Niger. J. Physiol. Sci. 39 (June 2024): 101 – 109
www.njps.physiologicalsociety.com

Niger. J. Physiol. Sci.

Full length Research Article

Efficacy of Aqueous Extract of *Talinum Triangulare* on the Microanatomy of the Hippocampus and Short-Term Memory of Scopolamine Hydrobromide-Induced Alzheimer's Type Cognitive Dysfunction Rats

***Eru E.M.[1], Udo-Affah G.U.[1], Bassey I.F.[1], Uruakpa K.C.[1], Paulinus S.O.[2], Oku M.E.[1], Umoh N.M.[1], , Anani S.E.[1], Igiri A.O.[1]**

[1]*Department of Anatomical Sciences, University of Calabar, Calabar, Nigeria*
[2]*Department of Radiography & Radiological Science, University of Calabar, Calabar, Nigeria*

Summary: The study aimed at elucidating the potency of aqueous extract of *Talinum Triangulare* on the hippocampal neurons, astrocytes as well as assessing short term memory of scopolamine-induced Alzheimer's type rats. Fifty-four Wistar rats (180-200g) were used for the study; thirty experimental rats were randomly grouped into five, each containing six rats designated A, B, C, D and E while twenty-four rats were used to establish 50% lethal dose (LD50). Alzheimer's type cognitive dysfunction was intraperitoneally (ip) induced with scopolamine hydrobromide (1mg/kg, ip) for seven days in groups B-E prior to the oral administration of the aqueous extract (875 and 1750mg/kg) and donepezil (1mg/kg), followed by the novel object recognition test, histological and GFAP staining processes. Results revealed atrophied pyramidal cells, hyperchromatic, numerous glial cells with pale cytoplasmic inclusions and astrogliosis in groups B, C, and E while group D showed ameliorative potentials compared to group A. Also, short term memory was significantly higher in group D compared to groups B, C and E. In conclusion, aqueous extract of *Talinium triangulare* leaves reduced the potentials of scopolamine hydrobromide by restoring abnormal neurons, hence, enhancing cognitive memory in the rats used in the present study.

Keywords*: Alzheimer's disease, Astrogliosis, Atrophy, Rats, Scopolaminne hydrobromide. Talinium triangulare leaves*

**Authors for correspondence: erumba@unical.edu.ng, Tel: +2347065560843*

Manuscript received- February, 2023; Accepted- December, 2023

DOI: https://doi.org/10.54548/njps.v39i1.13

INTRODUCTION

Cognitive alteration is a very serious global health issue with neuropsychiatric and neuro-degradative ailments such as Alzheimer's disease (AD) which are debilitating in nature (Commenges *et al*., 2000). Neuroprotection is the process and relative mechanisms that protect the central nervous system upon neuron injury which may be as a result of vehement (stroke or trauma) or habitual nervous tissue ailments for example, AD (Kummar *et al*., 2006). Alzheimer's ailment is a habitual neuro-degrading malady defined analytically by mental and recall loss, with extracellular amyloid plaques and intracellular neurofibriliary entangles as it classical ensign pathologies (Zheng *et al*., 2007 and Lee *et al*., 2001). In addition, short-term and long-term memory among others is affected.

The universal incidence of nervous ailment from 60 years and above ranged from 5 to 7% (Prince *et al*., 2013). Akter *et al*.,(2012) inferred that AD is the most habitual kind of dementia followed by arteriovenous dementia. Moreover, there is scanty knowledge on the frequency of dementia and its sub-types in the sub-Saharan Africa (Paddick *et al*., 2013; Prince *et al*., 2013; Ferri *et al*., 2005) with extensive hints on public health policies. While certain reports disclosed minimal happenings of dementia in sub-Saharan Africa (Prince *et al*., 2013; Paraiso *et al*., 2011; Yusuf *et al*., 2011), others disclosed happenings rates analogized with those in the Western countries (Paddick *et al*., 2013; Guerchet *et al*., 2009). The incidence of AD in the Yoruba Africans was 2 to 3 times low analogized compared to the African Americans (Hendrie *et al*., 1995). Studies also disclosed an apparent contrast in AD happenings between males and females in which two thirds of AD cases were females (Alzheimer's Association, 2017). In 2010, the impact on health care budget revealed that dementia cost $ 800,000,000,000.00 and may hit $1,000,000,000,000.00 by 2018 worldwide (Prince *et al*., 2016), which is far more than what most developing countries faced with economic strife can afford. Also, there is also emotional as well as physical stress brought to the family of the AD patient (Sugimoto *et al*., 2000).

In the past 20 years, nutritional neuroscience came up with significant contributions to our understanding of the relation between nutrition and cognitive physiology (Gillette *et al*., 2007), where medicinal herbs such as *Talinum Triangulare* as a choice of preventing AD became supreme to multiple researchers. *Talinum Triangulare* popularly referred to as water leaf, is a common plant in our locality and grows in maximum part of Nigeria. This herb is known to contain antioxidants, antidiabetic, antibacterial, anti-inflammatory and antifungal qualities (Nkosi *et al*., 2006). The plant is documented to possess neuroprotective effects as AD influence largely the cerebral cortex and the hippocampus with loss of mass and atrophy as the disease advances (Apostilova *et al*., 2012). Moreover, neurodegenerative disease is ascribed to oxidative weight caused by the formation of disenthrall oxidants resulting to cell destruction by changing large molecules in the cell (Lim *et al*., 2001). The antioxidants in the cell and that from the plants such as *Talinum Triangulare* helps in neutralizing the extra disenthrall radicals, protect the cell from poisons and also interpose to obviate disease (Pharm-Huy *et al*., 2008). The disenthrall oxidant searching attribute of *Talinum Triangulare*, due to the elevated amount of polyphenols (Oboha *et al*., 2012) may provide a safe preference; hence, the need to investigate the efficacy of aqueous extract of *Talinum Triangulare* leaves using cognitive dysfunction rats in the present study.

MATERIALS AND METHODS

Animal breeding: In line with the principles of laboratory animal care (NIH publication NO. 85-23, revised 1985) including specific national laws applied, ethical approval (Approval No.: FAREC-FBMS 042ANA3719) was gotten from the Faculty of Basic Medical Sciences, College of Medical Sciences, University of Calabar, Calabar, Nigeria. A total of fifty four adult Wistar rats (female and male) with lade between 180-200g were obtained from the University of Calabar animal farm. The rats were housed for two weeks at standard conditions of temperature (27oC – 30oC) for acclimatization in animal house located in the Department of Human Anatomy, University of Calabar. Rat chow manufactured by the Agro Feed Mill Nigeria Ltd, Calabar and drinking water were used to feed the animals. The rats were haphazardly divided into five arrays; each array with six rats labelled A, B, C, D and E while the lingering twenty-four rats were used to establish the LD50 after acclimatization.

