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EFFECT OF GINKGO BILOBA ON D -GALACTOSE INDUCED OXIDATIVE STRESS IMPAIRED COGNITIVE FUNCTION IN ALBINO RATS

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INTRODUCTION

Cognitive impairment is a clinically relevant health problem in the elderly and it is considered an intermediate state between normal aging and dementia. This state can progress to dementia, and Alzheimer's disease is the most common form of neurodegenerative disorder.

Currently, despite pharmacological new findings, there is still no specific drug approved by the FDA for the treatment of cognitive impairment. The only drugs used, approved by the FDA for the treatment of mild and moderate Alzheimer's disease are AChEIs or memantines, although their effects are not very effective and have numerous side effects such as nausea, bradycardia, fatigue. Moreover, non-pharmacological treatments such as behavioral interventions, psychosocial support, physical activity including rehabilitation programs, diet and cognitive stimulation shown a benefit to patients. Consequently, several studies suggested an important role of diet in the prevention neurodegenerative diseases, showing a protective role against the damaging effects of neuroinflammation and oxidative stress (Decandia *et al*., 2023; Diet & inflammation review, 2018).

Cognitive Functions

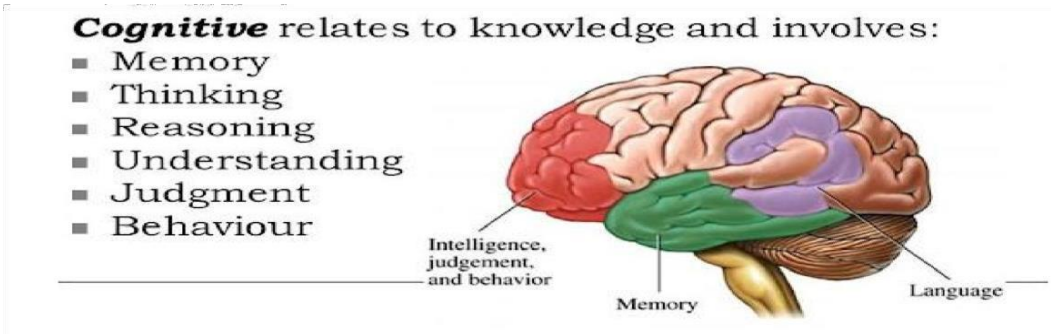


Figure No. 1: Human Brain.

Prevalence

The prevalence of Cognitive Impairment in India varies from 6.7% to 33% among older adults above 60 years, with some studies reporting it as high as 27.77% in middle-aged individuals. A study in Kerala's urban areas found a 26.1% prevalence cognitive impairment among the elderly, along with a 5.6% prevalence of dementia (Mohan *et al*., 2019).

Worldwide, dementia is estimated to affect 1.8% of people in their 60s, 5.1% of people in their 70s, 15.1% of people in their 80s, and 35.7% of people in their 90s.3 A study from the Centers for Disease Control and Prevention using the 2011 Behavioral Risk Factor Surveillance survey found that 12.7% of respondents aged 60 years. A recent study in 2024, focused on cognitive impairment in adults aged 40-60, found that 27.77% of the participants had cognitive impairment (Sharma *et al*., 2023).

Oxidative Stress and Brain

Oxidative stress might lead to cognitive and behavioral deficits. Persistent psychologic stress disrupt s oxidant-antioxidant balance within the brain, causing reduction in antioxidant enzyme function of glyoxalase, glutathione reductase, Mn SOD, Cu/Zn SOD. This leads to glutathione depletion, causing oxidative stress. Simultaneously occurring glutamate toxicity, calcium imbalance, and mitochondrial impairment collectively intensify oxidative stress, causing biochemical distress in the brain. This disrupts neurocircuitry, weakening hippocampal, amygdala, and cortical connections and ultimately causing behavioral and cognitive deficits (Salim, *et.al* 2014; Glo-Gsr anxiety study, *et al* 2023).

