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Memory Enhancing Activity of *Actinidia Delisiosa* Extract on Bilateral Common Carotid Artery Occlusion / Reperfusion in Rats

Chakali Ayyanna [1]* Vamshi Krishna .D [2,] Supraja. K [3], Naga Sudha .B [4]

1.Associate Professor, Department of Pharmacology Creative Educational Society's College of Pharmacy, Chinnatekur, Kurnool, A.P- 518218

2 & 3 **.**Creative Educational Society's Department of Pharmacology, Chinnatekur, Kurnool, A.P- 518218

4. Professor, Department of Pharmaceutical chemistry, Creative Educational Society's, Chinnatekur, Kurnool, A.P- 518218

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KEYWORDS

Cognition Enhancement, *Actinidia delisiosa*,acetyl cholinesterase, malondiadehyde , Glutathion

ABSTRACT:

The current study was undertaken to investigate the effects of *Actinidia delisiosa* on learning and memory in rats. Effect of drugs on learning and memory of rats was evaluated using Y-Maze served as the exteroceptive behavioural models for testing memory. The interoceptive behavioural models were BCCAO/R-induced amnesia. The ethanol extract of EEAD was administered orally in two doses (200, and 400 mg/kg) for 14 successive days to different groups of rats. In rats, EEAD (200 and 400 mg/kg, p. o.) dramatically improved memory performance.Various biochemical parameters such as acetylcholine esterase (AchE), malondiadehyde (MDA), and Glutathion level (GSH) from brain homogenate were evaluated on 14th day. In BCCAO/R-induced cognitive impairment, pre-treatment with Actinidia delisiosa ethanolic extract (200mg/kg p.o. and 400mg/kg p.o.) increased spontaneous behaviour in rats. In comparison to BCCAO/R-induced cognitive impairment in rats, pre-treatment with ethanolic extract of Actinidia delisiosa dramatically reduces MDA levels and enhances GSH levels.Anticholinesterase, anti-inflammatory, and antioxidant properties of EEAD may favourably contribute to its memory-enhancement effect. Therefore, EEAD fruit juice appears to be a promising candidate for improving memory, Thus, EEAD showed memory-enhancing activity in rats probably by inhibiting brain acetyl cholinesterase activity, through involvement of GABA-benzodiazepine pathway, and due to its antioxidant activity and it would be worthwhile to explore the potential of this plant in the management of Alzheimer patients. The findings from present investigation may conclude that extracts of fruits of *Actinidia delisiosa* possesses cognition enhancing property in BCCAO/R induced cognitive impairment in rats.

1. Introduction

One of the most crucial functions of the brain is memory. Memory is vital for survival because it is the process by which organisms are able to record their experiences and use this information to adapt their responses to the environment. Loss of memory and cognitive impaired functions are the major features of Alzheimer's disease (AD). Acetylcholine is present in sufficient amounts in the neo cortex to improve memory loss and learning deficiencies. Etiological variables have been identified as reduced cholinergic firing in the brain, an increase in oxidative stress, hypercholesterolemia, and neuroinflammatory responses. It contributes to memory loss. The central cholinergic system is involved in cognitive functions and plays an important role in learning and memory for humans and animals [1] .In India 4.1 million of people is living with dementia. There are an estimated 5.3 million Americans of all ages with Alzheimer's disease. In 2015, an estimated 7, 00.000 Americans age ≥65 years will die with AD, and many of them will die from complications caused by AD [2].

