory problems in Alzheimer’s disease, which is considered as irreversible, progressive neurological condition. Also, reduced brain size, degeneration and death of hippocampal neurons, and aggregations of amyloid and tau proteins are all pathological features [3].

Aluminium AL is a neurotoxin that has been linked to the development of AD [4]. It enters the human body through antacids, water, food, deodorants, and medicines. AL accumulates inside
the hippocampus and brain cerebral cortex, that known to be susceptible to AD [5,6]. Aluminium chloride AlCl₃ is neurotoxic, extremely physiologically reactive, and capable of causing harm to critical brain biochemistry. It can cause neuronal dysfunction and cell death by a series of disruptive events ranging from apoptosis to rapid, severe necrosis [7].

Rivastigmine RT is a cholinesterase inhibitor prescribed to treat AD dementia. On April 21, 2000, the FDA authorised it for the treatment of AD under the brand name “Exelon”[8]. This drug increases cholinergic activity by suppressing the enzyme acetylcholinesterase, which catalyzes the breakdown acetylcholine quite rapidly [9].

It is used to treat the symptoms of moderate to severe Alzheimer’s disease, for greater solubility, it is employed in the form of a tartrate salt. Rivastigmine belongs to a class of chemicals known as carbamates, which cause carbamoylation of the enzyme’s active site and inhibit its action [10].

Virgin coconut oil VCO is a useful food oil [11]. VCO is a natural plant extract that is studied for its ability to protect against the symptoms of AD. VCO demonstrated the potential to prevent brain cell death and enhance cognitive function in AD patients [12]. VCO is commonly derived from fresh coconut using a low-heat method with no additives to guarantee that bioactive constituents such as polyphenols, essential fat, and glycolipids are retained in the oil. VCO has been shown to have several potential beneficial properties as an antioxidant, anti-inflammatory, and anti-hypercholesterol agent. Many studies have been conducted to study various treatment strategies for avoiding the progression of AD, and VCO has been widely researched as a natural neuroprotectant against AD [13].

Vitamin D VD is a steroid hormone with classical roles in calcium metabolism and bone health regulation [14]. It has important roles in proliferation and differentiation, calcium signaling within the brain, and neurotrophic and neuroprotective actions; it may also alter neurotransmission and synaptic plasticity[15]. It penetrates the blood-brain barrier and binds to receptors present in neurons throughout the central nervous system [16]. Also, it inhibits neuronal necrosis via regulating voltage-gated calcium channels [17]. Moreover, it has antioxidant properties, that help to reduce oxidative stress induced by glutamate and dopaminergic toxins [18]. The current research is suggested to examine the effect of VCO as well as VD on Alzheimer’s disease in rats induced by aluminium chloride, as assessed by, behavioral aspects such as locomotors activity and exploratory, biochemical studies, acetylcholinesterase AchE, dopamine DA, glutathione GSH, superoxide dismutase SOD lipid and peroxidation LPO in the hippocampus include measuring, Neurofilament light chain NFL in blood. Also, histopathological examination of all rats hippocampus in the experimental study groups.

Material and Methods

Animals

Thirty adult male albino rats weighing 170 - 200 g were obtained from Mansour Scientific Foundation for Research and Development, Jeddah, Saudi Arabia. The animals were divided randomly into groups and housed in cages with relative humidity of 30-70%, in the temperature range of 20-24°C and light-controlled room on an alternating 12:12 h light/dark cycle with given free access to food and water. The animals were adapted to the laboratory conditions for two weeks before the beginning of the experiments. All experiments were approved and made regarding the rules of the Animal Care and Use Committee of King Abdul-Aziz University, Faculty of Pharmacology, Jeddah, Saudi Arabia (Reference NO. 784.20 animal study).

