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Phytochemical And Pharmacological Evaluation Of Selected Medicinal Plant For Anti-Alzheimer's Activity In Experimental Animals

Munesh Mani[1], Ritika Sharma[2], Danish khan[3], Farhad F Mehta[4], Richa Saxena[5], Preeti Kumari Diwakar[6], Waghulde Sandeep[7], Pydiraju Kondrapu[8]*

[1] Professor, SOS School of Pharmacy, Faculty of Pharmacy, IFTM University, Moradabad, Uttar Pradesh - 244102

[2] Assistant Professor, University Institute of Pharma Sciences, Chandigarh University, Gharuan, Mohali, Punjab -140413

[3] Assistant Professor, Sir Madanlal Institute of Pharmacy, Alampur Hauz, Agra Road, Etawah, Uttar Pradesh – 206001

[4]Assistant Professor, School of Pharmaceutical Sciences, UTD, RGPV University, Bhopal, M.P. 462033

[5]Assistant Professor, School of Pharmaceutical Sciences, IFTM University, Moradabad

[6]Assistant Professor, Radha Govind Institute of Pharmacy, Chandausi District Sambhal, Uttar Pradesh - 204411

[7]Professor, LSHGCT'S Gahlot Institute of Pharmacy Koparkhairane, Navi Mumbai Maharashtra - 400709

[8]Associate Professor, Adarsa College of Pharmacy, G. Kothapalli, Andhra Pradesh - 533285

***Corresponding Author:**

Dr. Pydiraju Kondrapu

Designation and Affiliation: Associate Professor, Adarsa College of Pharmacy, G. Kothapalli, Andhra Pradesh - 533285

Email ID: pydikondrapu604@gmail.com

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ABSTRACT

As the world's population ages, Alzheimer's Disease (AD), the most common cause of dementia worldwide, becomes more common. Since AD is the main cause of dementia and neurodegenerative diseases, it is one of the biggest healthcare concerns of the twenty-first century. Dementia is the term used to describe an acquired loss of cognitive function across multiple cognitive domains. Understanding the mechanism of AD and available treatments may be aided by the animal experiments used in this study to ascertain whether chemical substances cause the disease. In this study, the Wistar albino rat was used to test the effectiveness of an Alcoholic Aerial Parts Extract of Dalbergia sissoo (AEDS) against scopolamine-induced Alzheimer's disease. Alcoholic extract was not harmful up to 2000 mg/kg body weight, according to the acute toxicity study. Rats were administered AEDS at 125 mg/kg body weight, 250 mg/kg body weight, and 500 mg/kg body weight, along with donepezil at a dose of 2.5 mg/kg. The Morris water maze, the Y-maze, the novel object recognition test, and biochemical tests such as the neurotransmitter acetylcholinesterase activity, catalase activity, malonyl dialdehyde, and nitric oxide assays were used to evaluate the anti-Alzheimer activity. Findings indicated that extracts prevent Alzheimer's disease and raise acetylcholine and catalase levels. Phytochemical research revealed that the AEDS contains phenolic chemicals, flavonoids, and alkaloids. The findings indicated that the presence of strong antioxidants such phenolic compounds, flavonoids, and alkaloids in the aerial sections of Dalbergia sissoo confers notable anti-Alzheimer activity.

Keywords: *Alzheimer's disease, Scopolamine, Morris water maze, Dalbergia sisso*

1. INTRODUCTION

The progressive degradation of the structure and function of the central nervous system or the peripheral nervous system is a characteristic of a diverse set of disorders known as neurodegenerative diseases. Parkinson's disease and Alzheimer's disease are common neurodegenerative illnesses. Cholinergic neurones in the basal forebrain selectively deteriorate as a result of Alzheimer's disease, a chronic progressive neurodegenerative illness [1, 2]. Because of a decline in neuronal activity and decreased concentrations of neurotransmitters in intersynaptic space, which results in poor synaptic transmission and a deficit in cholinergic neurotransmission in the central nervous system, patients with Alzheimer's disease are known to have cognitive difficulties, including memory loss and reasoning impairments.