The most common cause of dementia in the elderly is Most likely Alzheimer's disease (AD), a chronic, degenerative, and incapacitating condition of the biological brain that disrupts a variety of cortical processes, including memory, judgement, orientation,

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understanding, learning, and language [3]. Amnesia can be brought on by biological or neurological factors (damage to the brain through physical injury, neurological disease or the use of certain drugs),or from functional or psychogenic causes (psychological factors, such as mental disorder, post-traumatic stress or psychological defence mechanisms)[4] .These deposits of amyloid are referred to as “plaques” and cause the brain cells to shrivel up and form “tangles”, which in turn lead to changes in the brain structure and cause the brain cell to die. The formation of plaques and tangles also prevents the production of some important brain chemicals, called neurotransmitters. Brain cell death causes the brain to shrink over time.As we all know, India, with its vast biodiversity and knowledge-rich ancient traditional medical systems such as Ayurveda, Siddha, Unani, and local health traditions, provides a strong base for the utilization of a large number of plants in general healthcare of the people [5] . The scientific name for the kiwi is *Actinidia deliciosa* [6]. It is also known as Chinese gooseberry, this fruit is popular all over the world, due to its high nutritional value, high content of vitamin C, in addition to excellent organoleptic qualities, in connection with its adaptability. Many researches [7-8] indicate that kiwi has more nutrients than other widely consumed fruits and emphasises its therapeutic effects in terms of healthy metabolism, iron content, digestive potential, antioxidant properties, immune function and also protective effects against coronary heart disease. Kiwi fruit, as a source of ascorbic acid and polyphenols, helps reduce the risk of artery hardening, cardiovascular disease, and some types of cancer [9] in irritable bowel syndrome [10] and also protects cells in vitro oxidative damage to DNA.The present study is based on hypothesis that fruits of *Actinidia delisiosa*can show cognition enhancing activity.

2. Materilas and Methods

Collection of fruit: The *Actinidia deliciosa*, were purchased from the local market of Kurnool. The fruit was authenticated by the botanist from Govt. Degree College for men, Kurnool.

Preparation of ethanolic extract of *Actinidia deliciosa:*
The fresh fruit of *Actinidia deliciosa* was prepared the help of juicer without addition of water. The fresh fruit was chopped into a small pieces and juice was collected. The juice was filtered with sterile cloth and the resultant filtrate was used mixed with 60% ethanol (v: v = 1: 2) and sonicated for 20 minutes at 40°C Supernatant was collected after centrifugation for 20 minutes at 4000 rpm. As the ethanolic extracts of kiwi fruit. Into make certain that the kiwifruit's components were the same in future studies [11].

Experimental animals: 30 Wistar rats weighing 180-220 g were employed in this study. They were maintained in separate polypropylene cages in a typical laboratory setting with sunlight, temperature, and ratio. Standard rat pellets were given to the animals, which were also naturally drinkable. The experimental protocol was approved by the Institutional Animal Ethical Committee of Creative Educational Society College of Pharmacy (IAEC/CESCOP/2019-OCT-11)

Acute toxicity studies: Acute oral toxicity study in experimental rats was carried out as per OECD-423 guidelines. Four doses (5, 50, 300, 2000 mg/kg body weight) ethanolic extract of *Actinidia deliciosa* were administered orally to groups containing three animals of the same age group and weight. The animals were regularly monitored for 1 hour continuously and then hourly for 4hr and finally after every 24 hr up to 14 days for any symptoms of toxicity and mortality, Alertness, Grooming, Touch response, Pain response, Limb position, Grip strength, Urination and Diarrhoea [12].

Experimental design:

30 animals were grouped into four, having six in each group (n=6). Group I treated as Normal, Group II treated as Sham control, Group III treated as MBCCAO/R and Group IV received the EEAD 200mg/kg, and Group V received EEAD 400 mg/kg for 14 days. After the treatment for 14 days, all five groups of animals underwent the antioxidant assessment tests. Then, Multiple bilateral common carotid artery occlusion/ reperfusion was performed to isolate the brains of the animals on 14th day of experiment and homogenate was prepared using ice-cold phosphate-buffered saline solution and stored at refrigerant for anti-oxidant parameter

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Determination of Neurobehavioral Studies:

Spontaneous Alteration Behaviour by Y-Maze Test:

We examined continuous spontaneous alternation behaviour using the Y-maze apparatus. The Y-maze apparatus was made of black plastic with three arms (36cm×7cm×13cm) extending from a central platform at1200.Each animal was placed at the end of one arm and allowed to move freely through the Y-maze during asessionlasting for 8min.Arm entry was defined as the entry of 4 paws into one arm. The sequence of arm entries was recorded visually. Alternation was defined as multiple entries into the 3 arm (A, B or C) (e.g., C–A–B, B–C–A, and A–B–C) on over lapping triplet sets. The percentage of spontaneous alternation was calculated as the ratio of the actual to possible alternations (definedasthetotalnumberofarmentriesminus2), multiplied by100: as shown in the following equation [13]

% Alternation=((number of alternations)/(total number of armentries-2))×100.

Estimation of anti oxidant parameters:

Estimation of malondialdehyde: 300 microns of 10% trichloroacetic acid (TCA) were added to 150 µL of each sample and centrifuged at 1000 rpm for 10 minutes at 4^0C. Around 300 µL of the supernatant was incubated with 300 µL 0.67% thiobarbituric acid at 100^0C for 25 minutes. The blend was cooled for 5 minutes and the thiobarbituric acid reactive substances (TBARS) concentration as pink stains was measured in a spectrophotometer at 535 nm [14].

Assay of superoxide dismutase (SOD)

Procedure: 0.1ml of homogenate brain tissue was added to 1.2ml of 0.052M sodium pyrophosphate buffer pH (9.3) followed by addition of 0.1ml of 196µM Phenazinemethosulphate solution, 0.3ml of Nitrobluetetrazolium (300 µM) solution, 0.2ml of NADH (790 µM) solution nicotinamide adenine dinucleotide (NADH) – phenazinemethosulphate,Nitrobluetetrazolium formation complex [15].

Mixture was incubated for 90sec at 30°C and the reaction was stopped by the addition of 0.1ml glacial acetic acid; add each 4ml n. butanol. The colour formed as the end point of the reaction was extracted into the butanol layer and measured at 520 nm. A control was prepared using 0.1ml of distilled water and 0.1ml of homogenate tissue.

Assay of catalase (CAT)

Principle: A ubiquitous enzyme called catalase is present in almost all living things that are exposed to oxygen (such as bacteria, plants, and animals). It accelerates hydrogen peroxide's breakdown into water and oxygen. It is a very important enzyme in protecting the cell from oxidative damage by reactive oxygen species (ROS) [16] .

Procedure: The tissue was homogenized in isotonic buffer (pH - 7.4) and centrifuged at 1000rpm for 10 minutes. 0.2ml of tissue homogenate added to 1.9ml of 50µM phosphate buffer. To this mixture 1ml of freshly prepared 30 µM hydrogen peroxide was added and was measured spectrophotometricaly at 240 nm.

Estimation of gluthathione reductase (GSH)

To 2 ml of the tissue homogenate and KCl mixture 4 ml of cold distilled water and 1 ml of 50% TCA. were added, and the mixture was centrifuged for 15 minutes at 3000 rpm.After 15minutes 2 ml of the supernatant was collected and to that mixture added 4 ml of 0.4 M Tris buffer (pH 8.9) and 0.1 ml of 0.01 M DTNB , At 412 nm, the absorbance was measured in comparison to a blank reagent. Tissue homogenate was replaced with 2 cc of distilled water for blank readings [17].

Total GSH was calculated using the formula: Co=(A*D)/E Where, A is absorbance at 412 nm, D is dilution factor, and E is the molar extinction coefficient (C=13,000 M−1 CM−1); Co is the concentration of GSH.

Estimation of neurotransmitters:

Estimation of dopamine **:** 0.05 ml 0.4 M HCl and 0.1 ml EDTA / Sodium acetate buffer (PH6. 9) were then added, followed by 0.2 ml of the (DTNB). (39.6 mg were dissolved in 10 ml pH 7.0 phosphate buffer (O-1 M)) The reaction results in production of 5-thio-2-nitrobenzoate that has yellow color due to the shift of electrons to the sulfur atom [19].