Plant extract preparation: Fresh water leaves (*Talinum Triangulare*) were bought from the Watt market, Calabar, Cross River State, Nigeria. In the Department of Botany, University of Calabar, the fresh *Talinum Triangulare* was registered (voucher number: HERB/BOT/UCC/332), acknowledged, and authenticated. In order to free debris, the leaves were washed and air-dried in the laboratory. With the use of a mixer grinder (model number Bravo3JARS),the dried samples were merged into powder (1600g) and macerated in 1000 mL of distilled water for 24 hours. With the use of a Whatman No.1 filter paper and chess cloth, the mixture was then filtered. A man-made vacuum (model number F.NR:1508.0271) was then used to concentrate the solution obtained. The syrupy residue was kept in a cool dry place for later use at 40-50°C.

Alzheimer's type cognitive dysfunction induction: The induction of Alzheimer's type cognitive dysfunction was done through ip administration of 1.0mg/kg body weight of scopolamine hydrobromide (SHB) to the rats in arrays B, C, D and E for seven days.

LD_{50} determination: Twenty-four rats were used to establish the LD50. According to the Lorke's method, the LD_{50} of ethanol extract of *Talinum Triangulare* was established to be >7000mg/kg. Using 12.5% (875mg/kg) and 25% (1750mg/kg) of the established LD50 (7000mg/kg body weight of aqueous extract of *Talinum Triangulare*), the dosage of ethanol extract administration was determined.

Donepezil administration and plant extract: Group A(negative control) was given animal chow and water ad libitum; array B received 1.0mg/kg body weight of SHB only to become the positive control; array C was given 1.0mg/kg body weight of SHB and 1.0mg/kg body weight of Donepezil; 1.0mg/kg body weight of SHB and 875mg/kg body weight aqueous wring of Talinum tiangulare was administered to group D and array E was given 1.0mg/kg body weight of SHB and 1750mg/kg body weight aqueous wring of Talinum tiangulare for fourteen days.

Histological method: The animals were immolated with their brain tissues perfused and treated24 hours after the last administration. The whole brain was eviscerated, weighed and fixed in 10% buffered formal saline. This was done to maintain the morphological integrity of the tissue (Williams *et al*., 2006). The hippocampus of the rats was dissected out and used for histological studies using the haematoxylin and eosin (H & E) staining method and observed under light microscope (OMAX: 40X-2500X). During tissue processing, the hippocampal tissue was dehydrated through ascending percentage of alcohol for an hour each. The tissue was cleared in two changes of xylene for an hour each, infiltrated and embedded in molten paraffin wax. The solid tissue blocks in paraffin were mounted in the rotator microtome and sections were cut at six µm. The cut sections were floated in warm water bath, then picked and mount with an albumenized slide. Paraffin slides of the hippocampal tissue were de-waxed of paraffin through two changes of xylene for 5 minutes each, rehydrated through ascending percentage of alcohol and rinsed under ceaseless tap water. For 15 minutes, cut tissues were later dyed with haematoxylin and rinsed under ceaseless tap water for 5 minutes. For a minute, cut tissues were distinguished in acid alcohol, blued and counter stain with Eosin for another 1 minute. Sections were rinsed in tap water, dehydrate and cleared in xylene, allowed to dry with few drops of distyrene plasticizer xylene (DPX) kept on the slide and cover slipped (Avwioro, 2010).

Immunohistochemical procedure: Sections of serial paraffin (5 µm thick) were deparaffinized and dehydrated. For 30 minutes, 0.05% hydrogen peroxide was used to block the endogenous peroxidase activity. At a pH of 7.4for 5 minutes, the slides were washed in phosphate-buffered

saline. In a 0.01M citrate buffer (pH 6) in a microwave for 5 minutes, sections were later placed. At 37oC, in 30 minutes, the slides were incubated in 1% Bovine serum albumin for. At room temperature for 90 minutes, two drops of antibodies {Glial fibrillary acidic protein (GFAP) and the anti-mouse immunoglobulins conjugated to a peroxidase-labelled dextran polymer} were applied to the sections and then incubated in 3,3'-diaminobenzidine for 15 minutes. With Mayer's haematoxylin and observed under light microscope, the slides were counterstained, dehydrated, cleared and mounted with DPX.

Novel Object Recognition Task: The novel object recognition task {(NORT) a relatively high-through-put, big and delicate course for assessing composites for cognition-accentuating action} in rat, is a facile assay for cognitive physiology.

Apparatus: The NORT utilizes the equipment for open field maze. This consists of a square area surrounded by high walls. The size of the area was large (50 x 50 cm). To prevent reflections, the walls were white and the bed of the field marked with blue lines that divide the barony into equivalent diminutive squares which was good for manual scoring} concealed with plexi-glass, with the equipments domiciled on the ground. With red line (centre square), a square barony in the very midpoint of the barony was also drawn. To permit animals to observe and familiarize their environments, the laboratory area was sufficiently but dimly lit while negating stress from bright lights. Two coequal materials of coequal colour, texture and size were gotten and new material, different from the coequal materials also gotten. Rats were picked and returned to their cages at the end of the ten minutes. To prevent odour being used as a cue, methylated spirit was accustomed to absolutely perfect the open field barony. After 3-5 minutes breach, a new rat was innovated into the field and data recorded after each test.

Statistical analysis: Data were analyzed using the ANOVA with the statistical package for social science version 22. A two-way post hoc test used in multiple comparisons with results expressed as mean ± SEM at $p < 0.05$.

RESULTS

Histological results: Sections of the hippocampus in groups B, C, D and E showed atrophied pyramidal cells, hyperchromatic and numerous glial cells with pale cytoplasmic inclusions. Some cell membranes were disrupted, indicating gliosis. Hypervacuolations were also seen (Plate 1 B-E). These effects were more in group B (Plate 1B). whereas group D showed strong ameliorative potentials of aqueous extract of *Talinum Triangulare* (plate 1D).