Multiple therapy options are necessary due to the complicated pathogenicity of Alzheimer's disease, which involves both hereditary and environmental components. Cholinergic deficiency, oxidative stress, inflammatory pathways (particularly NFκB), tau protein hyperphosphorylation and aggregation, and β and γ secretases that process APP are some of the more well-known mechanisms implicated in the pathophysiology of Alzheimer's disease [3]. The reduction in cortical levels of the neurotransmitter acetylcholine is one of the most significant alterations seen in the brain. Due to the high levels of acetylcholinesterase, an enzyme involved in the metabolic hydrolysis of acetylcholine at cholinergic synapses in the central and peripheral nervous systems, acetylcholine has a very short half-life in Alzheimer's disease patients [4, 5]. The most prevalent type of neurodegenerative disease, Alzheimer's disease, is also brought on by tau protein twisting into strands of dead and dying neurones and amyloid-β peptide building up into microscopic "plaques." Inflammation and oxidative stress are also linked to Alzheimer's disease in its early stages [6].

Only symptomatic and palliative treatments are available to treat Alzheimer's disease, and no pharmaceutical has been able to effectively halt the illness's progression for an extended period of time, despite tremendous efforts to study its aetiology and pathogenesis. [7] The primary goal of treating Alzheimer's disease is to raise the amount of acetylcholine in the synaptic cleft by blocking the cholinesterase enzymes that break down acetylcholine. Alzheimer's disease patients who have low acetylcholine levels are more likely to experience memory loss, poor memory, and a progressive decline in learning [8, 9].

Native to India and the Indian subcontinent, Dalbergia sissoo Roxb. (also known as "Indian rosewood" or "shisham") is a well-known timber tree in the Fabaceae family that is mostly prized for its valuable fuel and lumber. [10] According to reports, folklore and traditional medicine employ all parts of the tree to treat a variety of illnesses. The astringent quality of the roots, the stimulating and bitter taste of the leaves, and the altering power of the bark and wood. Oil is utilised to treat skin conditions, and excoriation is done with a mixture of sweet oil and leaf mucilage [11]. Blood illnesses, scabies, skin conditions, eye and nose disorders, stomach issues, and burning sensations are all treated with wood [12–15]. The literature has documented a wide range of biomodulatory effects, including anti-inflammatory, antileprotic, antispermatogenic, and osteogenic activity [16–19].

Cholinesterase inhibitors, gene therapy, immunotherapy, modifying tau and Aβ deposition, and regulating inflammation and oxidative damage are some of the treatment modalities for AD that have been studied recently. [20] Currently authorised treatments for AD include galantamine, memantine (NMDA receptor antagonist), donepezil, and rivastigmine (cholinesterase inhibitors). [21] Finding novel medications that can stop or slow the progression of the disease is still a vital task, though, as none of these medications address the underlying disease mechanism. [22] When it comes to treating illnesses where oxidative stress is the primary underlying cause, plant-based medications are appealing targets. [23] Thus, the current study investigates Dalbergia sissoo's anti-alziemer activity in test animals.

2. MATERIALS AND METHODS

Plant Material: *Dalberigia sissoo* aerial parts were collected from local region of Lucknow, U.P. and were authenticated and specimen were submitted to Pharmacy department.The collected *D. sissoo* aerial parts was washed thoroughly with water to remove any unwanted matter. Then, it was dried in shade, ground to a coarse powder with a mechanical grinder and passed through sieve no. 40 and stored in an air-tight container.