The animals were sacrificed, and their brains were swiftly removed and stored in ice-cold saline. In 0.1M Phosphate buffer, the tissues were weighed and homogenized (pH 8). 4ml of homogenate is added to a cuvette containing 2.6ml of phosphate buffer (0.1M, pH

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8) and 100l of DTNB. The contents of the cuvette were thoroughly mixed, and absorbance was measured in a spectrophotometer at 412nm. The basal reading was taken when absorbance reached a stable value. 20l of acetylthiocholine substrate was added, and the change in absorbance was measured. Change in the absorbance per added. The solution was then heated to 100°C for 6 minutes, and when the sample returned to room temperature, the spectrofluorimeter was used to read the excitation and emission spectra. Dopamine measurements were taken at 330-375 nm [18].

Estimation of Acetyl cholinesterase (AChE) Ellman's method: The enzyme Acetyl cholinesterase (AChE) is involved in cholinergic neurotransmission. Acetylcholine is degraded, bringing the neurotransmission process to a close. The most popular test is based on Ellman's technique and uses acetylthiocholine and 5, 5'-dithio-bis-2-nitrobenzoic acid as an alternate substrate minute was determined. Protein estimation was done using folin's method. Aqueous phase and 0.1 ml of the iodine solution (0.1 M in ethanol) for oxidation.After 2 minutes, the reaction was stopped by adding 0.1 ml Na2SO3 solution. After 1.5 minutes, 0.1 mL of acetic acid is

Statistical Analysis: The data were expressed as mean ± S.E.M from 6 animals [n=6]. The results were subjected to statistical analysis by using Unpaired t-test to calculate the significance difference if any among the groups. P<0.05 was considered as statistical significance using Graph Pad Prism Software.

3. Results and discussion

Table 1: Percentage yield of ethanolic extract of *Actinidia deliciosa*

SL.NO	Nameof theextract	Nature	Colour	Percentageyield (%)
1	Ethanolicextractof *Actinidiadeliciosa*	Sticky	Reddishbrown	5.2 %

To determine the presence of different phytoconstituents, a preliminary phytochemical examination of *Actinidia deliciosa* ethanolic extract was conducted. It produced the following findings

Table 2 : Preliminary phytochemical analysis

Sl.No	Nameofthetest	Results
1	Alkaloids	+
2	Glycosides	+
3	Tannins	+
4	Flavonoids	+
5	Steroids	+
6	Terpenoids	-
7	Phenolics	+

(+)Indicates present, (-)Indicates absent

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Table 3: Effect of *EEAD* Spontaneous Alteration Behaviour on MBCCAO/R induced cognitive impaired rats

Group	Drug treatment	Spontaneous alteration behaviour
1	**Normal**	82.71± 1.691
2	**Sham control**	71.80 ± 1.900
3	**MBCCAO/R**	44.30 ± 1.536###
4	**EE*AD* 200mg/Kg/P.O**	66.45 ± 2.473**
5	**EE*AD* 400mg/Kg/P.O**	74.42 ± 1.951***

All values are expressed as mean ±SEM, n=6. Data were analyzed by one- way ANOVA followedbyDunnett'stest.###p<0.001,**p<0.01,***p<0.05,NormalvsMBBCAO/R,###p<0.001,**p<0.01,***p<0.05,NormalvsShamcontrol,###p<0.001,**p<0.01,***p<0.05,MBBCAO/RvsTreatmentgroups.(i.e.:EEAD200mg/Kg/P.O or EEAD400mg/Kg/P.O)

Table 4: Effect of *EEAD* on brain LPO, SOD, CATALASE, GSH levels on MBCCAO/R induced cognitive impaired rats