Chemicals

Aluminium chloride AlCl₃ was purchased from Alpha Chemika Mumbai, Maharashtra, India supplied as white powder. Rivastigmine (Exelon®) 1.5 mg capsule was obtained from (Novartis Company, Basel, Switzerland). Sodium chloride (NaCl) and sodium hydroxide NaOH were obtained from (Panreac, Barcelona, Spain). Potassium chloride KCl, disodium phosphate Na₂HPO₄ and formaldehyde were obtained from (Riedel-dehaen, Selleze, Germany). AchE Enzyme Linked Immunosorbent Assay (ELISA) kit, DA ELISA kits, GSH ELISA kit, SOD ELISA Kit and LPO ELISA Kit were purchased from (My BioSource, San Diego, U.S.A.).

Preparation of chemicals

AlCl₃ was used to induce Alzheimer’s like disease in animals. Animals were administered AlCl₃ orally with 200 mg/kg b.w. for six weeks[19]. 2 grams of AlCl₃ dissolve in 40 ml of distilled water mix very well and store in the
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fridge in the dark until use. Rivastigmine tartrate RT administered orally at a dose of 1.5 mg/kg b.w. [20]. 29 mg RT dissolved in 29 ml of distilled water. The doses are prepared daily to maintain the drug’s effectiveness.

**Supplement**

VCO was purchased from iherb, brand name California Gold Nutrition. Was given at a dose of 5 ml/kg b.w. [21]. VD Vitamin D3 (Cholecalciferol C_{27}H_{44}O), oral drops were purchased from Alnahdi pharmacy, brand name vidrop, VD diet corresponds to a daily dose of 500 IU/kg/day[22] 1 ml of VD dissolve in 4.6 ml of corn oil and store in the fridge in the dark until use.

**Study groups**

The rats were divided into 5 experimental groups. The first group (control group) was given normal saline (0.9% NaCl) orally each day for 6 weeks, the second group was administered an oral dose of 0.4 ml AlCl, every day for 6 weeks, the third group was supplemented with VD orally daily at a dose of 0.2 ml and AlCl, after 1 hour at a dose of 0.4 ml for 6 weeks, the fourth group was supplemented with VCO orally daily at a dose of 1 ml and AlCl, after 1 hour at a dose of 0.4 ml for 6 weeks. The last group received 0.3 ml RT orally daily for 6 weeks, followed by AlCl, after 1 hour at the same dosage as the second group.

**Behaviour Study (Maze learning test)**

Is used to assess disruptive behavior, natural spontaneous alternation spatial memory, short term memory, and cognitive decline in rodents [23].

**Tissue preparations**

After the behavioral test, the animals were euthanized by decapitation following the rules and regulations of King Abdul-Aziz University, Faculty of Pharmacology, Jeddah, Saudi Arabia. After dissection, their whole brains were removed carefully and washed in saline and divided in a sagittal plane into two halves. The right sided cerebral hemisphere for histopathological examination and the hippocampus of the left side was dissected for homogenate preparation. The tissue was immediately frozen in -80 °C for preparation of homogenate. For biochemical studies the frozen hippocampus were homogenized by using digital high-speed homogenizer (Homogenizer revolution per minute 15,000 rpm w) (Category: Emulsifier/Homogenizers Model: KRH-1). Then, centrifuged for 15 minutes. at 3000 revolutions per min by using (Fristaden Lab Digital Centrifuge 0-4000RPM | LCD Display | Bench-Top). After that supernatant was separated, aliquot and stored in Eppendorf tube at -80 °C for the biochemical assay.

**Biochemical assay**

ELISA kits were used for the measurement of hippocampus and cortex tissues homogenate for AchE (Cat. # MBS725468), SOD ELISA Kit (Cat. # MBS036924), DA Elisa kit (Cat. # MBS725908), GSH ELISA Kit (Cat. # MBS265966), LPO ELISA Kit (Cat. # MBS2515688) and NFL ELISA Kit (Cat. #MBS939608)

**Histopathological studies**

After fixation of brain tissues in formalin saline (10%) for 24 hours, brain tissues were subjected to serial dilution of alcohol for dehydration, sectioned in to 5 µm thickness and stained with Hematoxyline and Eosine (H & E) and Congo red stain to be examined by a light microscope.

