IJBPAS, May, 2024, 13(5): 2396-2410

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

EVALUATION OF NOOTROPIC ACTIVITY OF GUGGUL AGAINST SCOPOLAMINE INDUCED MEMORY IMPAIRMENT IN ALBINO WISTAR RATS

DAMMATI ND*[1], ROHITHA B[2], SRILAKSHMI B[3], NAVYA SRI G[4], SUREKHA G[5], NAMITHA G[6], MOINA S[7] AND CHOWDARY YL[8]

Department of Pharmacology, Chalapathi Institute of Pharmaceutical Sciences (Autonomous) Lam, Guntur - 522034, Andhra Pradesh, India

***Corresponding Author: Ms. Dammati Nalini Devi: E Mail: nalinidevi86@gmail.com**

Received 25th May 2023; Revised 26th July 2023; Accepted 1st Sept. 2023; Available online 1st May 2024

https://doi.org/10.31032/IJBPAS/2024/13.5.8035

ABSTRACT

Background: Guggul is a natural gum resin used as traditional Indian medicine.

Objective: The objective of the present study is to evaluate the nootropic activity of guggul oleo gum resin against scopolamine induced memory impairment in albino wistar rats.

Methods: In this study 25 albino wistar rats were randomly selected and divided in to 5 groups. Each group consists of 5 animals - Group: 1(Control), Group: 2(negative control scopolamine 20mg/kg I.P), Group: 3(standard Donepazil 5mg/kg P.O), Group: 4&5 different versus of extract AECW (50mg&100mg/kg P.O). Memory impairment was induced with scopolamine at a single dose of 20mg/kg body weight i.p to the groups expect control group at 7th day estimation of behavioral studies was carried out by the Y-Maze, Labyrinth Maze, Elevated Plus Maze.

Results: AECW treated groups shows transfer latency similar to the donepezil treated groups. Scopolamine treated groups show more transfer latency when compared with donepazil treated groups.

Conclusion: The AECW has shown significant effect at a dose of 50mg/kg which is similar to standard donepazil 5mg/kg due to presence of phytochemical constituents like flavonoids, tannins,

alkaloids, steroids. The investigation shows AECW has nootropic activity and at a standard level useful to mankind. Y-maze, Elevated Plus maze & Labyrinth maze are the standard methods used for evaluating memory.

Keywords: Nootropic activity, scopolamine, donepazil, AECW, Y-maze, labyrinth maze, elevated plus maze, flavonoids, alkaloids, tannins, steroids

INTRODUCTION

Neurons are the functional units of the brain and nervous system. These exhibit electrical excitability property. That is an ability to respond to the stimulus and to convert it into an action potential. Stimulus is the change in the environment that is strong enough to initiate the action potential (nerve impulse). The nerve impulse is an electrical signal that propagates along the surface of a membrane of the neuron. These nerve impulses begin and travel due to the movement of the ions like sodium and potassium between the interstitial fluid and inside the neuron through the specific ion channels in its plasma membrane.

Neuroglia is the homeostatic cells of the nervous system. Neuroglia occupies half of the volume of the CNS. These actively participate in the activities of the nervous tissue. These are smaller and 5-25 times more numerous than neurons. This neuroglia doesn't generate any action potentials. These are having the ability to multiply and divide in the mature nervous system. In case of any injury, these neuroglia multiply to fill the space formerly occupied by the neurons. Gliomas are the brain tumors derived from the glial cells which are having high malignant and grow rapidly. Two types are the Schwann cells and satellite cells found in the PNS **[1]**.

Neuroglia is of 6 types: 4 types of astrocytes (star shaped), oligodendrocytes, microglia and ependymal cells found in the CNS.

NEURODEGENERATIVE DISEASES

There are different types of neurodegenerative diseases like Parkinsonism, Alzheimer's disease, Huntington's disease, Ataxia, and Dementia.

PARKINSONISM

Parkinsonism is caused by loss of dopaminergic neurons in substantianigra pars compacta. It may lead to gradual degeneration of dopamine releasing neurons causes lack of control and coordination of muscle movement, risk factor involved in Parkinsonism or oxidative stress, formation of free radicals and a number of environmental toxins. There is no definitive test for diagnosis of Parkinson disease. It is diagnosed on clinical based criteria. Rest tremor,

bradykinesia, rigidity and loss of postural refluxes are the cardinal sighs of Parkinson's disease **[1]**.

HUNTINGTON DISEASE

Huntington disease (HD) mostly occurs in between the ages of 30 and 50 years. 8% of cases start before the age of 20 years known as Juvenile HD. HD affects both men and women equally. It is an inherited autosomal dominant disorder caused in exon 1 of gene coding CAG expansion (trinucleotide repeat expansion) placed in the short arm of chromosome 4. It is a basal ganglia disease causes hyperkinetic movement disorder known as Chorea. HD is characterized by a shrinkage of brain and degeneration of stratum efferent medium spiny neurons are loosed **[2]**.