All values are expressed as mean ±SEM, n=6. Data were analyzed by one- way ANOVA

GROUP	DRUG TREATMENT	LPO (n MMDA/mgprotein)	SOD (µ/mgprotein)	CATALASE (U/mgprotein)	GSH (µ/mgprotein)
I	**Normal**	6.81 ± 0.67	18.57 ± 1.088	0.0051± 0.0002	0.178± 0.008
II	**Sham control**	7.892 ± 0.64	16.4 ±0.94	0.0047 ± 0.0003	0.0168± 0.007
III	**MBCCAO/R**	13.89±0.64###	9.48 ± 0.95###	0.0010 ± 0.0002###	0.081± 0.007###
IV	**EE*AD* 200mg/Kg/P.O**	9.53 ± 0.61**	14.92 ±0.56**	0.0020 ± 0.0002**	0.010 ± 0.006**
V	**EE*AD* 400mg/Kg/P.O**	7.25 ± 0.35***	17.23 ±0.63**	0.0043 ± 0.0007***	0.160 ±0.005***

followedbyDunnetts'stest.###p<0.001,**p<0.01,***p<0.05,NormalvsMBBCAO/R,###p<0.001,**p<0.01,***p<0.05,NormalvsShamcontrol,###p<0.001,**p<0.01,***p<0.05,MBBCAO/RvsTreatmentgroups.(i.e.:EEAD200mg/Kg/P.OorEEAD400mg/Kg/P.O)

Table 5: Effect of *EEAD* on brain Dopamine, AChE levels on MBCCAO/R induced cognitive impaired rats.

GROUP	DRUGTREATMENT	DOPAMINE	AChE (µmoles/minute/mg)
I	**Normal**	9.208 ± 0.343	0.024 ± 0.006
II	**Sham control**	6.697 ±0.33	0.019± 0.007
III	**MBCCAO/R**	4.697 ±0.338###	0.025 ± 0.006###

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IV	**EEAD200mg/Kg/P.O**	5.557 ±0.970**	0.036 ± 0.006**
V	**EEAD400mg/Kg/P.O**	7.850 ±0.510**	0.028 ± 0.006***

All values are expressed as mean ±SEM, n=6. Data were analyzed by one- way ANOVA followedbyDunnetts'stest.###p<0.001,**p<0.01,***p<0.05,NormalvsMBBCAO/R,###p<0.001,**p<0.01,***p<0.05,NormalvsShamcontrol,###p<0.001,**p<0.01,***p<0.05,MBBCAO/RvsTreatmentgroups.(i.e.:EEAD200mg/Kg/P.OorEEAD400mg/Kg/P.O)

Discussion:

Present study investigated the different doses of ethanolic extract of fruit of *Actinidia deliciosa*, on multiple bilateral common carotid artery produced cognitive impairment in rats utilizing behavioural and pharmacological paradigm .MBCCAO model is a relatively rapid and easy rat model that can be used to produce temporary or chronic cerebral ischemia [20]. Rats usually exhibit white matter damage accompanied by cognitive impairments resembling those associated with stroke in humans This model is relatively easy to use, and training researchers on this rat model decreases the mortality rate to<2%. MBCCAO technique performed by ligating bilateral common carotid artery of rat for a few minutes, followed by the release of the ligature (reperfusion period). This ligating and reperfusion activity of the artery causes damage to rat's brain cells.

Spontaneous alternation behaviour using the Y-mazes apparatus. The Y-maze apparatus was made of black plastic with three arms (36cm×7cm×13cm) extending from a central plat form at 120^0.Each animal was placed at the end of one arm and allowed to move freely through the Y maze during a session lasting for 8min. Arm entry was defined as the entry of 4 paws intone arm. The sequence of arm entries was recorded visually. Alternation was defined as multipl eentries in to the 3 arm (A,BorC) (e.g.C–A–B,B–C–A,and A–B–C) on overlapping triplet sets.