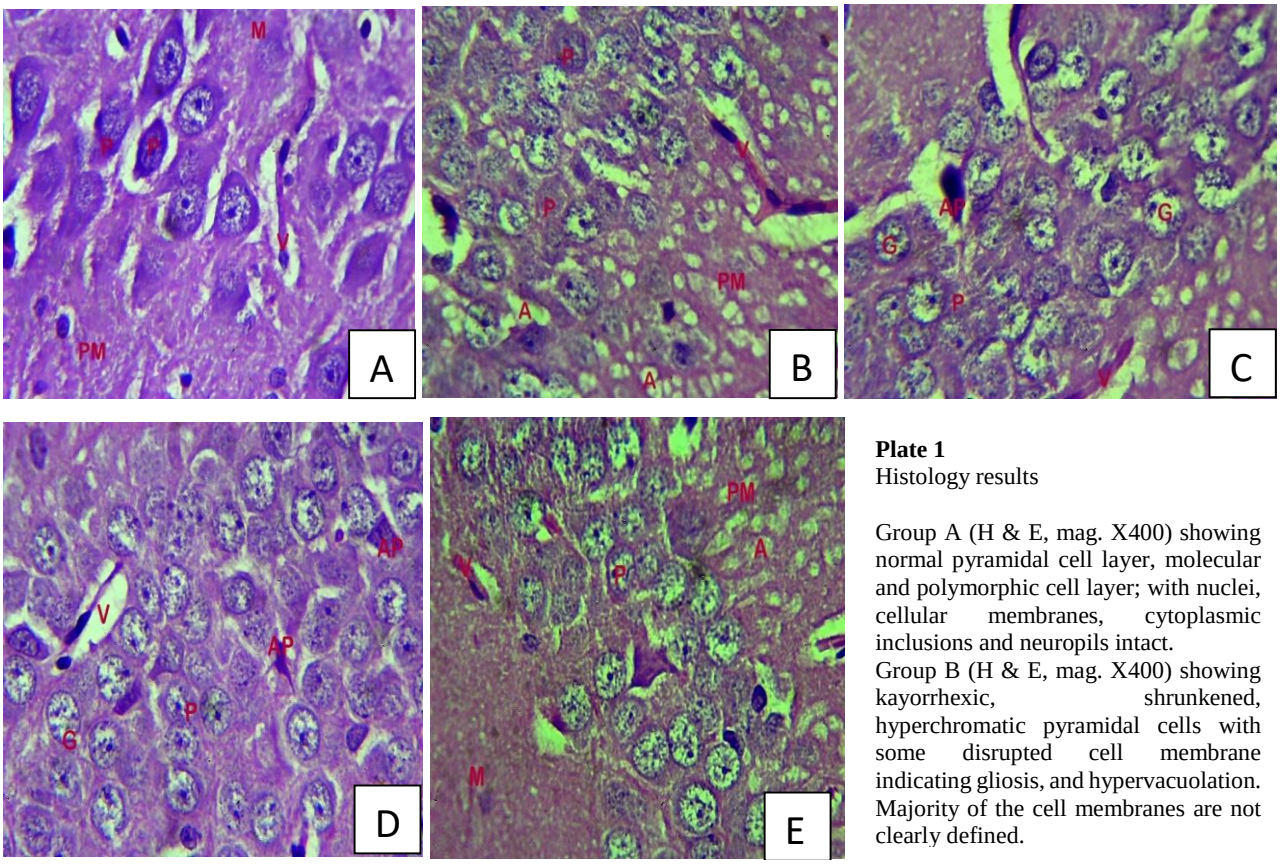

Plate 1
Histology results

Group A (H & E, mag. X400) showing normal pyramidal cell layer, molecular and polymorphic cell layer; with nuclei, cellular membranes, cytoplasmic inclusions and neuropils intact.
Group B (H & E, mag. X400) showing kayorrhexic, shrunkened, hyperchromatic pyramidal cells with some disrupted cell membrane indicating gliosis, and hypervacuolation. Majority of the cell membranes are not clearly defined.
Group C (H & E, mag. X400)showing kayorrhexic and atrophied pyramidal cell with hyperchromatic staining. There are vacuolations in the polymorphic layer filled with lipids. Majority of the cell membranes are not clearly defined.
Group D (H & E, mag. X400) showing mild atrophic, shrunken and hyperchromatic pyramidal cell with marked pale inclusions in the PM layer.
Group E (H & E, mag. X400)displaying kayorrhexic shrunken, hyperchromatic and atrophied pyramidal cells with numerous neuropils. Majority of the membranes are not clearly defined. Polymorphic layer is hypervacuolated, filled with lipids.

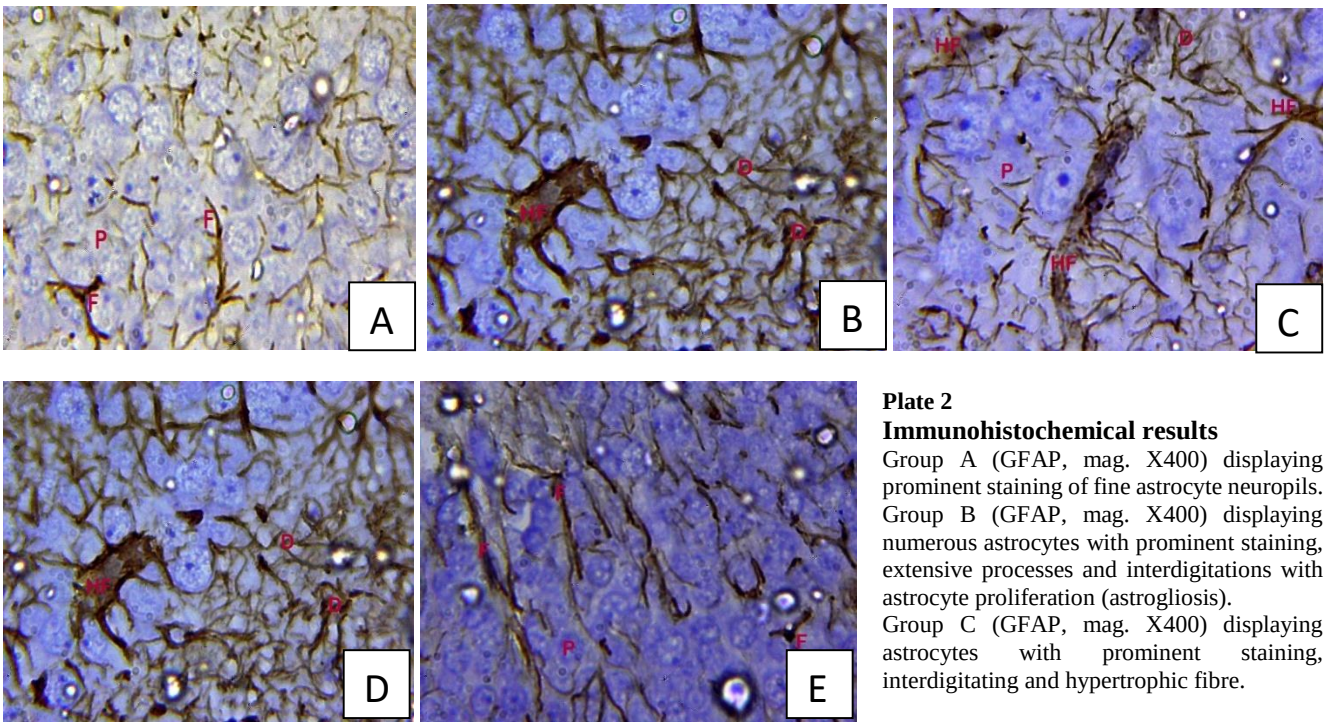

Plate 2
Immunohistochemical results
Group A (GFAP, mag. X400) displaying prominent staining of fine astrocyte neuropils.
Group B (GFAP, mag. X400) displaying numerous astrocytes with prominent staining, extensive processes and interdigitations with astrocyte proliferation (astrogliosis).
Group C (GFAP, mag. X400) displaying astrocytes with prominent staining, interdigitating and hypertrophic fibre.
Group D (GFAP, mag. X400) showing numerous astrocytes with prominent staining, extensive processes and interdigitations and hypertrophic fibre. There is astrocyte proliferation (astrogliosis).
Group E (GFAP, mag. X400) displaying prominent staining of fine astrocyte processes and fibres. There is astrogliosis.