SPINAL MUSCULAR ATROPY

It is an autosomal disorder the motor neurons in the spinal cord are degenerated. Therefore, the signal from these neurons is not transmitted. Atrophy – smaller, in which muscles become smaller due to no stimulation of nerve cells. It includes symptoms like weakness of voluntary muscles, spontaneous tongue movements, breathing problems **[3]**.

ATAXIA

Ataxia is due to the damage to the cerebellum which is a part of the brain that coordinates muscle movement. The chance of occurrence of ataxia is due to over consumption of alcohol, stroke tumor, brain degeneration, multiple sclerosis. It includes symptoms like lack of coordination, unsteady walk **[4]**.

AMYOTROPHIC LATREAL SCLEROSIS

It is also called as lougehrig's disease named after a baseball player who died from this disease. It is one of the neurodegenerative disease occur very rarely i.e., 1000 cases per year in India. In this disease death of both upper motor neurons (motor cortex of brain) and lower motor neurons (located in brain stem). It is characterized by the presence of bunina (aggregation of protein) bodies in the cytoplasm of brain. It includes symptoms like nasal speech, weight loss **[5]**.

DEMENTIA

Dementia is the 7th leading cause of death globally. Due to irreversible degeneration of cerebral cortex it leads to mental deterioration. It is short-term memory impairment and it leads to alzheimer's.

STAGES OF DEMENTIA

Dementia is categorized into 3 stages they are in early stage the person seems to be not suffering with dementia. It includes symptoms like being forgetful, getting lost in known places. In middle stage the symptoms like change in behavior, facing difficulty in communication are observed. In late stage

symptoms become more elevated. It includes difficulty in recognizing the loved ones. It seems very hard to walk, shows changes in behavior like aggression.

LEWY BODY DEMENTIA:

In this dementia clumps of abnormal protein particles that accumulate in brain known as lewybodies. These lewybodies are also seen in person suffering with parkinson's disease.

SIGNS AND SYMPTOMS:

The early signs of alzheimer's disease include forgetting recent events or conversations. As the disease progress a person with alzheimer's disease will develop severe memory impairment and loss the ability to carry out everyday task.

SIGNS: It includes loss of memory which disturbs daily life, confusion with time or place, losing the ability for retracing the steps, difficulty in resolving the problems, feels hard to understand the visual images, facing problem in speaking and writing, mood swings etc.

SYMPTOMS involve cognitive include trouble in thinking, decline in mental health, unable to concentrate, delusion, increased confusion in evening hours. Behavior includes aggression, agitation, personality changes, restlessness, irritability etc.

MOOD – Anger, loneliness, apathy.

PSYCHOLOGICAL – Depression, hallucination or paranoia. Also common inability in combining the muscle movement, loss of appetite, irregular speaks.

TREATMENT

No treatment is available for dementia. Anti dementia medicines and disease modifying therapies are developed but they have limited amount of efficacy. Care for dementia include – Should diagnose early to promote early management, optimize physical health, activity, cognition. Identify and treating the physical illness, behavioral changes are to be understand and these changes should be managed.

RISK FACTORS

Age is the major risk factor. The symptoms include depression and air pollution which takes place before the age of 65 years.

PREVENTION

By avoiding smoking, alcohol, weight management, maintenance of blood sugar, cholesterol and blood pressure levels.

SOCIAL AND ECONOMIC IMPACT

In 2019, estimated total global societal cost of dementia was 1.3 trillion US dollars and these costs are expected to increase 1.7 trillion US dollars or 2.8 trillion US dollars by 2030.

EPIDEMOLOGY

Dementia affects majorly the older people, more than 55 million people (5.4% men, 8.1% women over 65 years) suffering with dementia in the world wide. Alzheimer's contribute 60-70% of cases. This number is expected to increase 78 million in 2030. By 2050 it will be raised up to 139 million. Women are more prone to dementia than men over 65% of total deaths are due to alzheimer's are women, disability – adjusted life years due to dementia are roughly 60% higher in women than in men.

Every year 10 million new cases are observed. Dementia is currently 7th leading cause of death. It affects physical, psychological, social and economic impacts not only for the people who are suffering with dementia but also their carers, families and society. In India, Alzheimer's patients are estimated to increase to 11.4 million.

To support countries in evaluating dementia, WHO released "TOWARDS DEMENTIA – INCLUSIVE SOCIETY: WHO TOOLKIT FOR DEMENTIA FRIENDLY INITIATES IN 2021" **[6]**.