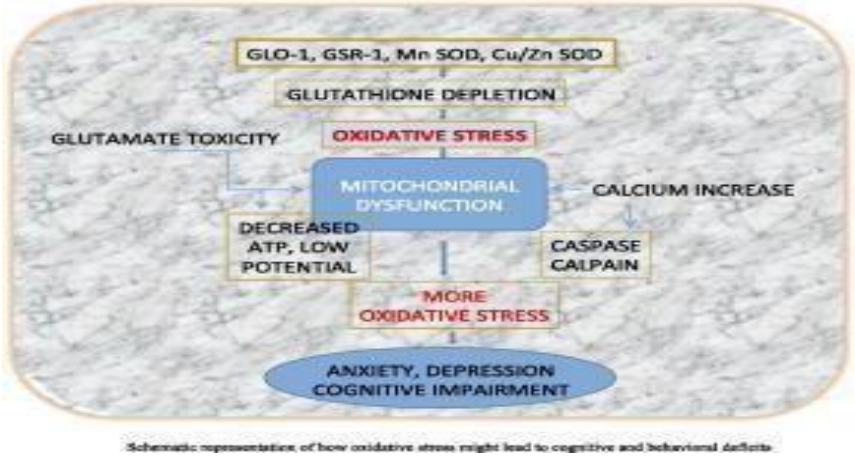


Figure No. 2: Flow chart of oxidative stress.

Mechanism of oxidative stress in the Limbic System

ROS Production: Excessive production of ROS such as superoxide radicals and hydrogen peroxide can damage the limbic system neurons.

Mitochondrial Dysfunction: Dysfunction of these organelle can lead increased ROS production and oxidative stress.

Introduction about D-Galactose

D-Galactose is a reducing sugar or monosaccharide which is abundantly present in milk products, fruits and vegetables and is usually converted into glucose by galactose-1-phosphateuridyltransferase and galactokinase. However, d-gal administration over long periods of time can lead to an enzymatic overload which impairs the body's natural ability to catalyze galactose into glucose, so causing an increase of galactitol and an activation of aldose reductase. This in turn causes a depletion in NADPH, which leads to an accumulation of hydrogen peroxide and other free radicals causing oxidative damage to the cells. Chronic administration of D-galactose at low doses has shown to induce changes that mimics natural aging processes in animals which includes cognitive impairment, oxidative stress, decreased immune response as well as gene transcriptional changes. Furthermore, continuous subcutaneous administration of D-galactose in rat contributes to marked increase in acetylcholinesterase level in the brain (Sadigh-Eteghad *et al*., 2017).

Effect of D-Galactose Induced Oxidative Stress

There is growing evidence that D-galactose induces oxidative stress in the brain, particularly at the mitochondrial level. When D-galactose levels increase, it is oxidized by the enzyme galactose oxidase, leading to the production of hydrogen peroxide (H_2O_2). This results in a reduction of antioxidant enzymes like SOD and the H_2O_2 further reacts with iron to produce hydroxyl radicals (OH^-). These ROS cause lipid peroxidation, disturb redox balance, and lead to neuronal damage (Sadigh-Eteghad *et al*., 2017).

In addition, D-galactose interacts with amino groups to form unstable compound (Schiff bases), which eventually become stable (Amadori products) and then irreversible advanced glycation end-products (AGEs). These AGEs bind with their receptors, stimulating NADPH oxidase and increasing ROS production, further damaging neurons and impairing cognitive function. Moreover, D-galactose can be converted into galactitol via galactose reductase, leading to osmotic stress and disrupted mitochondrial ETC, which increases ROS levels and contributes to mitochondrial dysfunction (MDPI report, *et.al* 2022; Frontiers pharmacology, *et. al.* 2023).

Several oxidative stress markers such as malondialdehyde, nitrite, H_2O_2, 8-oxoguanine, and NADPH oxidase subunits have been observed in studies. These indicate a strong imbalance between ROS production and antioxidant defenses, contributing to brain aging.

Furthermore, D-galactose damages mitochondrial respiratory chain enzymes, including Complex I (NADH-coQ oxidoreductase), Complex II (succinate-coQ oxidoreductase), Complex III (coQ-cytochrome C oxidoreductase), and Complex IV (cytochrome C oxidase). Some studies showed only Complex II was affected, possibly due to different methods used. The activity of tricarboxylic acid (TCA) cycle enzymes also declines, which further impairs energy metabolism. D-galactose also decreases levels of key antioxidant enzymes such as glutathione, catalase, glutathione peroxidase, glutathione-S-transferase and total antioxidant capacity. This weakened antioxidant system makes the brain more vulnerable to oxidative damage.