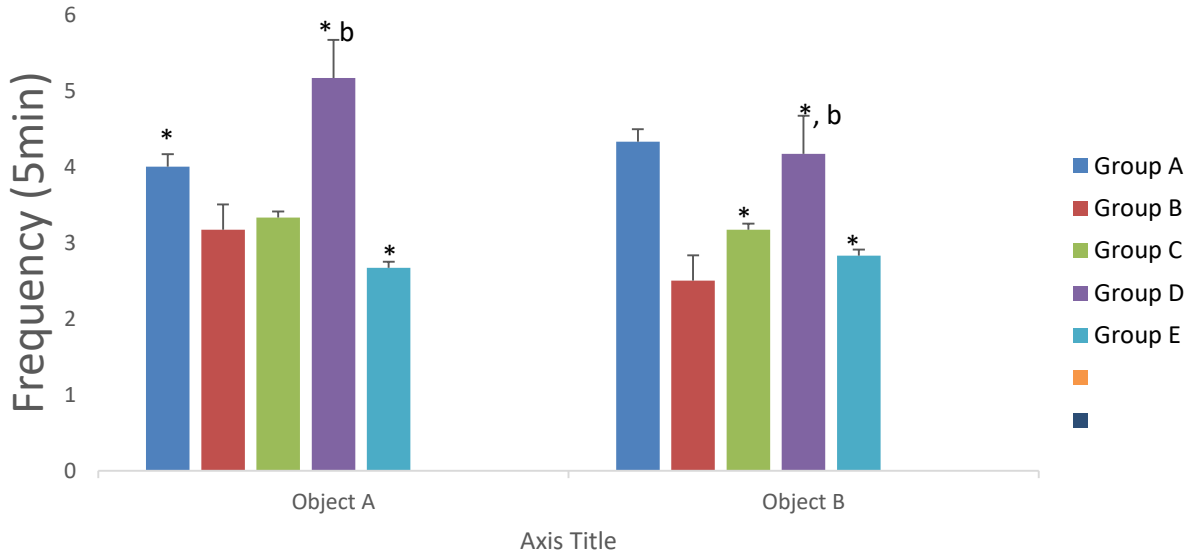

Figure 1:
Comparison of frequency of line crossing in the different experimental groups during the Novel Object Recognition Task
Values are set as mean ± SEM, n=6.
**= meaningfully variant SBB at p<0.05, b= meaningfully variant Donepezil at p<0.05*

Immunohistochemical results: The immunohistochemical sections revealed densely stained numerous astrogliosis with hypertrophied inter-digitizing fibres (Plate 2 B-E) compared to the negative control group (Plate 2A). These reactive astrocytes were less in groups C and E (plates 8 and 10) indicating pyramidal cell repairs.

Novel object recognition task: Neurobehavioral study comparing frequency of line crossing during habituation periods for object A and object B are: 4.00±0.52 and 4.33±0.71 (control group A); 3.17±0.01 and 2.50±0.89 (group B); 3.33±0.42 and 3.17±0.75 (group C); 5.17±1.05 and 4.17±1.01 (group D) and 2.67±0.33 and 2.83±0.31 (group E) (Figure1). In the object A prevalence of probationary test, array B was meaningfully decreased when compared with group A while the group E was meaningfully reduced analogized with the groups A to D. Group D was meaningfully elevated analogized with group A, B, C and E ($p<0.05$). Whereas, the object B prevalence of probationary test showed that group B was meaningfully decrease analogous to the control group at $p<0.05$ (Fig. 1).

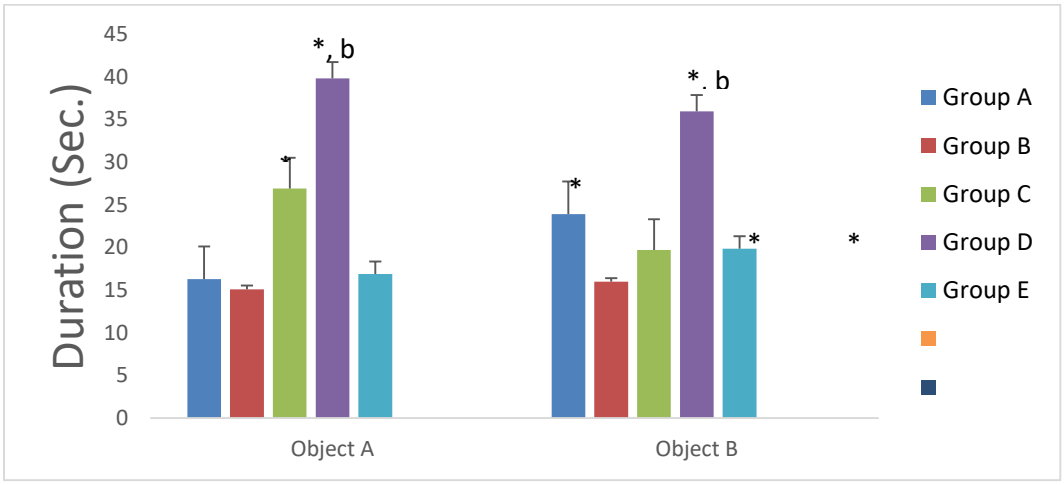

Figure 2:
Frequency of exploration test during habituation period (trial 1, day 1)
Values are seen as mean ± SEM, n=6
**= meaningfully variant control at p<0.05, b= meaningfully variant SHB at p<0.05*

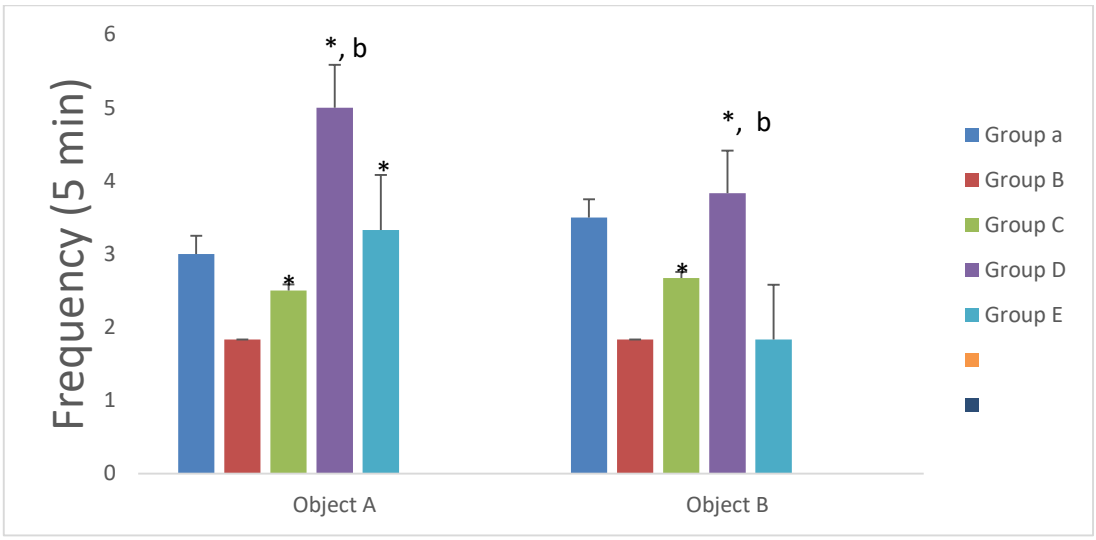

Figure 3:
Frequency of exploration test during the test period (trial 2, day 1)
Data are shown as mean ± SEM, n=6
**= meaningfully variant SHB at p<0.05, b= meaningfully variant Donepezil at p<0.05*