PATHOPHYSIOLOGY

Alzheimer's is associated with selective loss of cholinergic neurons (produces acetylcholine) in Hippocampus and Basal forebrain. Amyloid precursor protein (APP) plays a major role in alzheimer's disease. APP gene is located on chromosome 21, one of the regions linked to familial alzheimer's disease. APP undergoes amyloidogenic pathway in presence of alpha or beta secretase (proteolytic cleavage) results in mutation of APP and presenilin favors in A**β** formation (A**β** 40 and A**β** 42). A**β** 42 is formed by the cleavage of beta and the gamma secretase. These aggregate to form oligomers. These oligomers lead to formation of amyloid plaques and neurofibrillary tangles.

Amyloid plaques are amorphous extracellular deposits of **β –** amyloid protein consisting of A**β** 40 in small amounts, A**β** 42 in larger amounts due to increased activity of alpha secretase because of presenilin gene protein present on chromosome 14. These A**β** 40 and A**β** 42 are found in plasma in higher amounts act as biomarkers.

Neurofibrillary tangles are formed by hyperphosphorylation of TAU which is present in microtubule. The primary function of TAU protein is stabilization of axonal microtubules. The TAU P paired and deposited intracellularly as twisted paired helical filaments known as neurofibrillary tangles. They occur first in the hippocampus and then may be seen in throughout the cerebral cortex. When cell dies, these filaments aggregate as extracellular insoluble neurofibrillary tangles. Finally leads to

neuronal cell death. These neurofibrillary tangles are also produced due to the oxidative stress and inflammation leading to damage of mitochondria. Another feature of alzheimer's disease is granulovascular degeneration of hippocampal pyramidal cells by amyloid angiopathy.

Acetylcholine is a neurotransmitter that has a crucial role in peripheral and central nervous system. Enzyme cholineacetyltransferase (ChAT) is responsible for synthesizing acetylcholine from acetyl CoA. Acetylcholine is useful for processing memory and learning. These parameters are reduced in patients suffering with alzheimer's. In alzheimer's disease, loss of cholinergic neurons located in the basal forebrain, causes increased activity of acetylcholinesterase enzyme leading to decline in the levels of acetylcholine in neocortex and hippocampus.

The hallmarks of the disease are the accumulation of neurofibrillary tangles and amyloid plaques [7].