**Statistical analysis**

The statistical investigation was conducted utilizing the one-way analysis of variance (ANOVA) technique which is used to reveal the differences between groups in the different variables (biochemical measurements, blood analysis, and behavioral experiments) and Scheffe’s test was used for homogeneous groups and Dunnett’s C for heterogeneous groups.

**Results**

**Behaviour study (Maze learning test)**

According to Fig. 1, the rats were all less relative average than the control group (0.30), with the lowest percentage being when treated with AlCl after three weeks with a percent of (0.14), followed by (VCO+AlCl), (VD+AlCl) and (RT+AlCl) group respectively (0.19) for each. After six weeks revealed that the effect of AlCl had the least percentage (0.00), followed by (RT+AlCl), (VCO+AlCl), and (VD+AlCl), each having a ratio of (0.14-0.15-0.14).

The amounts of AchE in the hippocampus homogenate was substantially increased in AlCl group (3.63) compared to the control group (0.35) (P˂0.01). Hippocampus AchE levels were reduced significantly in (VCO+AlCl), (VD+AlCl) groups, and (RT+AlCl) group respectively (2.62-2.63) and (1.66) compared with AlCl group (0.35) (P˂0.01) (Fig. 2).

**Hippocampus homogenate levels of DA**

Figure 3 provide a comparison of the
hippocampal homogenate concentrations of DA in various examined groups with the control and AlCl₃ groups. The concentrations of DA in the hippocampal homogenate were considerably lower in the AlCl₃ group (1.80) compared to the control group (11.23) (P<0.01). While DA levels were significantly increased in (VCO+ AlCl₃), (VD+ AlCl₃) groups, and (RT+ AlCl₃) group, respectively (12.23-11.72) and (6.03) compared with rats treated with AlCl₃ (1.80) (P<0.01).

**Hippocampus homogenate levels of GSH**

GSH concentrations in the hippocampal homogenate were substantially lower in the AlCl₃ group (2.23) compared to the control (18.37) (P<0.01). Furthermore, GSH levels in rats treated with AlCl₃ were substantially lower than in the (VCO+ AlCl₃), (VD+ AlCl₃) groups and (RT+ AlCl₃) group respectively (13.83-13.17-9) compared with rats treated with AlCl₃ (2.23) (P<0.01) (Fig. 4).

**Hippocampus homogenate levels of SOD**

Figure 5, shows a comparison of the hippocampus homogenate amounts of SOD in various studied groups with control and AlCl₃ groups. The levels of SOD in the hippocampus homogenate were significantly decreased in AlCl₃ and (RT+ AlCl₃) groups respectively (96.17-113) compared with the control group (178) (P<0.01). While hippocampus SOD levels were significantly increased in (VCO+ AlCl₃), (VD+ AlCl₃) groups and (RT+ AlCl₃) groups respectively (181-177) and (113) compared with rats treated with AlCl₃ (96.17) (P<0.01).

**Hippocampus homogenate levels of LPO**

The levels of LPO in the hippocampus homogenate was significantly increased in AlCl₃ and (RT+ AlCl₃) groups (2.67 - 1.69) compared to control group (0.63) (P<0.01). Meanwhile, hippocampus LPO levels were significantly decreased in (VCO+ AlCl₃), (VD+ AlCl₃) groups and (RT+ AlCl₃) group respectively (6.88- 0.55) and (1.69) compared with rats treated with AlCl₃ (2.76) (P<0.01) (Fig. 6).

**Levels of NFL in plasma**

Figure 7, indicate the comparison of NFL plasma levels in the examined groups with the control and AlCl₃ groups. The levels of NFL in plasma was significantly increased in AlCl₃ and (RT+ AlCl₃) groups respectively (42.83-29.64) compared to control group (21.77) (P<0.01). Meanwhile, plasma NFL levels were significantly decreased in (VCO+ AlCl₃), (VD+ AlCl₃), groups and (RT+ AlCl₃) group respectively (21.32-21.52) and (29.64) compared with rats treated with AlCl₃ (42.83) (P<0.01).