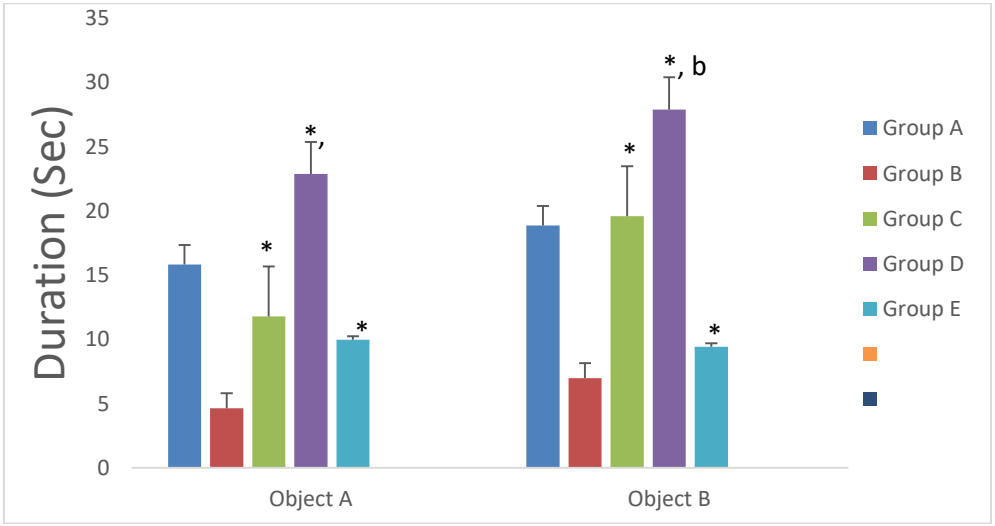

Figure 4:
Duration of exploration in NORT during trial 2, day 1(test period)
**=meaningfully variant SBB at p<0.05, b= meaningfully variant Donepezil at p<0.05*

The mean values from the duration of probationary test for object A and B during habituation period (trial 1, day 1) are: 16.31±3.66 and 23.92±6.14 (control group A); 15.11±5.29 and 15.98±5.40 (group B); 26.92±5.33 and 19.71±4.80 (group C); 39.82±8.88 and 35.96±8.74 (group D) and 16.90±3.59 and 19.85±4.32 (group E). The duration of exploratory test in object A revealed that array D was meaningfully raised comparable to other groups. The duration of probationary test in material B disclosed that array D was meaningfully raised analogous to other groups as shown in figure 2($p<0.05$). The mean values from the prevalence of probationary test for object A and B (trial 2, day 1) are: 3.00±0.58 and 3.50±0.43 (control group A); 1.83±0.70 and 1.83±0.79 (group B); 2.50±0.43 and 2.67±0.21 (group C); 5.00±0.86 and 3.83±0.87 (group D) and 3.33±1.02 and 1.83±0.48 (group E) trial 2 day 1. The prevalence of probationary test for object A in trial 2 day 1 displayed that group D was meaningfully elevated analogous to other arrays ($p<0.05$). The prevalence of probationary test for material B displayed that array D was meaningfully higher compared to array B and C with no meaningful variations between group E and group B (figure 3). The values of the duration of Novel Object Recognition Task for object A and B in trial 2 day 1 are: 15.82±2.57 and 18.86±1.95 (control group A); 4.63±1.78 and 6.97±2.38 (group B); 11.78±2.85 and 19.57±3.59 (group C); 22.86±3.35 and 27.88±7.19 (group D) and 9.96±1.03 and 9.41±2.48 (group E). The fate of period during NORT for object A revealed that array B was meaningfully reduced compared to array A while array D disclosed meaningfully elevation comparable to array A and B. Array E was meaningfully elevated when analogized with group B and meaningfully reduced when compared with array A. The continuance in array C was meaningfully longer analogous to array B (figure 4).The result of continuance of the NORT in material B disclosed that array D was meaningfully elevated when analogous to array B ($p<0.05$) and array E showed no meaningful decline when analogous to array B ($p<0.05$) and group E showed meaningful decrease when analogized with group D. Also, array C showed meaningful elevation compared with array B and group E as shown in figure 4($p<0.05$.

DISCUSSION

In rodents, the use of scopolamine to cause cognitive dysfunction is a recognized process employed to unclog inquiry and the output of aggrandize for Alzheimer's disease (AD) including other ailments (Nitta, 2002). The cognitive blemish conjugated with scopolamine hydrobromide (SHB) is cognate to that in AD. Following intraperitoneal injection of SHB, the cholinergic neurotransmitter is barred, causing cholinergic malfunction and cognitive remodelling in rats (Oh *et al*., 2009). Fan *et al*., (2005)displayed that SHB caused remembrance remodelling, is conjugated with blemished brain oxidative trouble status, this may be ameliorated with the administration of *Telfairia occidental* seeds aqueous extract at different doses (Eru *et al*., 2020a & 2021b). While B-D-glucagon polysaccharide suppresses glial cell aggregation, reduce cell count and sizes and volume in hyperglycaemic rodents (Agbor *et al*., 2022); and Citrus esculentus extract reversed radiation-induced damaged on the ultrastructure of albino Wistar rats' testes (Udoh *et al*., 2020), Averrhoa carambola aqueous fruit extract ameliorates effects of diazepam on rats hippocampus (Anani *et al*., 2020) and Ziziphus jujube fruits extract offer protective influence on oxidative brain damage and reduce the activity of AChE in hypothyroid rat brain tissues (Mohammed, 2021). In the present study, rats with SHB-induced memory remodelling were used to examine the capability of aqueous extract of *Talinum Triangulare* on its hippocampus.

Loss of cellular integrity, degeneration of cells and cellular vacuolations in the hippocampus with atrophic and kayorrhexic cells were observed in group B treated with SHB alone in the present study. These histological changes imply cellular damage which may account for the poor performance observed in the neurobehavioral tests. These features are similar to Eru *et al*., (2022, 2021a, 2021b, 2020a, 2020b); Deb *et al*., (2005) where scopolamine caused obvious impairment of recall in behavioural tests which correlate with the histomorphological alterations in the hippocampal rats. The exact mechanism responsible for this degeneration is presently not clear but may be due to oxidants formation as oxidative troubles cause neurons injury (Zou *et al*., 2015). Reports have it that SHB triggers the induction of oxidants and cause free radical injury (Fan *et al*., 2005: Lin and Beal, 2006). The hippocampus plays a good part in cognition, mood regulation, and response to stress, learning and memory (Balu and Lucki, 2009). Oxidative stress affects the hippocampus mostly (Candelario-Jalil *et al*., 2001). These degenerative changes may lead to hippocampal dysfunction characterized by impaired study and recall (Khakpai *et al*., 2012).