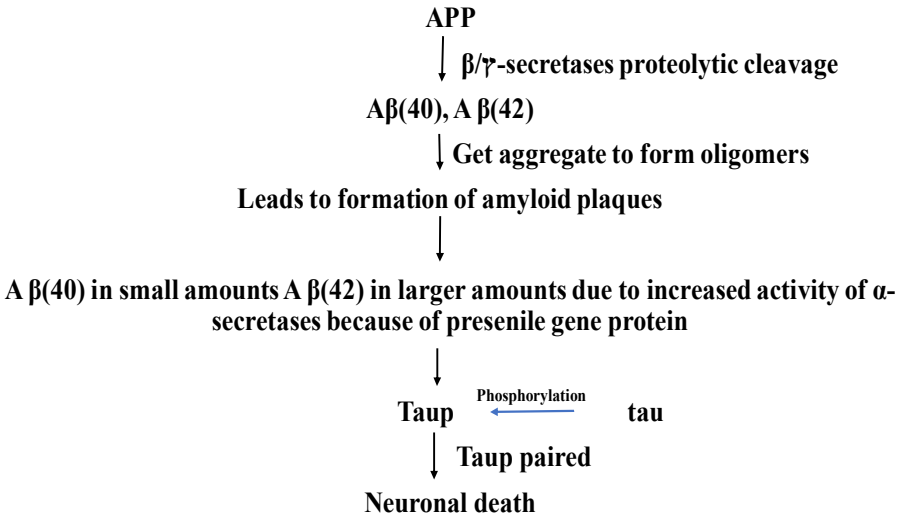

Table 1: Drugs Used In The Treatment of Alzheimer's

S. No.	DRUGS	MECHANISM OF ACTION	ADVERSE REACTIONS
1.	CHOLINESTERASE INHIBITORS:		
	Donepazil	i. It acts by selective and reversible inhibition of acetylcholinesterase enzyme and increases the cholinergic transmission and thereby it reduces the symptoms of alzheimer's disease. ii. Through the modulation of amyloid proteins and the down regulation of NMDA receptors, glutamate induces excitatory behavior.	Muscle cramps, lack of appetite, trouble sleeping, and nightmares [8].
	Galantamine	Competitively reversible acetylcholinesterase inhibitors improve acetylcholine intrinsic action on nicotinic receptors, increasing cholinergic transmission in the CNS. Acetylcholine breakdown is prevented by the drugs binding to the acyl binding pocket and choline binding site of the enzyme active site.	Bradycardia, atrioventricular block, particularly in people who have just taken drugs, and fainting risk [9].
	Rivastigmine	Rivastigmine binds to and inactivates cholinesterase (such as acetylcholinesterase and butyrylcholinesterase), which results in an increase in the concentration of acetylcholine at cholinergic synapses by stopping the hydrolysis of acetylcholine. Comparatively to acetylcholinesterase and butyrylcholinesterase in peripheral tissues, rivastigmine's anticholinesterase action is particularly selective for these enzymes in the brain.	Nausea, vomiting, loss of appetite, weight reduction, stomach ache, diarrohea [10].
2.	MEMANTINE: Aducanumab	The advancement of alzheimer's disease decreases the affinity of monoclonal Ig 1 antibody (aducanumab) binds to amyloid beta protein at amino acids 3-7.	Headache, nausea, delirium, confusion, altered mental status, disorientation, dizziness, abnormal vision [11].
	Lecanemab	A humanized IgG1 monoclonal antibody called lecanemab is being tested as a potential alzheimer's disease treatment. Aβ peptides do, however, exist in a variety of structural states, including soluble monomers, soluble aggregates of increasing size, and insoluble fibrils and plaque. Compared to monomers or insoluble fibrils, soluble Aβ aggregates like Aβ protofibrils are more hazardous. Therefore, it has been proposed that lecanemab could be utilized to treat Alzheimer's disease by concentrating on and decreasing the quantity of Aβ protofibrils.	Infusion responses, effusions, amyloid imaging anomalies, and brain hemorrhage [12].

MATERIALS AND METHODS

Plant material

Fresh resin of *Commiphora weightii* was purchased from local market in Guntur in the state of Andhra Pradesh. The plant material was taxonomically identified and authenticated by Dr. P. Satya Narayana Raju garu M.Sc., M.Phl., Ph.D from Department of Botany and Microbiology, Acharya Nagarjuna University, Nagarjuna Nagar, AP on 2nd Dec. 2022. The material was powdered using mechanical grinder and then passed through 40 mesh sieve to get uniform powder that is preserved in air tight container.

Preparation of the extract

10gm of powdered resin with 100ml of water was done for 24hrs on water bath shaker (150rpm) at 60°C. The solution was filtered through muslin cloth and then refiltered through Whattman's filter No.1. The stock solutions of *Commiphora weightii* extract (AECW) was used for the nootropic activity **[13]**.

Animals

Male albino wistar rats were used for this experiment as estrogen (female sex hormone) was discovered to show effect on the memory. Animals were obtained from disease free animal house. Wistar rats weighing 150-250g were taken for the study. The animals have free access to food and water, and they were housed in a natural (12h each) light dark cycle. Food feeded to animals consists of wheat flour kneaded with small amount of water and followed by drops of refined vegetable oil. The animals were habituated for 5days to laboratory conditions in advance of behavioral experiments. The experimental design was approved by IAEC committee and the laboratory animals were monitored and cared as per CPCSEA guidelines, Ministry of Forests and Environment, Govt. of India (registration No.1048/PO/Re/S/07/CPCSEA).

Preliminary phytochemical screening

As attempt was made to observe the presence and absence of diverse phytochemical constituents in AECW, viz., flavonoids (Shinoda test), tannins (Ferric chloride test), steroids (Liberman-Burchard's test), diterpenoids (Salkowski test), carbohydrates (Molisch test) and alkaloids (Wagner's test) according to standard methods.

Acute toxicity studies

The acute oral toxicity studies were done according to OECD 423 guidelines. The study was conducted on male albino wistar rats weighing between 150-250g and was divided into 5 groups. Each group contain 5 rats. They were fasted overnight and maintained with water *ad libitim*. The selected functional foods were administered at a level of 2000mg per kg body weight. Standard diet and water were

continued during the study as usual. The doses of selected herbal isolate were fixed based on acute toxicity studies.

Drugs

The drugs used in the study are Donepezil, Scopolamine injection (Dr. Reddy's Laboratories).

Vehicle

The plant extract (AECW) was administered orally to rats. Donepazil was dissolved in sterile water and Scopolamine in normal saline and injected i.p to rats. Volume of oral administration and i.p. injection was 1ml/1000g of rat.