Moreover, D-galactose causes mtDNA mutations by reducing DNA repair enzymes like 8- oxoguanine DNA glycosylase 1 (OGG1) and polymerase γ, and inducing mtDNA deletions through NOX-related pathways. These mutations further impair mitochondrial structure and function. Benzodiazepine receptors have been shown to play a possible protective role in mitochondrial respiration, but D-galactose reduces their effectiveness as well.

The brain regions most affected by D-galactose-induced changes include the hippocampus, cerebral cortex, auditory cortex, and ventral cochlear nucleus (Sadigh-Eteghad *et al*., 2017).

Effect of D-Galactose on Cognitive Function

Learning and memory deficit occurring in age-related neurodegenerative disorders is associated with cholinergic decline. Therefore, the acetylcholinesterase activity and acetylcholine level were estimated after behavioral tests. According to previous studies, D-galactose exerted asignificant increase in acetylcholineesterasea activity and a decrease in acetylcholine level compared with the control.

Three types of glial cells such as astrocytes, oligodendrocytes, and microglia are seen in the brain. The most abundant cells in the central nervous system are astrocytes. Previous studies revealed that the activation of microglia cells and astrocytes plays pivotal roles in neurodegenerative D-galactose was given, activated microglial cells and astrocytes in hippo campus, prefrontal cortex and whole brain were observed. BDNF is essential for neuronal proliferation, excitability, synaptic transmission and plasticity. In addition, BDNF plays a crucial role in supporting the survival and growth of sensory and motor neurons, all of which have major roles in cognitive function D-galactose caused BDNF deficit, subsequently leading to cognitive impairment.

These findings suggested that D-galactose impaired cognitive function via the mechanisms involved in the increase in oxidative stress, apoptosis, inflammation, neuro modulation, the activation of astrocytes, microgila BDNF deficiency and the decrease in antioxidant enzymes. (chenn et. al., 2016).

LITERATURE REVIEW

Guangjing Xie *et al.* Neurochem Res. 2024, The research aimed to assess how treadmill exercise could help prevent brain aging and neurodegenerative diseases caused by oxidative stress, using a D-galactose-induced aging rat injected with D-galactose showed cognitive impairment and oxidative damage, but treadmill exercise helped reverse these effects. In the MWM,exercised mice performed better, indicating improved learning and memory. Exercise also reduced hippocampal and mitochondrial damage.

Mari Golub/Louise Lanoue *et al*., 2023, Evaluated hippocampal-dependent spatial working memory, learning and memory using MWM task in which rats swim to a hidden platform using distant (outside the tank), visual landmarks. It can also be indicative of damage to cortical regions of the brain. It can measure the effect of neurocognitive disorders on spatial learning and possible neural treatments.

Othman MZ *et al.,* 2022, Evaluated the MWM has been a reliable tool for testing spatial learning and memory since 1981. It is widely used due to its effectiveness in evaluating hippocampal function and adaptability to different species and test setups. Despite its strengths, some experimental and analysis gaps remain, prompting ongoing improvements. Recent updates include advanced computerized analysis and flexible testing protocols for better accuracy. With proper adaptation and careful protocol selection, MWM continues to be a valuable method for studying learning and memory.

Jiaming Liu *et. al.*, Journal of Agricultural and Food Chemistry, 2021, To investigate how *Ginkgo biloba*, protects against cognitive impairment in mice via regulation of gut microbiota.Ginkgolide is one of the main bioactive components of *Ginkgo biloba* leaf extracts with neuroprotective activity. Cognitive function was assessed by an object recognition test and an open-field test. Amyloid deposition and neuro pathological change were detected.

Thandi Mamorapelo Dorothy Fasemore *et. al.*, 2021, Investigated the role of D-Galactose. D-galactose is a sugar that, when given in high amounts, produces large amounts of ROS. These ROS damage brain cells and speed up aging, making D-galactose a common substance used to create animal models of brain aging for research.

Wei Ge, Chao Ren, Lei xing, *et.al.* 2021, Evaluated the effect of *Ginkgo biloba* extract has shown promise in protecting the brain and preventing memory loss in several brain-related diseases. It also reduced Aβ plaques, increased new born neurons, and improved dendrite structure (more branching and spine density) in the hippocampus. *Ginkgo biloba* extract helps protect against memory loss in Alzheimer's by reducing Aβ buildup and promoting neurogenesis in the brain, especially in the hippocampus of rats.