Preparation of Extract: In a Soxhlet extractor, 50 g of the air-dried powdered aerial portions of D. sissoo were weighed and extracted using 90% ethanol. A rotary evaporator was used to concentrate the extract in a vacuum at 40 °C after it had been filtered with Whatman filter paper #1. Until it was time to utilise it, the concentrated extract was kept at 40 °C in the dark. After that, the entire investigation was conducted using an alcoholic extract of D. sissoo. [24, 25]

Phytochemical investigation: Thin layer chromatography (TLC) and initial phytochemical screening were performed on the plant's crude extract to identify the presence of tannins, alkaloids, carbohydrates, glycosides, amino acids, phytosterol, saponins, flavonoids, and alkaloids. [26]

In-vivo study:

Animals: A study named "Phytochemical and Pharmacological Evaluation of Selected Medicinal Plant for Anti-Alzheimer's Activity in Experimental Animals" was recognised by the CPCSEA by the Institutional Animal Ethics Committee (IAEC). Two weeks before the experiment, Wistar rats weighing 200–300 g of either sex were kept in the animal home for acclimatisation under regulated conditions of light (12 hours) and temperature 25 ± 1°C. Animals have unlimited access to food and water. After receiving approval from the institutional animal ethics committee, all pharmacological procedures were conducted in accordance with the guidelines set forth by the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). [27]

Acute Oral Toxicity Study: As advised by OECD Guideline 423, an acute oral toxicity investigation was performed on the alcoholic extract of Dalbergia sissoo aerial parts. At various intervals of 0, 30 minutes, 1, 2, 4, 6, 8, 12 hours, and then every day for 14 days, the animals were closely observed for the manifestation of any toxic signs or symptoms. Even at the maximum dose of 2000 mg/kg, no hazardous indications of any type were observed in clinical parameters during the acute

toxicity investigation. Therefore, it can be said that the alcoholic extract of Dalbergia sissoo has an LD50 of more than 2000 mg/kg. [28]

Grouping of Animals: Animals were divided into six groups, each of six animals. [29]

- Group I: Control group oral administered by distilled water (20 ml/kg).
- Group II: Intraperitoneal injection by scopolamine hydrochloride (0.5 mg/kg) (Disease control).
- Group III: Animal oral administered by donepezil tablet standard (2.5 mg/kg) and Alzheimer's induced with Scopolamine (Standard).
- Group IV: Animals orally administered by extract which is dissolved in NS (125 mg/kg) and Alzheimer's induced with Scopolamine (Low dose).
- Group V: Animals oral administered by extract which is dissolved in NS (250 mg/kg) and Alzheimer's induced with Scopolamine (Intermediate dose).
- Group VI: Animals oral administered by extract which is dissolved in NS (500 mg/kg) and Alzheimer's induced with Scopolamine (High dose)

Anti-Alzheimer's Activity:

Morris Water Maze Test: The rats were given 120 seconds to swim without the platform to acclimatise before the test began on the tenth day of the treatment period. Each animal underwent four 120-second learning trials with a 60-second intertrial gap over the course of the following four days. The rat was positioned in the water facing the pool wall diagonally opposite the quadrant where the platform was stored for every learning experiment. [30] The escape latency time for each trial was the amount of time it took the animal to find the submerged platform. The animals were led to the platform and given 60 seconds to rest there if they couldn't find it in 120 seconds. In this instance, the escape latency time was 120 seconds. These meetings were documented as acquisition tests or concealed platform experiments. [31] The platform was taken out of the water on day 15, which was 24 hours after the last learning trial, and they had a probing trial session to gauge their memory retention. After being submerged in water diagonally across from the target quadrant, each rat was given 60 seconds to swim and locate the quadrant where the platform had been previously positioned. It was noted how long the animal stayed in the target quadrant.

Y- Maze Test: Working memory capacity was assessed using the Y-maze test. In a single session, spontaneous change activity was recorded in a T-maze made of painted black wood. With 10 cm at the top and 3 cm at the bottom, each arm measured 12 cm in height and 40 cm in length, convergent in the middle in an equilateral triangle. Each mouse was placed at the end of an arm and allowed free passage through the maze for eight minutes. To be able to swap arms, the rats need to know which arm they have already visited. The order of arm entrances, including any possible returns into the same arm, will be visually documented. Researchers were able to assess immediate working memory capacity by recording spontaneous alternation behaviour. Entry is considered complete when the mouse's hind paws are completely inside the arm. [32] The definition of "alternation" was stated as successive entries into the three different arms (A, B, and C) on overlapping triplet sets. The percentage of trials with all three arms—ABC, BCA, or CAB but not BAB—was recorded as an alternative to measure short-term memory. On the ninth day following treatment of the final dose arm, the percentage change was calculated and entries were made.