The cytoarchitecture of groups C, D and E showed mild effects analogous to array B treated with SHB. In our study, all the test arrays were able to ameliorate insults inflicted by SHB on pyramidal cells of the hippocampus. A study has it that loss of neurons, gliosis, swollen or destroyed axons and myelin sheath are characteristics of chemically induced neurodegeneration (Cavanagh, 1984). This is true because the neurodegenerative disease caused by SHB in group B showed atrophied pyramidal cells and numerous vacuolations filled with lipids (plate 2). The normal integrity of the soma as well as it processes is very important for normal nervous system. When the soma is injured, different degenerative changes occur because of obstacle in blood flow leading to ischemia and hypoxia, crushing of nerve fibres and injection of toxic substances/chemicals (Abbas and Nelson, 2004). In the hippocampus, the cornus amonis 1 (CA1) and cornus amonis 2 (CA2) subfields are vulnerable to cell injury (George and Schneider, 1998) which is in line with the pyramidal cells in group B, C, D and E that showed atrophied pyramidal cells, loss of plasma membranes and pale staining cytoplasm of the glial cells. The present study also confirmed involvement of pyramidal cells found in the pyramidal layer as the degenerative changes observed in the hippocampus were predominantly evident mostly in the experimental groups that received SHB. These degenerative changes may lead to dysfunction of the hippocampus characterized by inability to establish new memory. Furthermore, the distorted cytoarchitecture of the hippocampus observed was mild in the experimental group C, D, and E analogous to the positive control. The observed output showed dose affiliated format of cellular

restoration with array E having the most enhanced capabilities from the aqueous extract of *Talinum Triangulare* on the hippocampal pyramidal cells which could improve studying and recall. This agrees with David *et al*., (2008) who reported that *Talinum triangulare* has beneficial outputs on cerebral nerve cells. These ameliorative potentials could be ascribed to the elevated polyphenols (antioxidants) component in the plant which according to Pharm-Hey *et al*., (2008), could help to expunge excess free radicals, avert cell poisons as well as avert more harm from the SHB.

Immunohistochemical staining displayed little GFAP positive astrocytes in the hippocampus of array A rats. In contrast, astrogliosis and hypertrophied fibres were boldly elevated in the hippocampus of the SHB administered array. This corroborates Anderson *et al*., (2016) who documented that certain growth of astrocytes were observed after injury when the reactive reply is to preventive scar within the wound. The reactive astrocyte from other arrays administered with donepezil and *Talinum triangulare* were boldly elevated in the hippocampus when analogized with the negative control and reduce astrogliosis when analogized with the positive control administered with SHB.

The astrocytes (characteristic star-shaped cells in the brain and spinal cord called) have numerous functions including making available comprehensive structural, metabolic and trophic assistance to neurons (Sofroniew and Vinters, 2010; Verkhratsky and Butt, 2013) and regulate regular synaptic transmission (Halassa and Haydon, 2010). In response to damage inflicted on the central nervous system, astrocytes counter from their normal quiescence mode to a so called reactive mode. This headway of reactive gliosis is noticed by structural modifications (hypertrophy), physiological remodelling and absolute elevation in the expression of astrocyte specific intermediate filament and GFAP (Middeldop and Hol, 2011). The reactive astrocytes present in the nervous tissue indicate the early stage of neuronal cell loss (Abbas and Nelson, 2004). A drastic elevation in GFAP-positive astrocytes was seen in SHB administered array analogous to the control animals in the present study. Consequently, the investigation of GFAP is a sign for comprehending neurodegenerative alteration. Gliosis that happens in the scopolamine treated group could be caused by the creation of free radicals. Nerve cells are exactly susceptible to oxidative destruction due to their increased polyunsaturated fatty acid inclusion in cell envelops, increased oxygen devour and asthenic antioxidant aegis (Rego and Oliveira, 2003). Antioxidants may minimize such a reactive gliosis by decreasing the dangerous influence of reactive oxygen species in the central nervous system (Bell *et al*., 1995).

Immunohistochemical result from the present research displayed less expression of GFAP positive reactive astrocyte in group C to E. Reactive astrocytes chaperone all immediate wound and gradual neurological ailment. Reactive astrocytes while aiding cellular brace to beget axons (Anderson *et al*., 2016), can as well impair axon revitalization (Silver and Miller, 2004). When antioxidants are consumed either in diets or medications, it will neutralize reactive oxygen species from the living system and give health benefits. *Talinum Triangulare* leaves have been reported to possess antioxidants and minerals that help brain physiology. The depletion in GFAP expression cells in these experimental arrays may be ascribed to the antioxidant capabilities of the plant aqueous extract used in the present study. Studies have it that medicinal plants are rich source of antioxidant components such as flavonoids, quinones, phenolics, alkaloids and vitamins C which can reduce the rate of occurrence of oxidative stress and related disease (Zhang *et al*., 2011), *Talinum Triangulare* extract contain many of these compounds.

Neurobehavioural parameter revealed that animals in array B administered SHB alone, displayed less prevalence and continuance in examination of new recognition analogous with array A that was fed with animal chow and water ad libitum. This indicates memory impairment as recall loss is generally the precocious signal of Alzheimer's illness. This result is similar to Mugwagwa *et al*., (2015) who reported SHB caused recall impairment in long duration recall novel object recognition performance. In the current research, animals in arrays D and E revealed increased prevalence and continuance of examination of new material. This demonstrates that aqueous extract of *Talinum triangulare* may have memory-aiding activity against scopolamine-induced memory impairment. Our findings support David *et al*., (2008) who reported that *Talinum triangulare* possess favourable results on neurons of the cerebral cortex and may enhance the cognitive ability in Swiss albino rats. Consequently, data collected from NORT displayed that SHB induce studying and recall inhibition. This may be due to oxidative trouble from SHB, evidenced in the histological alterations and astrogliosis in the hippocampus of the Wistar rats.

In conclusion, aqueous extract of *Talinium triangulare* reduced atrophied cells and astrogliosis with enhanced spatial learning and memory in scopolamine induced-Alzheimer type cognitive rats in the present study. Although, the potentials of aqueous extract of *Talinium triangulare* were dose dependent.

REFERENCES

Abbas A. & Nelson F.K. (2004). Robbins and Cotran pathologic basis of diseases (7th ed.). *New Delhi, Elsevier. P* 244.

Agbor C. A., Fischer C. E., Agaba E. A. & Nnenna W. A. (2022). Neuroprotective effect of beta-D-glucagon polysaccharide on hyperglycaemia-induced cerebral injury in diabetic animal model. *Eur J Anat*, 26(2): 217-224.

Akter S. F. U., Rani M. F. A., Nordin M. S., Ab-Rahman J., Aris M. A. B. & Rathor M. Y. (2012). Dementia: prevalence and risk factors. *Int Rev Soc Sci Hum*, 2(2): 176-184.

Alzheimer Association (2017). Alzheimer disease facts and figures. *Alzh Dement*. 4 (2): 325-373.