Drug treatment

In the current study, the rats were split into five different groups for testing nootropic activity. Each group consists of five animals. Group I is represented as Control and administered only saline. Group II is represented as negative control and administered only Scopolamine (20mg per kg i.p). Group III is represented as Standard and administered Scopolamine (20mg per kg i.p) and Donepezil (5mg per kg p.o). Group IV and Group V are represented as Test and administered Scopolamine (20mg per kg p.o) and different doses of extract AECW (50mg and 100mg per kg p.o). All control group animals received vehicle (saline) for 7 consecutive days. Group IV and Group V received AECW orally for 7 successive days using water as solvent. Group II and Group III received scopolamine only on 1st day (single induction). 90 minutes after administration of last dose (on 5th and 6th days) rats were exposed to training sessions by using Y-maze, Labyrinth maze, Elevated plus maze. Retention of memory was recorded on 7th day. Amnesia was caused by Scopolamine (20mg per kg i.p.)

Y-Maze

It consists of 3 arms with 40cm length, 35cm height and 12cm width that are labeled as A, B, C. Rats were placed at one end and allowed to move freely through the maze during a 5 min session. Transfer latency time was examined visually by recording the pattern of entrance into each arm in the maze for each rat. Transfer latency is defined as successive entries into three arms on overlapping triplet set (i.e., LSS, SSL and so on). Accordingly, the transfer latency was calculated **[14]**.

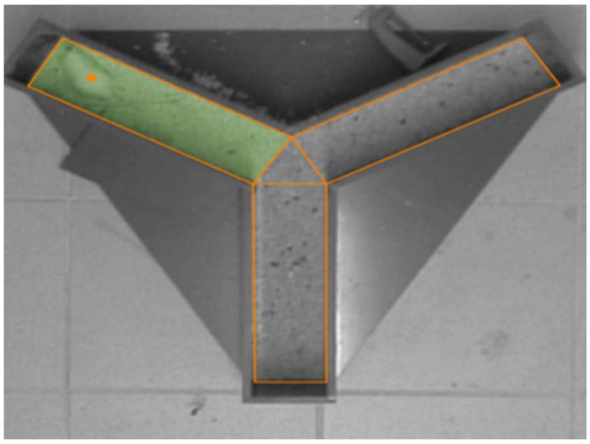

Figure 1: Y-MAZE
https://www.any-maze.com/applications/y-maze/

Labyrinth maze

It is one the finest devices for studying spatial memory in animals. It consists of 3 chambers. Prior to the experiment all the animals are allowed to habituate the equipment for the good results. One the first day (i.e, sixth and seventh day of drug treatment), the rats were allowed to enter into the Chamber A and the door was opened to facilitate the entry of the animal into the next chamber to prevent back entry the door was closed. Time required for the animal to reach the Chamber B (where the food is placed) from the Chamber A was noted on first day (training session) for each animal. Each animal was permitted to explore the maze for 3 min with all the doors opened before returning to its home cage. Retention of this learned task (memory) was examined **[15]**.

Figure 2: Labyrinth Maze

Elevated plus maze

It involves 2 open arms (16cm x 5cm) and 2 enclosed arms. The maze was elevated at the height of 25cm. Animals were located associate distinct bases at the tip of associate degree open arm facing removed from central platform and the time took by them to move from there to either one of the closed arm transfer latency was documented. The retention of the learned task was examined on 7th day **[16]**.

Figure 3: Elevated Plus Maze
https://www.researchgate.net/figure/Elevated-plus-maze-This-apparatus-consists-of-a-cross-shaped-platform-elevated-50cm_fig1_235405881

RESULTS AND DISCUSSION:

Preliminary phytochemical screening:

The result of phytochemical screening shows the presence of Carbohydrates, Flavonoids, Tannins, Alkaloids, Diterpenoids and Steroids by using standard method.

FOURIER TRASFORM INFRA-RED (FTIR) SPECTROSCOPY (Table 3)

Y-MAZE

Effect on transfer latency (using Y-maze):

Time required to reach the rewarded chamber is described as latency and it explains about memory of animals on 7th day. AECW 100mg per kg administered orally for 7 days have shown significant effect on transfer latency **(Table 4, Figure 4)** equal to standard

donepazil (5mg per kg). Amnesia was induced by Scopolamine (20mg per kg i.p) have increased the latency scores of animals which were significantly reduced by standard Donepazil (5mg per kg) and AECW (50mg and 100mg per kg p.o). $p< 0.0001$ represents significant reduction latency scores which in turn represents raise in memory level which were decreased by scopolamine (20mg per kg i.p).

Effect on transfer latency (using Labyrinth maze):

Time required to reach the rewarded chamber is described as latency and it explains about memory of animals on 7th day. AECW 100mg per kg administered orally for 7 days have shown significant effect on transfer latency equal to standard donepazil (5mg per kg). Amnesia was induced by Scopolamine (20mg per kg i.p) have increased the latency **(Table 5, Figure 5)** scores of animals which were significantly reduced by standard Donepazil (5mg per kg) and AECW (50mg and 100mg per kg p.o). $p < 0.0001$ represents significant reduction latency scores which in turn represent raise in memory level which were decreased by scopolamine (20mg per kg i.p).