**Histopathological studies**

Histopathological investigation of the hippocampus confirmed the current findings. After six weeks, light microscopic inspection of the control group sections showed its normal shape and structure, which included Cornu Ammonis CA and dentate gyrus DG. CA had a C-shaped body with the following regions: CA1, CA2, CA3, and CA4 (Fig. 8). Both CA1 and CA3 (Figs. 9 A, B) were composed of well-defined 3 layers: outer molecular ML, middle pyramidal cell PCL and inner polymorphic layers PmL. The PCL was the main one and was formed of several layers of densely packed small pyramidal cells PC in CA1 and less densely packed large PC in CA3. The PCs were triangular in shape with vesicular nuclei and prominent processes. Both ML and PmL consisted of a pinkish background “neuropil” that contained axons and dendrites, few scattered glial cells gl some blood capillaries bc (Figs. 9 B, C). The DG (Fig. 9 C) had a V-shaped structure enclosing CA4 region by upper and lower limbs. Each limb was formed of 3 layers; outer ML, intermediate granular GCL and inner PmL. The GCL consisted of closely packed small granular cells with rounded nuclei. The ML and PmL contained few gl and bc. Congo red-stained brain sections of the control group showed no detection of any amyloid plaque deposition in different regions of the hippocampus (Figs. 9 D, E, F). While examination of the hippocampus from AlCl₃-treated group revealed the presence of variable changes in different regions of the hippocampus. The main changes were seen in the PCL of CA1 and CA3, which appeared disorganized as compared to the control group (Figs. 10 A, B). Many PC showed degenerative changes where they lost their shape and appeared shrunken with dark cytoplasm and condensed nuclei. Both ML and PmL contained few gl and bc. Congo red staining of the hippocampus (Figs. 10 A, B) showed increased gl and dilated bc. Regarding DG (Fig. 10 C), the main changes occurred in GCL that appeared disorganized with many GC appeared degenerated and shrunk with vacuolated cytoplasm and condensed nucleus. ML and PmL layers of DG showed increased gl with pericellular haloes, dilated bc. Congo red staining of the hippocampus (Figs. 10 D, E, F) showed multifocal, moderate deposition of amyloid plaques in ML and PmL of the hippocampus compared to the control group.
While the investigation of the hippocampus from AlCl\textsubscript{3} and VD group revealed improved histopathological changes as compared with AlCl\textsubscript{3} group with relatively normal thickness of the PCL in CA1 and CA3. Most of the PC were preserved in shape and showing vesicular nuclei but few cells appeared shrinked with dark condensed nuclei. ML and PmL showed increased gl and some dilated (bc) (Figs. 11A, B). Regarding DG (Fig. 11 C), VD administration resulted in improved architecture with the main changes occurred in GCL. However, some granular cells appeared shrinked with condensed nuclei. Congo red staining of hippocampus (Figs. 11 D, E, F) showed no deposition of amyloid plaques in ML and PmL of the hippocampus as compared with AlCl\textsubscript{3} group. Furthermore, the hippocampus of AlCl\textsubscript{3} and VCO group showed some improvement of the histopathological changes as compared with AlCl\textsubscript{3} group with relatively more or less normal thickness of the PCL in both CA1 and CA3. Although many PC were normal in shape, some cells appeared shrinked with dark condensed nuclei. Also, the ML and PmL showed increased gl and some dilated bc (Figs. 12 A, B). Regarding DG (Fig. 12 C), VCO administration resulted in moderately improved architecture with the main changes occurred in GCL as compared with AlCl\textsubscript{3} group. However, some GC still appeared shrinked with condensed nuclei. Congo red staining (Figs. 12 D, E, F) showed mild deposition of amyloid plaques in the ML and PmL of different hippocampal regions as compared with AlCl\textsubscript{3} group. On the other hand, examination of the hippocampus from AlCl\textsubscript{3} and RT group revealed better histopathological changes as compared with AlCl\textsubscript{3} group with relatively normal thickness of the PCL in CA1 and CA3. Most of the PC were conserved in shape and showing vesicular nuclei. However, some cells appeared shrinked with dark condensed nuclei. Also, the ML and PmL showed increased gl and some dilated bc (Figs. 13 A, B). Regarding DG (Fig. 13 C), VD administration resulted in improved architecture with the main changes occurred in GCL. However, some GC appeared shrinked with condensed nuclei. Congo red staining of hippocampus(Figs. 13 D, E, F) showed no deposition of amyloid plaques in ML and PmL layers of the hippocampus as compared with AlCl\textsubscript{3} group.