Anani S. E., Eru E. M., Okon D. E., Uruakpa K. C.,, Ugbem T. I. & Igiri A. O. (2020). The Effect of Averrhoa Carambola (Star Fruit) Aqueous Fruit Extract on the Hippocampal Astrocyte Expression following Diazepam-Induced Neurotoxicity in Wistar Rats. *Trop J Nat Prod Res*, 4(12):1170-1173.

Anderson L, Oldridge N, Thompson D. R., Zwisler A. D, Rees K., Martin N. & Taylor R. S. (2016). Exercise-Based Cardiac Rehabilitation for Coronary Heart Disease: Cochrane Systematic Review and Meta-Analysis. *J Ame Coll Cardiol*. 67 (1):1-12.

Apostolova L. G., Green A. E., Babakchanian S., Hwang K. S, Chou Y. Y., Toga A. W. & Thompson P. M. (2012).

Hippocampal atrophy and ventricular enlargement in normal aging, mild cognitive impairment and Alzheimer's disease. *Alzh Dis Asso Disord*, 26 (1): 17-27.

Avwioro O. G. (2010). Histochemistry and tissue pathology, principles and techniques. *Nigeria, Claverianum press, Pp 143-145.*

Balu D. T. & Lucki I. (2009). Adult hippocampal neurogenesis: regulation, functional implications, and contribution to disease pathology. *Neurosci Biobe Rev*, 33 (3): 232-252.

Bell I. R., Peterson J. M. & Schwartz G. E. (1995). Medical histories and psychological profiles of middle-aged women with and without self-reported illness from environmental chemicals. *J Clin Psy*, 56 (4), 151-160.

Candelario-Jalil E., Mhadu N. H., Al-Dalain S. M., Martinez G. & Leon O. S. (2001). Time course of oxidative damage in different brain regions following transient cerebral ischemia in gerbils. *Neurosci Res*, 41(3): 233-241.

Cavanagh J. B. (1984). The problems of neurons with long axons. *Lancet*, 2: 1284-1287.

Commenges D., Scotet V., Renaud S., Jacqmin-Gadda H., Barberger-Gateau P. & Dartigues J-F. (2000). Intake of flavonoids and risk of dementia. Euro J Epidemiol. 16,(4), 357-363.

David A. O., Adelukun E. A., Ayoka A. O., Oluwayinka O. P., Omotoso E. O., Odukoya S. A., & Adeyemi D. O. (2008). Waterleaf (*Talinum Triangulare*) enhances cerebral functions in swiss albino mice. *J Neurol Sci*. 25(4): 239-246.

Deb D., Nayak V., Bairy K. L., Rao K. G. G., Shetty J., Hegde M. V. & Koshy S. S. (2013). Antiamnesic and Neuroprotective Effects of Low Dose of Ramipril and Losartan in Scopolamine-Induced Amnesia Model of Dementia. *Res J Pharm Biol Chem Sci*, 4(1): 1174-1182.

Eru E. M., Anani S. E., Paulinus S. O., Mesembe O. E., Uruakpa K. C., Umoh N. M., Oku M. E., Akpaso M. I., & Igiri A. O. (2022). Scopolamine-Induced Alzheimer's Disease in Wistar Rats; Aqueous *Talinum Triangulare* Potency on the Hippocampal Nissl Bodies and Long-Term Learning and Memory. *Trop J Nat Prod Res*, 6(1):117-122.

Eru M. E., Gabriel U. U., Samson O. P., Kelechi C. U., Michael E. O., Sadeyeng E. A, Nwosu C. B., Theresa N., Nsikak M. U., Anozeng O. I. (2021a). Restorative Potentials of Aqueous Telfairia occidentalis Seeds Extract on the Hippocampal Nissl Granules and Short Term Memory in Scopolamine Hydrobromide-Induced Alzheimer's Type Cognitive Dysfunction Rats. *Trop J Nat Prod Res*. 5(1):182-187.

Eru E. M., Paulinus S. O., Udo-Affah G. U., Uruakpa K. C., Oku M. E., Anani S. E., Umoh N. M., Akpaso M. I. & Igiri A. O. (2021b). Hippocampal Astrogliotic Reduction in Scopolamine Hydrobromide-Induced Alzheimer's Cognitive Dysfunction Wistar Rats following Administration of Aqueous Extract of Telfairia occidentalis (Hook F.) Seeds. *Niger. J. Physiol. Sci*, 36: 241 - 244

Eru E. M., Paulinus S. O., Igiri A. O. & Akpaso M. I. (2020a). Enhanced Effect of Aqueous Extract of Telfairia occidentalis Seed on the Microstructure of the Hippocampus of Scopolamine Hydrobromide-Induced Cognitive Dysfunction Rats, *Asian J Res Rep Neu*. 3(1):5-10.

Eru E. M., Paulinus S. O., Udoh-Affah G. U., Uruakpa K. C., Oku M. E., Edet U. I. & Igiri A. O. (2020b). Neurobehavioral Enhancement in Scopolamine Hydrobromide-Induced Alzheimer Type Cognitive Dysfunction in Rats Following Administration of Ethanol Seed Extract of Telfairia occidentalis (Hook.f.) Cucurbitaceae. *Trop J Nat Prod Res*, 4(7):282-285

Fan Y., Hu J., Li .J, Yang Z., Xin X., Wang J., Ding J. & Geng N. (2005). Effect of acidic oligosaccharide sugar chain on scopolamine-induced memory impairment in rats and its related mechanisms. *Neurosci Lett*. 374 (3): 222-226.

Ferri C. P., Prince M., Brayne, C., Brodaty, H., Fratiglioni L., Ganguli M., Hall, K., Hasegawa K., Hendrie H., Huang Y., Jorm A., Mathers C., Menezes P. R., Rimmer E. & Scazufca M. (2005). Global prevalence of dementia: a Delphi consensus study. *The Lancet*, 366 (9503): 2112-2117.

George D. & Scneider E. (1998). Encyclopedia of medicinal plants. Pp. 246-249.

Gillette G. S., Abellan V. K. G., Andrieu S., Barberger G. P., Berr C., Bonnefoy M., Dartigues J. F., De Groot L., Ferry M., Galan P., Herberg S., Jeandel C., Morris M. C., Nourhashemi F., Payette H., Poulain J. P., Portet F., Roussel A. M., Ritz P., Rolland Y. & Vellas B (2007) IANA task force on nutrition and cognitive decline with aging. *J Nutri He Ag* 11: 132-52.

Guerchet M., Houinato D., Paraiso M. N, Von Ahsen, N., Nubukpo P., Otto, M., Clement, J. P., Preux, P. M., Dartigues, J. F. (2009). Cognitive impairment and dementia in elderly people living in rural Benin, West Africa. *Dementia and Geriatric Cognitive Disorders*, 27(1): 34-41.

Halassa M. M. & Haydon P. G. (2010) Integrated brain circuits: astrocytic networks modulate neuronal activity and behaviour. *Annual Review of Physiology*. 72: 335-355.

Hendrie H. C., Osuntokun B. O., Hall K.S., Ogunniyi A. O., Hui S. L., Unverzagt F. W., Gureje O., Rodenberg, C. A., Balyewu, O., Musick, B. S. (1995). Prevalence of Alzheimer's disease and dementia in two communities: Nigerian Africans and African Americans. Amer J Psych, 152(10): 1485-1492.