Effect on transfer latency (using Elevated plus maze):

Time required to reach the rewarded chamber is described as latency and it explains about memory of animals on 7th day. AECW 100mg per kg administered orally for 7 days have shown significant effect on transfer latency **(Table 6, Figure 6)** equal to standard donepazil (5mg per kg). Amnesia was induced by Scopolamine (20mg per kg i.p) have increased the latency scores of animals which were significantly reduced by standard Donepazil (5mg per kg) and AECW (50mg and 100mg per kg p.o). $p< 0.0001$ represents significant reduction latency scores which in turn represent raise in memory level which were decreased by scopolamine (20mg per kg i.p). Might be due to the presence of alkaloids and steroids in AECW **(Table 2)**.

Table 2: Phytochemical screening results

Phytochemical constituents	Presence(+)/Absence(-)
Alkaloids	+
Glycosides	-
Carbohydrates	+
Flavonoids	+
Tannins	+
Resins	-
Volatile oils	-
Fixed oils	-
Diterpenoids	+
Steroids	+

Note: + indicates – Present; indicates- Absent

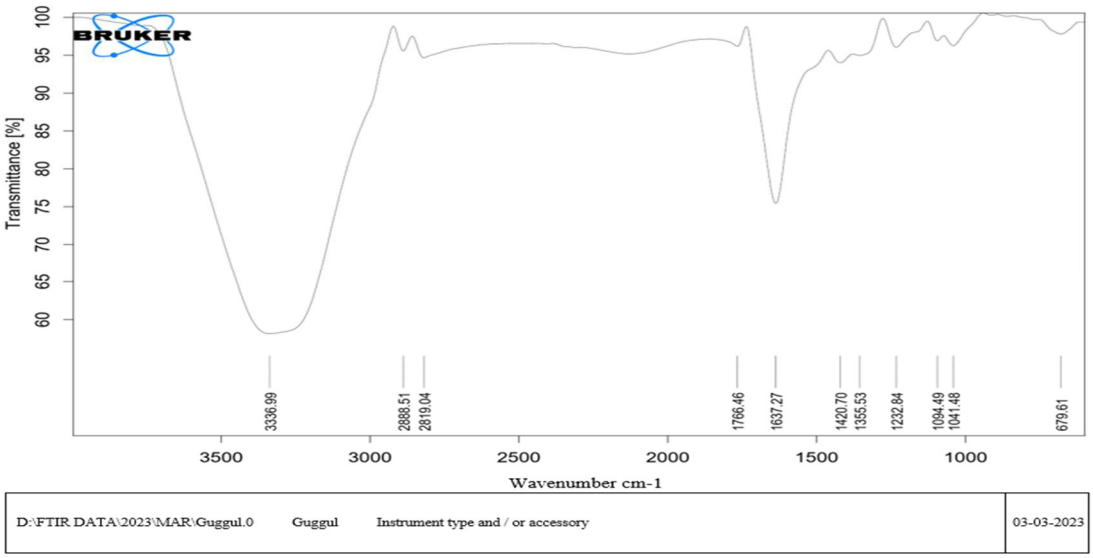

Table 3: IR spectra of compound isolated from aqueous extract of *Commiphora weightii*

S. No.	Characteristic Band	Wave number (cm^{-1})	Reference wave number (cm^{-1})
1.	O-H	3336.99	3200-3500
2.	C-H (alkane)	2888.51	2840-3000
3.	C-H (aromatic)	1766.46	1650-2000
4.	C=C	1637.27	1626-1662

Table 4: Spatial working memory activity of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using Y-maze method

GROUP	DAY-7
Control	11.4 ± 0.41
Negative control	15.8 ± 0.30
Standard	7.2 ± 0.30 **
AECW (50mg)	9.6 ± 0.2
AECW (100mg)	8 ± 0.44 **