**Discussion**

Alzheimer’s disease is a common neurodegenerative disease that impairs memory, cognitive abilities, and, subsequently, the ability to accomplish everyday tasks [24]. AD has been identified as a severe medical and societal issue that is aggravated by the growing number of elderly people. The approach for currently available Alzheimer’s medications are based on inhibiting brain AchE and increasing the effectiveness of certain neuroprotective treatments [25]. Natural products provide several choices for slowing the progression and symptoms of many diseases, including Alzheimer’s [26].

The present study attempted to evaluate whether the administration of VCO and VD would have preventive effects on AD induced by AlCl\textsubscript{3}, with behavioral, physiological disturbances and histological damages in male albino Wister rats. The finding of the study revealed that six weeks oral administration of AlCl\textsubscript{3}, (200mg/kg b.w) in rats leads to behavioral, physiological, and histological changes. The study finding was consistent with [27]. AlCl\textsubscript{3} in AD considered cognitive deficits to be the most important clinical indicator and a severe health concern. The results suggested that oral dose of VCO and VD for six weeks reduced significantly AchE activity as well as increased GHS,SOD and NFL in (VCO+AlCl\textsubscript{3}) and (VD+AlCl\textsubscript{3}) groups compared to AlCl\textsubscript{3} group, the present results were in agreement with [28,29,30,31]. CVO has protective effects due to the prevalence of phenolic compounds and a diversity of substances capable of inhibiting AchE, resulting in increased AchE amount. Furthermore, the current investigation found that six weeks oral administration of RT with AlCl\textsubscript{3} resulted in a considerable reduction in AchE activities of the homogenate hippocampus [32]. It has been reported that AlCl\textsubscript{3} administration caused loss of neurons in the hippocampus but the results also revealed a neuroprotective potential of VCO and VD by reducing degeneration of the brain tissue, many earlier research discovered that polyphenols in VCO might stimulate the brain by lowering cell death in the hippocampus due to the antioxidant impact, VD is also an antioxidant. It enhances cellular activities that reduce oxidative stress induced by neurotransmitter toxicity. As a result, it prevents neuronal apoptosis [33,34].

**Conclusions**

In conclusion, the current study revealed that model AD induced by AlCl\textsubscript{3}, caused behavioral, physiological, and histological changes in Albino Wister rats and that AlCl\textsubscript{3} caused increases in the levels of NFL in blood and AchE, LPO in...
hippocampus homogenate, resulting in oxidative stress and a significant decline in antioxidant enzymes GSH as well as SOD compared to the control group. In contrast, the present research discovered that VCO and VD has a strong preventative influence in the hippocampus, as indicated by behavioral, biochemical, and histological improvements in rats. The preventive effect of VCO and VD reversed the memory impairment caused by AD induced by AlCl$_3$. It also reduced nerve impairment in AD rats by reducing oxidative activity and increasing antioxidant enzymes. According to the findings of the study, the usage of VCO and VD can slow the progression of AD, help to reduce symptoms associated with the disease, as well as improve memory deficits by decreasing behavioral disturbances as proven in this study. In my opinion, we should support the natural alternatives treatment for AD and enhance people to maintain VD levels which improves cognitive abilities and memory according to the results and provides an effective treatment that helps AD patients avoid the side effects of chemical drugs and encourages further research and studies on the treatment of AD.