Novel Object Recognition Test: Rats were individually given a single 10-minute familiarisation session on the first day (habituation phase, day 13 of treatment) to acquaint themselves with the equipment. This session took place in the vacant space. Two objects (A and B) were floor-fixed and positioned symmetrically in the centre line of the area for a single 10-minute session on the second day (acquisition phase, day 14 of treatment). 10 cm apart and 8 cm from the closest wall (each item takes up about 5 cm of space due to its size). The two items were the same size but different shapes, manufactured of the same material, and had a similar colour and scent. In the open area, rats were free to investigate the items. To represent the rats' level of exploration activity, the amount of time spent on each object was displayed in seconds. Rats were given two objects to investigate in the open field on the third day (retention phase, day 15 of treatment): the well-known object A and the new object C, which were different in shape but comparable in colour and size. [33, 34] Each rat's recognition index (for the retention session) was determined and represented as the ratio.

Recognition index (RI) = time exploring novel object/ (Time exploring novel object + time exploring familiar object) ×100%.

Biochemical Test:

Preparation of Brain Sample: Rats from each group were put to death using a carbon dioxide chamber after the learning and memory paradigms in Scopolamine-induced amnesia were evaluated; the brains were promptly taken out and submerged in ice-cold saline. On a petri dish that had been chilled on crushed ice, the frontal cortex, hippocampus, and septum—as well as any other areas of interest—were promptly separated. After being weighed, the tissues were mixed in 0.1M phosphate

buffer (pH 8). In order to analyse acetylcholinesterase, catalase, MDA, and nitric oxide, samples of rat brain homogenates were gathered in several test tubes. Enzymatic tests were performed using the supernatant. [35]

Estimation Acetyl Cholinesterase Enzyme Levels in the Brain: A cuvette containing 100µl of DTNB and 2.6 ml of phosphate buffer (0.1M, pH 8) was filled with a 0.4 ml aliquot of the homogenate. After completely mixing the cuvette's contents with bubbling air, the absorbance was measured in a spectrophotometer at 412 nm. [36] The absorbance was noted as the basal reading once it stabilised. After adding 20 µl of the substrate, acetylthiocholine, the absorbance change was noted. Thus, the absorbance change per minute was calculated.

Determination of Catalase (CAT) Principle: The Aebi H technique was used to measure the catalase activity. A cuvette containing 1.9 ml of 50 mM phosphate buffer (pH 7.0) was filled with 0.1 ml of supernatant. The addition of 1.0 ml of newly made 30 mM H2O2 initiated the reaction. Changes in absorbance at 240 nm were used to spectrophotometrically assess the rate of H2O2 breakdown. Catalase activity was measured in units/mg protein. [37] When H2O2 is added, the reaction starts right away. After thoroughly mixing the solutions, the absorbances were measured at 15 and 30 seconds (t1 and t2, respectively). At 240 nm, the absorbance was measured.

Determination of Malonyl Dialdehyde (MDA): The Ohkawa et al. technique was used to measure MDA. From the tissue homogenate, 1 millilitre of suspension medium was extracted and placed in a tube. Before adding 0.5 ml of 8% Thiobarbituric Acid (TBA) reagent, 0.5 ml of Trichloroacetic Acid (TCA) was added. The tubes were placed in the water bath at 80 oC for 30 minutes after being wrapped in aluminium foil. The tubes were removed after 30 minutes and submerged in cold water for another 30 minutes. These tubes were centrifuged at 3000 rpm for 15 minutes. At room temperature, the absorbance of the supernatant was measured at 540 nm using a suitable blank solution (1 ml of distilled water, 0.5 ml of 30% TCA, and 0.5 ml of 0.8% TBA). MDA value was expressed as n moles MDA/mg of protein. [38]