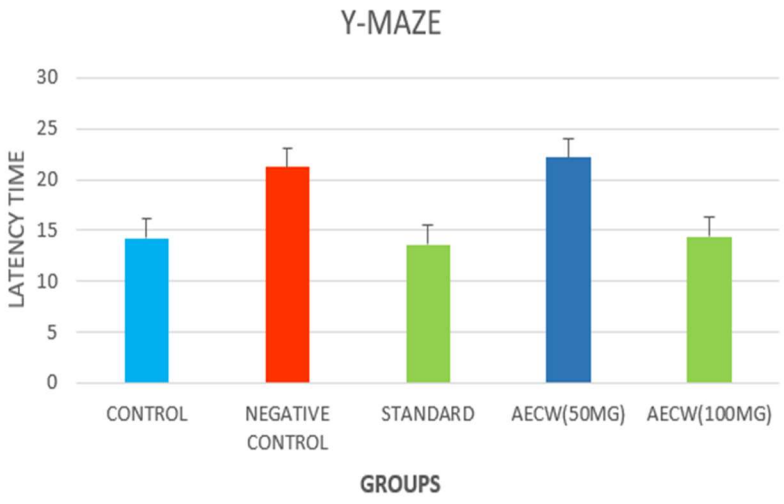

Figure 4: Spatial working memory activity of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using Y-maze method.1
Data represent Mean ± SEM, (n=5). One-way Anova. Significance was shown at p<0.0001 when compared with control. **denotes p < 0.0001 which is compared with control group of rats

Table 5: Spatial working memory activity of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using labyrinth maze on 1st day

GROUPS	DAY-1	DAY-6	DAY-7
Control	85.6 ± 1.14	79.6 ± 1.54	77.4 ± 1.28
Negative control	111 ± 1.87	118.6 ± 0.98	115.6 ±2.02
Standard	68.4 ± 1.11	66.2 ± 1.32	63.4 ± 1.25 **
AECW (50mg)	74.8 ± 1.04	74 ± 1.15	74.8 ± 1.27
AECW (100mg)	66.2 ± 1.24	64 ± 0.68	64.2 ± 1.16 **

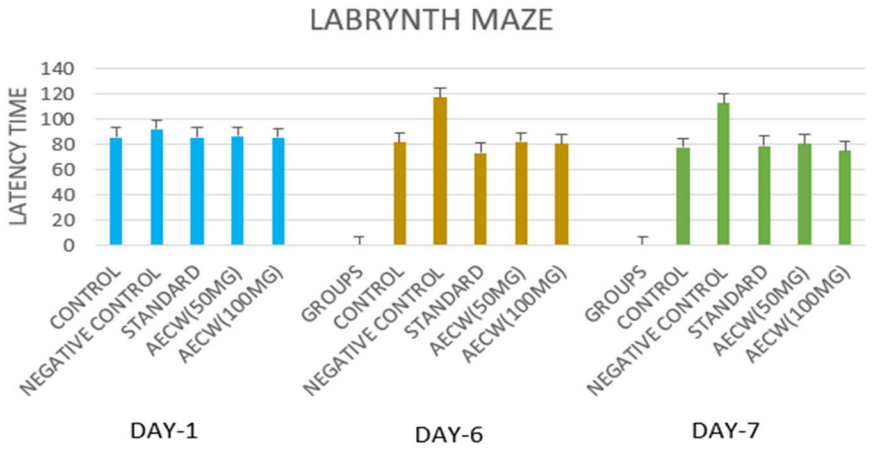

Figure.5: Spatial working memory activity of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using labyrinth maze method on 1st, 6th and 7th day.
Data represent Mean ±SEM, (n=5). One way Anova. Significance was shown at p<0.0001 when compared with control. **denotes p < 0.0001 which is compared with control group of rats.

Table 6: The learning and memory enhancement of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using Elevated Plus Maze method.

GROUP	DAY-7
Control	39.8 ± 15.97
Negative control	27.6 ± 0.41
Standard	10.6 ± 0.32 **
AECW (50mg)	13.6 ± 0.41
AECW (100mg)	12.8 ± 0.30 **

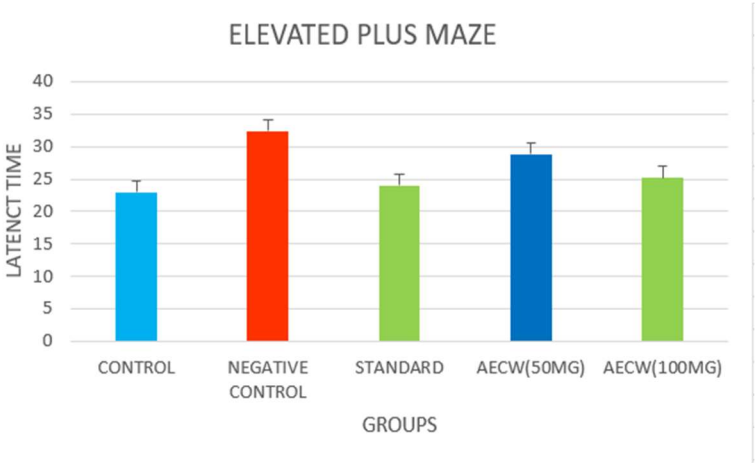

Figure 6: The learning and memory enhancement of resin extract of *Commiphora weightii* against scopolamine induced memory impairment in Albino Wistar rats by using Elevated Plus Maze method.
Data represent Mean ±SEM, (n=5). One-way Anova. Significance was shown at p<0.0001 when compared with control.denotes p < 0.0001 which is compared with control group of rats**

DISCUSSION

The AECW has shown significant effect at a dose of 100mg per kg which is similar to standard donepazil 5mg per kg. Due to presence of phytochemical constituents called flavonoids. Investigation reveals AECW has nootropic activity at a standard level useful to mankind. Y maze, labyrinth maze and elevated plus maze are standard methods for evaluating nootropic activity.