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Conflict of interest

The authors has no conflict of interest to report.

Ethical approve

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Author`s contribution

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Ohoud Aldemh, Fatima Alnafaei and Ebtesam Bawazir. The first draft of the manuscript was written by Ohoud Aldemh and co-authors commented on previous versions of the manuscript. Co-authors read and approved the final manuscript.

Fig.1. Evaluation of preventive impact of virgin coconut oil and vitamin D on AlCl$_3$ induced Alzheimer’s disease in rats by using maze learning test

Data was expressed as mean +/- standard error

*: significance versus control (P<0.01)

#: significance versus AlCl$_3$ (P<0.01)

Significance was made using One way ANOVA test (Scheffe’s test)

Fig. 2. Impact of virgin coconut oil and vitamin D on acetylcholinesterase levels in hippocampus homogenate (u/ mg) for protection from AlCl3 induced Alzheimer’s disease in rats. Data was expressed as mean +/- standard error
*: relevance compared to the control (P˂0.01).
#: relevance compared to AlCl3 (P˂0.01).

Fig. 3. Effect of virgin coconut oil and vitamin D on dopamine levels in hippocampus homogenate (pg/mg) for protection from AlCl3 induced Alzheimer’s disease in rats. Data was expressed as mean +/- standard error
*: significance versus control (P<0.01)
#: significance versus AlCl3 (P<0.01)
Significance was made using One Way ANOVA test (Scheffe’s test)
Fig. 4. Influence of virgin coconut oil and vitamin D on glutathione levels in hippocampus homogenate (u/mg) for protection from AlCl3 induced Alzheimer’s disease in rats.

Data was expressed as mean ± standard error
*: relevance against control (P˂0.01)
#: relevance versus AlCl3 (P˂0.01)
The one-way ANOVA test (Scheffe’s test) was used to evaluate relevance.

Fig. 5. The influence of virgin coconut oil and vitamin D on superoxide dismutase concentrations in hippocampal homogenates (µmol/mg) for protection against AlCl3-induced Alzheimer’s disease in rats.

Data was recorded as mean ± standard error
*: significance versus control (P<0.01)
#: significance versus AlCl3 (P<0.01)
Significance was made using One way ANOVA test (Scheffe’s test)
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Fig. 6. Effect of virgin coconut oil and vitamin D on lipid peroxide concentrations in hippocampal homogenate (µmol/mg) for AlCl₃-induced Alzheimer’s disease prevention in rats.

Data was recorded as mean +/- standard error

*: significance versus control (P<0.01)
#: significance versus AlCl₃ (P<0.01)

Significance was made using One way ANOVA test (Scheffe’s test)

Fig. 7. Virgin coconut oil and vitamin D reaction of Neurofilament Light Chain amounts in plasma (pg/ml) were shown to be preventive versus AlCl₃ induced Alzheimer’s disease in rats.

The findings were recorded as mean +/- standard error

*: significance versus control (P<0.01).
#: significance relative to AlCl₃ (P<0.01).

Significance was made using One way ANOVA test (Scheffe’s test)
Fig. 8. A photomicrographs of H & E stained brain sections from control group showing C-shaped CA and DG. Notice the different parts of CA: CA1, CA2, CA3, and CA4. [H&E x 40].