Determination of Nitric Oxide (NO): A colorimetric assay using the Griess reagent (1:1 solution of 1% sulfanilamide in 5% H3PO4 and 1% naphthylamine diamine dihydrochloric acid in water) was used to measure the amount of nitric oxide produced by the concentration of nitrate in the supernatant. After mixing the Griess reagent with an equivalent volume of the supernatant, the mixture was allowed to sit at room temperature in the dark for ten minutes. A spectrophotometer was used to measure the absorbance at 540 nm. A sodium nitrite standard curve was used to measure the amount of nitrite present in the supernatant. [39]

Statistical Analysis: The data were analyzed using BioStat Pro (version 5.9.8). For each category, the results are expressed as mean ± SEM. A one-way Analysis of variance (ANOVA) was used to analyze statistical differences, followed by the Tukey-Kramer test. Tests at $P \leq 0.05$were considered statistically significant. *** indicated $p < 0.001$, ** indicates $p < 0.01$, * indicates $p < 0.1$. [40]

3. RESULTS AND OBSERVATION

The yield of the alcoholic aerial parts extract of Dalbergia sissoo was precisely 8.7 percent w/w dry weight. The alcoholic extract of the aerial portions of Dalbergia sissoo contains carbohydrates, proteins, amino acids, phytosterol, alkaloids, phenolic compounds, and flavonoids, according to preliminary phytochemical analysis.

Acute Toxicity: The LD50 of the alcoholic extract was calculated to be 2000 mg/kg. The 1/4th, 1/8th, and 1/16th doses—that is, the low dose of 125 mg/kg, the intermediate dose of 250 mg/kg, and the high dose of 500 mg/kg for the test groups with dose conversion—were chosen for the study since this plant is also known to be edible, indicating its safety.

Morris Water Maze Test: In contrast to the controls, scopolamine treatment during the acquisition sessions (days 11–14) produced noticeably longer escape latencies; this effect was lessened by co-administration of the test formulation. In the control group, but not in the rats treated with Scopolamine, there were notable reductions in escape latencies on days 2–4 (treatment days 12–14) of the acquisition sessions in comparison to day 1 (treatment day 11). Rats given both the test formulation and Scopolamine concurrently showed a restoration of these differences. Rats co-administered with the test formulation and Scopolamine spent more time swimming in the target quadrant than the disease controls during the probe trial session on day 15, but rats treated with Scopolamine spent significantly less time swimming in the target quadrant than the controls.

Table 1: Effect of Alcoholic Extracts of aerial parts of *Dalbergia sissoo* on Escape Latency Time in Scopolamine-Induced Alzheimer in Wistar Albino Rats

Day / **Group**	**Escape Latency Time (Seconds)**			
	Day 11	**Day 12**	**Day 13**	**Day 14**
Control	89.75 ±7.32	80.64 ± 3.99	59.38 ± 1.56	30.62 ± 3.63

Disease Control	102.54 ± 5.86#	88.61 ± 2.54#	68.62 ± 0.53#	40.63 ± 5.74 #
Standard	83.06 ± 2.54***	57.64 ± 2.57***	29.63 ± 1.81***	17.65 ± 1.62***
Low Dose (125 mg/kg)	96.52 ± 3.64**	79.08 ± 3.65**	57.11 ± 2.64***	30.06 ± 4.64***
Medium Dose (250mg/kg)	87.75 ± 4.11**	71.43 ± 2.54**	44.54± 2.65***	25.65 ± 2.64***
High Dose (500mg/kg)	78.86 ± 0.72**	58.64 ± 4.63**	40.64± 1.54***	21.11 ± 3.75***

Values are expressed as mean ± SEM (n=6). ***P <0.001 compared with disease control, **P<0.01 compared with toxicant control and #P<0.05 compared with vehicle control. Data was analyzed using one-way ANOVA followed by the Tukey-Kramer multiple comparisons test.