Lílian Juliana Lissner Ana Paula Toniazzo *et., at.* 2021, Assessment of object recognition test and its variants, and the MWM are behavioral tasks that are widely used to evaluate learning and memory in many animal models. In the object recognition test, learning is evaluated based on animals' curiosity for new stimuli (a novel object) (Ennaceur and Delacour, 1988a), while the MWM assesses memory through the search for survival within the pool and spatial orientation to escape the water (Morris, 1981). In both behavioral tasks, distinct types of memory are involved and many methodological variations and purposes have been developed throughout the years. Despite the different protocol or test requires subtle experimental conditions and some researchers report difficulties in defining appropriate learning indexes. MWM assessments, on the other hand, are considerably efficient at pointing out hippocampal dysfunction, but some level of stress as well as the severity and stages of disease models should be taken into consideration.

PLANT PROFILE

Figure No. 3: Parts of *Ginkgo Biloba.*

Plant Name: *Ginkgo Biloba*

Synonyms: Maiden hair tree

Family: Ginkgoaceae

Parts: Leaves

Kingdom: Plantae

Clade: Tracheophytes

Division: Ginkgophyta

Class: Ginkgoopside

Subclass: Ginkgoidae

Order: Ginkgoales

BOTANICAL DESCRIPTION

Table No. 1: Powder Characteristics of *Ginkgo Biloba*(Leaves).

Characteristic	Description
Colour	Green to yellowish-green
Odour	Slight, characteristic herbal odor
Taste	Slightly bitter
Texture	Fine, soft powder
Foreign Matter	Should be minimal or absent (per pharmacopoeial standards)
Microscopic Features	Contains fragments of: – Palisade parenchyma – Epidermal cells with anomocytic stomata – Trichomes (non-glandular hairs, few in number) – Calcium oxalate crystals (sometimes observed) – Vascular elements: xylem vessels with spiral or reticulate thickening
Moisture Content	Should be below 10% (ideal for proper storage)
Ash Values	– Total ash: ~10% or less – Acid-insoluble ash: very low

Geographical Distribution

Ginkgo biloba, also known as the maidenhair tree, is found in several places across India, particularly in the hilly regions of the north and northeast. Specifically, it has been documented in states like Uttarakhand, Himachal Pradesh, West Bengal, Jammu and Kashmir, Meghalaya, Tamil Nadu, and Punjab. While some trees are found in the Industrial Park of Kalimpong, West Bengal, the most notable areas for *Ginkgo biloba* cultivation are in the Himalayan region.

Phytochemical Constituents

Flavonoids -Flavone glycosides (24–27%) – quercetin, kaempferol, isorhamnetin Terpenoids - Ginkgolides A, B, C, J; Bilobalide.

Organic Acids -Shikimic acid, hydroxybenzoic acid Proanthocyanidins - Catechin, epicatechin

Carbohydrates - Glucose, rhamnose, and other polysaccharides Others - Sitosterols, polyprenols, vitamin C

Medicinal Uses

1. Improves Brain Blood Flow

Ginkgo biloba promotes vasodilation (widening of blood vessels) in the brain's capillaries, enhancing circulation. This improves oxygen and nutrient delivery and may be especially useful in conditions of poor cerebral perfusion like aging or stroke.

2. Enhances Oxygen & Nutrient Delivery

By improving microcirculation, *Ginkgo* helps ensure better delivery of oxygen and nutrients to brain tissues. This supports healthy cellular function and helps protect against hypoxic damage.

3. Boosts Brain Glucose Metabolism

Ginkgo improves glucose uptake and metabolism in the brain. This ensures a steady energy supply to neurons, essential for maintaining cognitive functions, memory, and alertness.

4. Improves Neurotransmitter Function

It modulates the activity of amine neurotransmitters such as dopamine, norepinephrine, and serotonin, which play key roles in mood regulation, focus, memory, and motivation.

5. Powerful Antioxidant Action

Ginkgo acts as a potent antioxidant, scavenging free radicals and protecting neurons from oxidative stress. This may help slow age-related neurodegeneration and cognitive decline.

6. Enhances Memory and Cognition

Clinical studies show improved short-term and long-term memory, attention span, and overall cognitive performance in both young and elderly individuals.