Khakpai F., Nasehi M., Haeri-Rohani A., Eidi A., Zarrindast M. R. (2012). Scopolamine induced memory impairment: possible involvement of NMDA receptor mechanisms of dorsal hippocampus and/or septum. *Beh Br Res*, 231: 1-10.

Kummar S., Gutierrez M., Doroshow J. H., Murgo A. J. (2006). Drug development in oncology: classical cytotoxics and molecularly targeted agents. *Bri J Clin Pharmacol*,1 (62): 15-26.

Lee C. H., Herman T., Clandinin T. R., Lee R. Zipursky S. L. (2001). N-cadherin regulates target specificity in the Drosophila visual system. *Neuron* 30 (2) 437-450.

Lim G. P., Chu T., Yang F., Beech W., Frautschy S. A. & Cole G. M. (2001). The curry spice curcumin reduces oxidative damage and amyloid pathology in an Alzheimer transgenic mouse. *J Neurosci*, 21: 8370-8377.

Lin M. T. & Beal M. F. (2006). Mitochondrial dysfunction and oxidative stress in neurodegenerative diseases. *Nature*, 443: 787-795.

Middeldop J. & Hol E. M. (2011). GFAP in health and disease. *Prog Neurobiol*. 93 (3): 421-443.

Mohammed S. A. (2021). Effects of ziziphus jujube fruits extract on memory impairment induced hypothyroidism during breastfeeding and adolescence in the rats. *Jordan J Biol Sci*, 15(1)119-125.

Mugwagwa A. T., Gadaga L. L., Pote W. & Tagwireyi D. (2015) Antiamnesic effects of hydrethanolic extract of crinmummacowanii on scopolamine-induced memory impairment in mice. *J Neurodegen Dis*. 2015: 24250-24259.

Nkosi C. Z., Okpoku A. R. & Terbalanche S. E. (2006). Antioxidative effects of pumpkin seed (Cucurbita pepo) protein isolate in CCl4-Induced liver injury in low-protein fed rats. *Phytother Res*. 20 (11): 935-40.

Nitta A., Katono Y., Itoh A., Hasegawa T. Nabeshima T. (2002). Nicotine reverse scopolamine-induced impairment of performance in passive avoidance task in rats through its action on the dopaminergic neuronal system. *Pharmacol Biochem Beh*. 49: 807-812.

Oboha G., Ademilayia A. O., Akinyemia A. J., Henleb T., Jamiyu A., Saliua J.A. & Uwe Schwarzenbolzb U. (2012). Inhibitory effects of polyphenol-rich extracts of jute leaf (corchorusolitorius) on key enzyme linked to type 2 diabetes (α-amylase and α-glucosidase) and hypertension (angiotensin 1 converting) in vitro. *J Funct food*. 4:456-458.

Oh J. B., Choi B. J., Chang M. S. & Park K. S. (2009). Nelumbo nucifera semen extract improves memory in rats with scopolamine-induced amnesia through the induction of choline acetylcholinesterase expression. *Neurosci Lett*. 55(1): 41-44.

Paddick S., Longdon A. R., Kisoli A., Dotchin C., Gray W. K., Dewhurst F., Chaote P., Kalaria R., Jusabani A. M. & Walker R. (2013) Dementia prevalence estimates in sub-Saharan Africa: comparison of two diagnostic criteria. *Glob Healt Act*, 6: 1-7.

Paraïso M. N., Guerchet M., Saizonou J., Cowppli-Bony P., Mouanga A. M., Nubukpo P., Preux P. M. & Houinato D. S. (2011). Prevalence of dementia among elderly people living in Cotonou, an urban area of Benin (West Africa)," *Neuroepidemiol*, 36(4): 245-251.

Pharm-Huy L. A., He H. & Phar-Huy C. (2008). Free radical. Antioxidants in disease and health. *Int J Biomed Sci.* 4 (2): 89-96.

Prince M., Bryce R., Albanese E., Wimo A., Ribeiro W. & Ferri C. P. (2013). The global prevalence of dementia: a systematic review and metaanalysis. *Alzh Dement*, 9(1): 63-75.

Prince M., Comas-Herrera A., Knapp M., Guerchet M. & Karagiannidom M. (2016). World Alzheimer Report 2016 improving Healthcare for people living with demetia. Coverage, quality and cost now and in the future.

Rego C. & Oliveira C. R. (2003) Mitochondrial dysfunction and reactive oxygen species in excitotoxicity and apoptosis: implications for the pathogenesis of neurodegenerative diseases. *Neurochem Re*s, 28: 1563-1574.

Silver J. & Miller J. H. (2004). Regeneration beyond the glial scar. *Nat Rev Neurosci*, 5, 146-156.

Sofroniew M. V. & Vinters H. V. (2010). Astrocytes: Biology and pathology. *Acta Neuropath*, 119: 7-35.

Sugimoto H., Yamanishi Y., Iimura Y. & Kawakami Y. (2000). Donepezil hydrochloride (E2020) and other acetylcholinesterase inhibitors. *Curr Med Chem*, 7: 303-339.

Udoh B. E., Erim A. E., Paulinus S. O., Eru E. M., Ukpong E. V., Efanga I., Archibong B. E., Egong A. E., Udo E. E., Egbe N. O. (2020). Effects of Cyperus esculentus (Tiger Nut) Extract on the Irradiated Testes of Wistar Rats. *Trop J Nat Prod Res*, 4(11):966-969.

Verkhrasky A. & Butt A. (2013). Numbers: how many glial cells are in the brain? Glial Physiol and Pathophysiol. John Wiley and Sons. Pp 93-96.

Williams A., Marcus J. F., Portoghese P. S. & Carlezon W. (2006). Amygdala-prefrontal dissociation of subliminal and paraliminal fear. *Hum Brain Mapp*. 27 (8): 652-661.

Yusuf A. J., Baiyewu O., Sheikh T. L. & Shehu A. U. (2011). Prevalence of dementia and dementia subtypes among community dwelling elderly people in northern Nigeria. *Int Psychoger*, 23(3): 379-386.

Zhang L., Ravipati A. S., Koyyalamudi S. R., Jeong S. C., Reddy N., Smith P. T., Barlett J., Shanmugam K., Munch G., Wu M. J. (2011). Antioxidant and antiiflammatory activities of selected medicinal plants containing phenolic and flavonoid compouds. *J Agri Food Chem*, 59 (23): 12361-12367.

Zheng G. Z., Zhang D., Saraf A., Kolasa T., Bhatia P., Patel M., Lannoye G. S., Richardson P., Stewart A., Rogers J. C., Brioni J. D. & Surowy C. S. (2007). Fatty acid amide hydrolase inhibitors display broad selectivity and inhibit multiple carboxylesterases as off-targets. *Neuropharmacol*. 4 (52): 1095-1105.

Zou L., Zhou T., Pannell B. K., Ziegler A. C. & Best T. M. (2015). Biological and physiological role of reactive oxygen species: the good, the bad and the ugly. *Acta Physiolo*, 214: 329-348