Y Maze Test: When compared to the control group, the scopolamine therapy was linked to a significant decline in short-term memory ability, as evidenced by a lower spontaneous alternation percentage. While Scopolamine reduced the spontaneous alternation %, all three doses of the alcoholic extract of D. sissoo aerial parts pretreatment markedly increased it.

Novel Object Recognition Test: When compared to the illness control group (Scopolamine), all three groups treated with alcoholic extract of dried aerial parts of Dalbergia sissoo exhibited a dose-dependent rise in Recognition index.

Table 2: Effect of Alcoholic Extracts of D. sissoo extract on Percentage Alternation and Recognition Index in Scopolamine Induced Alzheimer in Wistar Albino Rats

Groups	Percentage Alternation	Recognition Index
Control	57.53 ± 4.11	57.13 ± 2.75
Disease Control	35.56 ± 4.87#	44.54 ± 5.65#
Standard Control	60.32 ± 2.53***	62.11 ± 3.75***
Low Dose (125 mg/kg)	48.62 ±4.11**	57.94 ± 2.51**
Medium Dose (250 mg/kg)	51.54 ± 3.75***	59.16 ± 3.65***
High Dose (500 mg/kg)	55.21 ±1.65***	61.62 ± 2.76***

Values are expressed as mean ± SEM (n=6). ***P <0.001 compared with disease control, **P<0.01 compared with toxicant control and #P<0.05 compared with vehicle control. Data was analyzed using one-way ANOVA followed by the Tukey-Kramer multiple comparisons test.

Biochemical Studies:

Estimation of Acetyl Cholinesterase Enzyme Levels in the Brain: When compared to the negative control group (Scopolamine), the acetylcholinesterase level decreased in a dose-dependent manner for all three test dosages of the alcoholic extract of Dalbergia sissoo's aerial parts.

Determination of Catalase (Cat): In comparison to the negative control group (Scopolamine), all three test doses of the alcoholic extract of Dalbergia sissoo's aerial parts demonstrated a dose-dependent increase in catalase activity.

Determination of Malonyldialdehyde (MDA): When compared to the disease control group (Scopolamine), all three test doses of the alcoholic extract of Dalbergia sissoo aerial parts demonstrated a dose-dependent decrease in Malonyl dialdehyde levels.

Determination of Nitric Oxide (NO): Comparing the three test dosages of the alcoholic extract of Dalbergia sissoo's aerial parts to the disease control group (Scopolamine), a dose-dependent drop in nitrite levels was observed.

Table 3: Effect of alcoholic Extract of aerial parts of *Dalbergia sissoo* Acetyl-Choline Esterase Level, Catalase Levels, MDA Levels, Nitrite Levels in Scopolamine Induced Alzheimer in Albino Wistar Rats

Groups	Enzyme Levels	Catalase Levels	MDA Levels	Nitrite Levels
Control	0.077 ±0.0002	36.54 ±1.64	0.031 ± 0.0003	8.11 ± 0.04
Disease Control	0.089 ± 0.0001#	27.38 ± 3.75#	0.036 ± 0.0002#	21.65 ± 0.48#
Standard Control	0.069 ±0.0003***	41.54 ± 1.57***	0.012 ± 0.0001***	10.87 ± 0.57***
Low Dose (125 mg/kg)	0.077 ± 0.0001**	35.87 ±2.23**	0.025 ± 0.0002**	16.64 ± 1.03**
Medium Dose (250 mg/kg)	0.075 ± 0.0002**	37.65 ± 1.43**	0.021 ± 0.0001***	14.37 ± 0.78**
High Dose (500 mg/kg)	0.071 ± 0.0004***	39.11 ± 2.57***	0.017 ± 0.0001***	12.97 ± 0.25***

Values are expressed as mean ± SEM (n=6). ***P <0.001 compared with disease control, **P<0.01 compared with toxicant control and #P<0.05 compared with vehicle control. Data was analyzed using one-way ANOVA followed by the Tukey-Kramer multiple comparisons test.