One of the important mechanisms in the development and progression of AD is oxidative stress. In the present study, BCCAO/R decreased SOD (Table 5) and GSH (Table 4) and increased the MDA (Table 4) and protein carbonyl levels in the rat hippocampus homogenates. It has been shown that MBCCAO/R administration causes neurochemical changes in the brain as well as changes in the oxidative status of the brain [21]·Consequently, the ethanolic extract treatment restored the antioxidants status as evidenced by an increase of SOD (Table 4), and GSH (Table 5) while the levels of MDA(Table 4) and protein carbonyl significantly decrease which supports its antioxidant property. Lipid peroxidation has been identified as a key mechanism of brain damage. The mechanism involves an oxidation process that produces additional radical species as well as toxic by-products that can be harmful to the host system [22]· Polysaturated lipids are especially susceptible to this type of damage when in an oxidizing environment and they can react to form lipid peroxides. [23].As lipid peroxides are inherently unstable, they further break down to produce a variety of other chemicals, such as reactive carbonyl compounds [24]·In the present study we observed increase in the tissue MDA activity in ischemic reperfused brain when compared with the sham groups and the results were in agreement with previous studies. Treatment with EEAD (400mg/kg p.o) provided memory enhancing when compared with the MDA (Table 4) activity observed. Reactive species can be decreased or eliminated by a number of enzymatic and no enzymatic antioxidant mechanisms. One of the most significant antioxidant enzymes is SOD, which catalyses the dismutation of superoxide anion (O-2) into hydrogen peroxide and molecule oxygen [25]. In the present study, SOD and CAT activity decreased in the ischemic reperfused group compare to sham group and the results were in agreement with previous studies[25].This may be due to an excessive formation of superoxide anions. Decrease in SOD activity can result in the decreased removal of superoxide anions, which can be harmful to the brain. The decrease in enzyme levels may be explained by the possibility that too many superoxide anions may inactivate SOD, which will then inactivate the H_2O_2-scavenging enzyme. The reduced SOD and CAT activity were increased by administration of the EEAD significantly when compared with the O/R group (Table 4).

Acetylcholine is considered to be one of the important neurotransmitter involved in the regulation of cognitive functions. Cognitive dysfunction has been shown to be associated with impaired cholinergic transmission and the

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facilitation of central cholinergic transmission resulting in improved memory.Moreover,selective loss of cholinergic neurons in certain brain parts appeared to be a characteristic feature of senile dementia [26].The degeneration and dysfunction of cortical cholinergic neurons is closely associated with cognitive deficits of AD[27].Thus, the drugs which enhance cholinergic function can be used for treatment of dementia closely related to AD.EEAD200,400mg/Kg administered for14 days significantly improved memory of rats. Physostigmine, a cholinesterase inhibitor, could improve memory in normal subjects [28] as well as in patients with dementia [29].

These results could suggest that the increase of behavioural scores in the Y-maze (Table 3), and the along with the decrease of AChE activity (Table 4), And the increase of dopamine levels (Table 4), and also the MDA content and protein carbonyl level could be correlated with the involvement of the ethanolic extract in memory enhancing against BCCAO/R induced oxidative stress generation in the rat hippocampus

Conclusion

The results of present study concluded that ethanolic extract of *Actinidia deliciosa* (*EEAD*) possessed cognitive enhancement activity against MBCCAO/R induced cognitive impairment in rats. The data obtained from the study are consistent with the concept that ligating and reperfusion activity of the artery causes damage to rat's brain cells from MBCCAO/R play a major role in inducing cognitive impairment. *EEAD* restored the levels of behavioural and antioxidant parameters (CAT, SOD, GSH, and LPO) and neurotransmitters (Dopamine, AChE) levels to normal. Hence, it can be concluded that 400 mg/kg shows significant cognitive enhancement activity when compared to 200mg/kg may be due to the active constituent's present flavonoids in *EEAD*.Further studies are recommended to elucidate the mechanism of the cognitive enhancement activity of ethanolic extract of *Actinidia deliciosa*

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