7. Neuroprotective Effects

Ginkgo helps prevent neuronal damage, especially in the hippocampus, by reducing the harmful effects of stress hormones like corticosterone. This protects brain structure and function.

8. Improves Blood Perfusion in Multiple Organs

Besides the brain, *Ginkgo* enhances blood flow in other vascular beds, including the: Ocular (eyes) – helpful in glaucoma and diabetic retinopathy.

Cochlear (ears) – may reduce tinnitus and improve hearing Cutaneous (skin) – supports healing and skin vitality Coronary (heart) – aids in cardiovascular health.

9. Cardiovascular Benefits

Ginkgo leaf extracts are widely used in preventing and managing cardiovascular diseases, thanks to their vasodilatory, anti-atherosclerotic, and anti-platelet properties.

10. Anti-inflammatory Effects

It reduces chronic inflammation in the brain, which is often linked to neurodegenerative diseases like Alzheimer’s and Parkinson’s. This also helps in conditions like brain fog and mental fatigue.

11. Supports Mood and Stress Regulation

By balancing neurotransmitters and improving brain resilience, *Ginkgo* may help in stress related mood disorders, including anxiety and mild depression.

12. Protects Against Neuronal Death

Ginkgo helps preserve neuronal integrity by reducing oxidative damage and inhibiting apoptosis (cell death) in brain cells. This effect is crucial in maintaining long-term brain health.

13. Stroke Recovery & Brain Fog

Its ability to increase cerebral blood flow and reduce oxidative damage makes it helpful in stroke rehabilitation, recovery from brain injuries, and management of mental fatigue or brain fog.

AIM AND OBJECTIVE

Aim

To study the effect of *Ginkgo Biloba* extract on D-Galactose induced
Oxidative stress impaired cognitive function in Albino rats.

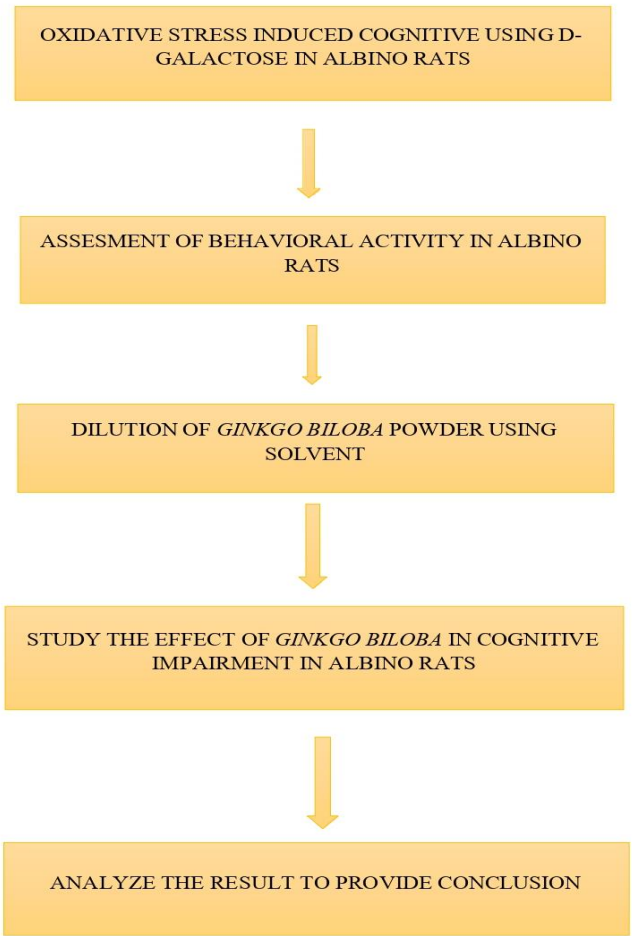
Objectives

1. Assessment of Behavioural activity in Albino rats.

MWM OFM

2. To evaluate the **pharmacological activity** of *Ginkgo biloba* in rats. Common areas of interest include Cognitive enhancement (spatial memory, learning) Antioxidant activity

PLAN OF WORK



MATERIAL AND METHOD

MATERIALS

D-Galactose

Ginkgo bilobo powder extract Solvent = Distilled Water.