4. DISCUSSION

In the Morris Water Maze test, Dalbergia sissoo's aerial sections were examined for the anti-Alzheimer model. Rats' spatial learning and memory have been extensively tested using the MWM task [36]. It demonstrated a dose-dependent increase in time spent in the target quadrant and a decrease in escape delay time. Because behavioural evaluation can be started a day after the last stress session and finished within hours, the Y-maze task is an effective spatial memory test for chronically stressed rats. The assessment of spatial recognition-memory from the Y maze's output shows hippocampal degradation when the cumulative acts of stress are at their highest [39]. The percentage alternation increased in the Y maze test for AD rats that had been given alcoholic extracts of Dalbergia sissoo aerial parts. In animal models of neurological illnesses, the Novel Object Recognition experiment is used to evaluate memory, specifically memory recognition. This test examines the innate tendency of rodents to spend more time examining an unknown object than a familiar one. The choice to investigate the unknown object is an example of how memory is used for identification and learning [21]. Additionally, the Recognition Index increased in AD rats that had been given alcoholic extracts of Dalbergia sissoo's aerial parts prior to the Novel Object Recognition test. All of this suggests that rats' spatial memory has improved.

Acetylcholine, a neurotransmitter that stimulates cholinergic transmission by activating nicotinic and metabotropic muscarinic receptors, is broken down by acetylcholines-terase. Therefore, by decreasing ACh breakdown, acetylcholinesterase inhibitors can promote cholinergic transmission. Cognitive impairment results from the loss of ACh, which is brought on by the hydrolytic activity of AChE [40]. An examination of the neurotransmitter content in the cerebral cortex of AD rats that had received an extract from the aerial portions of Dalbergia sissoo revealed a decrease in the amount of AchE. All cells have an antioxidant defence system that includes the glycoprotein CAT, which shields them from oxidative damage brought on by reactive oxygen species. It catalyses the breakdown of H2O2 to H2O and O2, protecting the cell from highly harmful hydroxyl radicals. [41] After being treatment with an extract from the aerial portions of Dalbergia sissoo, AD rats showed an increase in catalase activity. Elevated MDA levels indicate heightened oxidative stress. It has been proposed that memory impairment is associated with an aberrant increase in MDA [42]. However, AD rats pretreated with an extract from the aerial portions of Dalbergia sissoo showed a decrease in the levels of Malonyl dialdehyde (MDA), a lipid peroxidation product. Prior to the buildup of Aβ and phosphorylated tau, oxidative and nitrosative stress have been identified in AD brains as a result of rising reactive oxygen and nitrogen species levels, respectively [43]. When compared to the negative control group, the nitrite levels of the AD rats pretreatment with Dalbergia sissoo significantly decreased. The age-related decline in cognitive function that eventually leads to Alzheimer's disease in older adults is caused by reactive oxygen species [44]. It has also been stated that Dalbergia sissoo has antioxidant qualities. AEDS may have a neuroprotective impact because of its antioxidant properties, which limit oxidative stress on vulnerable brain cells and prevent brain damage. [45] Dementia symptoms are linked to compromised neurotransmission in the impacted brain areas.

5. CONCLUSION

The results of this study suggest that by dramatically lowering the levels of acetylcholinesterase in the brain, an alcoholic extract of Dalbergia sissoo's aerial parts may help prevent or treat Alzheimer's disease. A preliminary phytochemical examination of an alcoholic Dalbergia sissoo extract revealed the presence of phenolic chemicals, alkaloids, flavonoids, and carbohydrates. Nonetheless, the anti-amnesic effects of Dalbergia sissoo may be due to the antioxidants and anti-Alzheimer

characteristics found in its alcoholic extract. This study proposes the use of Dalbergia sissoo plant extracts as a possible treatment for AD. To categorise the active phytochemicals and demonstrate the mode of action, more research is advised.

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