Fig. 9. Representative photomicrographs of the hippocampal regions from control group showing: A) 3 layers of CA1. ML, PCL and PmL. PCL was formed of well-organized compact layers of small PC. Both ML and PmL contained some gl with small dark nuclei and perinuclear halos and few bc inside the pinkish neuropil matrix. [H&E x 200, Inset x 400]. B) CA3 was formed of 3 layers; ML, PCL, and PmL. PCL was formed of less packed large PC with large vesicular nuclei with prominent nucleoli and pale basophilic cytoplasm (Inset). Both ML and PmL contained some gl with small dark nuclei and perinuclear halos and few bc inside the pinkish neuropil matrix. [H&E x 200, Inset x 400]. C) DG contained 3 layers: ML, GCL, and PmL. GCL was formed of densely packed rounded to oval GC. [H&E x 200, Inset x 400]. D, E, F showed Congo red-stained sections of CA1, CA3 and DG from control group with no detection of any amyloid plaques deposition [Congo red x 200].
Fig. 10. Representative photomicrographs of the hippocampal regions from AlCl₃-treated group showing: A) CA1 with thinner and disorganized PCL as compared to the control group. Most PC appeared shrunken with loss of their vesicular shape and dark cytoplasm (thick arrow). Both ML and PmL layers contained more gl and dilated bc. [H&E x 200, Inset x 400] B) CA3 with thinner or loose PCL as compared to the control group. Most PC appeared shrunken and lost their shape with dark cytoplasm (thick arrow). ML and PmL layers contained more gl and dilated bc. [H&E x 200, Inset x 400] C) DG with dense GCL and dark condensed GC. [H&E x 200, Inset x 400] (D, E, F) showed Congo red-stained sections of CA1, CA3 and DG with multifocal, moderate deposition of amyloid plaques in ML and PmL layers as compared to the control group (arrow head).

Fig. 11. Representative photomicrographs of the hippocampal regions from AlCl₃ and VD treated group showing: A) CA1 was formed of three layers; ML, PCL and PmL. PCL was formed of multiple compact layers of small PC containing large vesicular nuclei and pale basophilic cytoplasm (Inset). Few cells appeared shrunken with dark condensed nuclei (thick arrow) Both ML and PmL contained more gl and more dilated bc. [H&E x 200, Inset x 400] B) CA3 was formed of three layers; the ML, PCL and PmL. PCL was formed of less packed large PC with large vesicular nuclei and pale basophilic cytoplasm (Inset). Both ML and PmL contained some gl and bc. Few cells appeared shrunken with dark condensed nuclei (thick arrow) [H&E x 200, Inset x 400] C) DG was formed of three layers; ML, GCL and PmL. The inset of GCL contained densely packed rounded to oval GC. [H&E x 200, Inset x 400] (D, E, F) showed Congo red-stained sections of CA1, CA3 and DG with no detection of any amyloid plaques deposition [Congo red x 200].

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Fig. 12. Representative photomicrographs of the hippocampal regions from AlCl₃ and CVO treated group showing: A) CA1 was formed of three layers; ML, PCL and PmL. PCL was formed of multiple compact layers of small PC containing large vesicular nuclei and pale basophilic cytoplasm (Inset). Some cells appeared shrunked with dark condensed nuclei (thick arrow). Both ML and PmL contained more gl and more dilated bc. [H&E x 200, Inset x 400] B) CA3 was formed of three layers; the ML, PCL and PmL. PCL was formed of less packed large PC with large vesicular nuclei and pale basophilic cytoplasm (Inset). Notice the presence of some shrunked cells with dark condensed nuclei (thick arrow). Both ML and PmL contained some gl and bc. [H&E x 200, Inset x 400] C) DG was formed of three layers; ML, GCL and PmL. The inset of GCL contained densely packed rounded to oval GC. [H&E x 200, Inset x 400] (D, E, F) showed Congo red-stained sections of CA1, CA3 and DG with mild detection of few amyloid plaques deposition (arrow) [Congo red x 200].