Standard food – deprived to about 80% of normal calori in take. Experimental Animal-Male or Female Albino Rats 300gms

Groups

METHODS

Group A: Control (n = 2)

Group B: D-Galactose + *Ginkgo biloba* treated (n = 2)

MWM OFM

Powdered Plant Material

Standardize extract powder of *Ginkgo Biloba* was purchased from Online source (Flipkart) .It is Manufactured by "HEILEN BIOPHARM "Gujarat India.

LICNO:10722999000267, BATCH NO:SVP01052025.

Figure No. 4: *Ginkgo biloba powdered extract.*

METHODOLOGY

Drug and Treatment

Oral Administration of D-Galactose

According to Josiane Bubni., D-galactose solution was administered by oral route. It was dissolved in water for administration at the dose of 100 mg/kg of body weight, and given by oral gavage, once a day, over a period of 2 weeks. Animals were randomized into two groups: control animals (receiving water by oral gavage) or test animals (receiving d-galactose by oral gavage). After receiving of d-galactose for 2 weeks, the Albino rats exhibited signs of cognitive decline.

Oral Administration of *Ginkgo Biloba*

After administration of d-galactose for 2 weeks *Ginkgo biloba* solution was administered by oral route. It was dissolved in water for administration at the dose of 100 mg/kg of body weight, and given by oral gavage, once a day, over a period

of 2 weeks. Animals were randomized into two groups: control animals (receiving water by oral gavage) or test animals (receiving *Ginkgo biloba* by oral gavage).

Dose Calculation

For a 300gm rat =0.3kg. Dose =100mg/kg.

Required dose(mg)=dose(mg/kg)*Body weight(kg). Dose =100mg/kg*0.3kg=30mg per rat per day.

Dilution Process:

Volume (ml)=Mass (mg)/concentration(mg/ml). Volume to inject=30mg/10mg/ml=3ml.

Fig. No. 5: Dilution of *Ginkgo Biloba* extract powder.

Fig No. 6: Administration of diluted *Ginkgo Biloba.*

BEHAVIOURAL EXPERIMENT

Morris Water Maze

The MWM is one of the most widely used tasks in behavioral neuroscience for studying the psychological processes and neural mechanisms of spatial learning and memory.

Procedure

Habituation pre training

All rats were first accustomed to the water maze over a period of 2 days. On day 1, they were allowed to swim freely without any quadrant markings.

Acquisition training phase

On day 2, quadrant markings were introduced. After this acclimatization, the rats were trained to locate a hidden platform submerged about 2 cm below the water surface in the northwest quadrant. Each training sessioOPn included 4 entries into the maze (one from each quadrant), with a 1-minute gap between each entry. This training pattern was repeated three times daily for 4 days, with 30-minute intervals between each session.

After completing the training period, a baseline assessment was carried out. During this, data swimming speed, northwest latency (time taken to reach the northwest quadrant), and escape latency (time taken to reach the hidden platform). After this baseline test, the rats received their assigned drug treatments and were tested again at 2 weeks. No further training was given during this testing period, as only one round of training was done at the beginning, following the experimental protocol.

Fig. No. 7: Behavioral activity of MWM.

Table No. 2: Behavioral Parameters Measured.

Parameter	Measures
Escape latency	Time taken to reach the platform
Northwest latency	Time taken to reach the northwest quadrant
Swimming speed	To control for motor deficits
Time In target quadrant	Reflects memory in the probe trial
Number of platform crossings	Indicates memory precision during probe

Open Field Maze

An OFM is a behavioural testing apparatus used to assess the memory by evaluating the ability of the animal to recognize a stimulus or object and measuring locomotor activity, anxiety like behaviour and cognitive function (spatial memory).

Procedure

- ✓ The OFM is a common measure of general activity and exploratory behaviour in both mice and rats, where both the quantity and quality of the activity can be measured.
- ✓ The OFM apparatus (rectangular) was constructed of Plexiglas covered by white paper and measured75 × 75 cm with walls 40 cm and the floor is divided into 25 smaller rectangular units.
- ✓ The test was performed according to the protocol proposed by Todd. Briefly, rats were given 5 min exposure to the apparatus a day before the actual experiment for acclimatization of the novel arena.
- ✓ On the experiment day, the rats were conveyed to the test room in their home cages and were handled by the base of their tails.
- ✓ Each rat was placed at the centre to allow it to explore the apparatus for5 min after which they were returned to their home cage.
- ✓ The OFM apparatus was cleaned with 70% alcohol and allowed to dry after each test in order to avoid the effect of odour on the next rat.
- ✓ Using a video camera positioned at the centre of the arena above the apparatus, each trial was recorded for later analysis.
- ✓ Videos for each rat was analysed for locomotor activity (number of line crosses).