Statistical Analysis:

From the results of FTIR, we identified the presence of guggulsterone in our compound and the above graphical results were denoted as standard error of mean (SEM). Data was analyzed using one way ANOVA. p values $p< 0.0001$ were considered as statistically significant.

6. CONCLUSION

The AECW has shown significant effect dose of 100mg per kg which is similar to standard Donepazil 5mg per kg due to presence of phytochemical constituents like Flavonoids, tannins, alkaloids, steroids. The investigation shows AECW has nootropic activity and at a standard level useful to mankind. Behavioral models for studying drugs or conditions that effect cognitive process were standardized and evaluated by using the aqueous extract of *C.weightii* (AECW) has shown significant nootropic activity by Y-maze, elevated plus maze, labyrinth maze.

ACKNOWLEDGEMENT:

This work would not have been possible without the services provided by Chalapathi Institute of Pharmaceutical Sciences (Autonomous) and so we are extremely grateful to the institution. We are grateful to research guide D.Nalini Devi and N. Rama Rao for their great support.

CONFLICTS OF INTEREST:

Authors declare no conflict of interest.

REFERENCES

[1] Tortora's Principles of Anatomy and Physiology. (2017); 15th edition, pg.no; 355, 359.

[2] George DeMaagd and Ashok Philip, Parkinson's disease and Its Management Part 1. Pharmacy and Therapeutics. August 2015; 40: 8.

[3] Emilia M.Gatto *et al*., Huntington disease: Advantages in the understanding of its mechanisms: Clinical Parkinsonism and Related Disorders May 2020; 3:4.

[4] Adele D' Amico *et al*., Spinal muscular atrophy: Orphanet Journal of Rare Diseases 2011; 6:71.

[5] Tetsuo Ashizawa *et al*., Ataxia: Continum 2016; 22(4): pg.no 1208-1209.

[6] Sara Zarei *et al*., A comprehensive review of amyotrophic lateral sclerosis: Surgical Neurology International. 2015, 6:171.

[7] Dementia. World Health Organization. https://www.who.int/news-room/fact-sheets/detail/dementia.2022.

[8] James M. Ritter *et al*., Rang and Dale's Pharmacology. (2020); 9th edition, pg.no; 519, 520.

[9] Chang Yell Shin *et al*., The Effects of Donepazil, an Acetylcholinesterase Inhibitor, on Impaired Learning and Memory in Rodents: Biomol Ther 2018; 26(3): pg.no 274, 275.

[10] Gordon K Wilcock *et al*., Efficacy and safety of galantamine in patients with mild to moderate Alzheimer's disease: multicentre randomized, controlled trail BMJ December 2000; 321:1.

[11] Thomas Muller, Rivastigmine in the Treatment of patients with Alzheimer's disease: Neuropsychiatric Disease and Treatment 2007; 3(2): pg.no 211.

[12] Samira Abid Beshir *et al*., Aducanumab Therapy to treat Alzheimer's disease; A narrative review: International Journal of Alzheimer's Disease 2022: pg.no 1-2.

[13] Amir Abbas Tahami Monfared *et al*., Long-Term Health Outcomes of Lecanemab in Patients with Early Alzheimer's Disease Using Simulation Modeling: Neurol Ther 2022;11:1.

[14] Bhardwaj *et al*., Protective Effects of *Commiphora weightii* in Metabolic Activity of Streptozotocin (STZ) Induced Diabetes in Rats: Journal of Diabetic and Endocrinology 2014; 5(3): pg.no 20.

[15] Ming Ru *et al*., Association between Y-maze acquisition Learning and Major Histocompatibility Complex Class II Polymorphisms in Mice: BioMed Research International 2018; pg.no 2 Article ID: 6381932.

[16] K Ashok kumar *et al*., Evaluation of Memory Enhancement Activity of Rodents in Spatial Arrangement by using Labyrinth maze: International Journal of Pharma Research and Health Sciences 2014; 2(2): pg.no 104.

[17] Dinesh Dhingra *et al*., Memory-Enhancing Activity of Palmatine in Mice Using Elevated Plus Maze and Morris Water Maze: Advances in Pharmacological Sciences 2012; pg.no 2 Article ID: 357368.