Fig. 13. Representative photomicrographs of the hippocampal regions from AlCl₃ and Riva treated group showing: A) CA1 was formed of three layers; outer ML, middle PCL, inner PmL. PCL was formed of multiple compact layers of small PC containing large vesicular nuclei and pale basophilic cytoplasm (Inset). Both ML and PmL contained more gl and more dilated bc. [H&E x 200, Inset x 400] B) CA3 was formed of three layers; the ML, PCL and PmL. PCL was formed of less packed large PC with large vesicular nuclei and pale basophilic cytoplasm (Inset). Some cells appeared shrunked with dark condensed nuclei (thick arrow). Both ML and PmL contained some gl and bc. Few cells appeared shrunked with dark condensed nuclei (thick arrow). [H&E x 200, Inset x 400] C) DG was formed of three layers; ML, GCL and PmL. The inset of GCL contained densely packed rounded to oval GC. [H&E x 200, Inset x 400]. (D, E, F) showed Congo red-stained sections of CA1, CA3 and DG with no detection of any amyloid plaque deposition [Congo red x 200].

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References


دراسة مقارنة لتأثير زيت جوز الهند البكر وفيتامين د على مرض الزهايمر المستحدث بواسطة كلوريد الألومنيوم في قرن أمون في ذكور الجرذان البيضاء

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قسم العلوم البيولوجية - كلية العلوم - جامعة جدة - جدة - المملكة العربية السعودية.

الملخص
مرض الزهايمر هو اضطراب عصبي يؤدي إلى الخرف في جميع أنحاء العالم. تهدف هذه الدراسة إلى تحديد آثار زيت جوز الهند البكر وفيتامين د على مرض الزهايمر المستحدث بواسطة كلوريد الألومنيوم في الجرذان. تم تقسيم ثلاثين ذكر بغال من الجرذان البيضاء إلى خمس مجموعات (ستة جرذان لكل مجموعة) الأولى استخدمت كمجموعة ضابطة، المجموعة الثانية أعطيت زيت جوز الهند البكر بعد ساعة واحدة من تناول كلوريد الألومنيوم بجرعة ملم لكل كجم من وزن الجسم يوميا لمدة أسابيع، المجموعة الثالثة أعطيت فيتامين د بعد ساعة واحدة من تناول كلوريد الألومنيوم بجرعة ملم لكل كجم من وزن الجسم يوميا لمدة أسابيع، المجموعة الرابعة أعطيت زيت جوز الهند البكر وفموي بعد ساعة واحدة من تناول كلوريد الألومنيوم بجرعة ملم لكل كجم من وزن الجسم يوميا لمدة أسابيع، المجموعة الخامسة أعطيت فيتامين د يوميا لمدة أسابيع وجرعة ملم كل كجم من وزن الجسم يوميا لمدة أسابيع. بعد انتهاء المدة المحددة، تم إجراء الاختبارات السلوكي والفيزيولوجية والفيزيولوجية في منطقة قرن أمون والنزاعات المكاني لتحديد تأثير زيت جوز الهند البكر وفيتامين د على مرض الزهايمر المستحدث بواسطة كلوريد الألومنيوم. أدى كلوريد الألومنيوم إلى انخفاض كبير في الذاكرة واختبارات مثالية التعلم. علاوة على ذلك، هناك زيادة كبيرة في مستوى إنزيم أسيتيكلين استراز وبيروكسيد الأوزون وسلسلة بروتينات الأوزون في الجرذان المعالجة بش Redirect遷تعاني مع كلوريد الألومنيوم. و فيتامين د، و فيتامين D، وأظهرت الدراسات أن نстроенج الوعي البصري وゅبطة من الاستراحات المكاني، بما في ذلك ألموهية البكر، ووذبانية الوعي في النسيج النسيجي. وفي هذه الدراسة، فإن جرعات يومية من زيت جوز الهند البكر وفيتامين D، يعكس كرارة الجرذان البيضاء على تحسين الوعي والذاكرة في ذكور الجرذان البيضاء.

الكلمات الدالة: مرض الزهايمر، كلوريد الألومنيوم، فيتامين D، زيت جوز الهند البكر، قرن أمون.