Fig. No. 8: Behavioral activity of OFM.

Table No. 3: Behavioral Parameters Measured.

PARAMETERS	MEASURES
Line crossing	Locomotor activity
Rearing	Exploratory behaviour
Time in centre	Time spend in centre(spatial assessment)
Latency to move	Time before first movement(cognitive dullness)

RESULT AND OBSERVATION

Result

Table 1: Neuroprotective effect of Morris Water Maze of Rat.

Parameters	Control	D-Galactose treated	*Ginkgo biloba* treated
Escape latency	55 sec	85 sec	60 sec
Northwest latency	25 sec	50 sec	30 sec
Swimming speed	Normal	Reduced	Near Normal
Time in target quadrant	High	Low	Moderate
Number of platform crossing	6	2	7

Result

Table 2: Neuroprotective effect of Open Field Maze of rats.

Parameters	Control	D-Galactose Treated	*Ginkgo Biloba* Treated
Line crossing	High(50)	Low(20-25)	Moderate(40-45)
Rearing	High	Low	Moderate
Time in centre	Low	High	Reduced to normal level
Latency to move	Short	Long	Long

DISCISSION

Discussion of MWM

- ✓ The MWM test in our study revealed significant changes in spatial learning and memory across different groups, confirming its validity as a sensitive tool for detecting cognitive deficits.
- ✓ Control rats showed a gradual reduction in escape latency, indicating intact learning and memory. In contrast, D-galactose-treated rats displayed prolonged escape latency and decreased time spent in the target quadrant, signifying impaired spatial memory and learning. These findings are consistent with those of **Zhou *et al.* (2017)**, who demonstrated that chronic D-galactose administration led to oxidative stress and memory impairment in

albino rats.

- ✓ Notably, rats treated with *Ginkgo biloba* extract showed improved spatial learning, reduced escape latency, and increased platform crossings in the probe trial. This supports the neuroprotective effect of *Ginkgo Biloba* against D-galactose-induced cognitive deficits. A similar observation was made by **Wei Ge *et al.* (2021**), who found that *Ginkgo Biloba* improved memory and neurogenesis in albino rats by reducing Aβ accumulation and enhancing dendritic complexity in the hippocampus (**American Journal of Translational Research).**
- ✓ The cognitive benefits of *Ginkgo Biloba* in both studies are likely attributed to its antioxidant properties and ability to modulate signaling pathways like BDNF and Nrf2. Together, these findings reinforce the potential of *Ginkgo biloba* as a therapeutic agent in neurodegenerative diseases.

Discussion of OFM

- ✓ In this study, D-galactose-treated rats showed reduced novelty-induced locomotion and impaired habituation after 2 weeks, indicating early cognitive decline. This aligns with **Zhu *et al.* (2015),** who reported decreased locomotor activity and oxidative stress in D- galactose-induced aging models. Treatment with *Ginkgo biloba* significantly reversed these impairments, supporting its neuroprotective role. Similar improvements were observed by **Santos *et al.* (2019),** where *Ginkgo biloba* enhanced exploratory behavior and antioxidant levels. These findings confirm that GB mitigates D-galactose-induced behavioral deficits via antioxidative and neuroprotective mechanisms.

CONCLUSION

The present study demonstrates that oral administration of D-galactose induces significant cognitive impairment and oxidative stress in albino rats, as evidenced by altered behavioral parameters in both the MWM and OFM tests. These impairments were characterized by increased escape latency, reduced time spent in the target quadrant, and diminished exploratory behavior.

However, concurrent treatment with *Ginkgo biloba* extract significantly ameliorated these behavioral deficits. Rats treated with *Ginkgo biloba* showed improved spatial memory, reduced anxiety-like behavior, and enhanced locomotor activity, suggesting its neuroprotective potential. The results support that *Ginkgo biloba* exerts protective effects against D-galactose- induced cognitive decline, possibly through antioxidant and neuroenhancing mechanisms.

These findings suggest that *Ginkgo biloba* may serve as a potential therapeutic agent for managing age-related cognitive decline and oxidative stress-induced neurodegeneration.

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